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## The Association between Secondhand Smoke Exposure and Metabolic Syndrome in Children with Elevated BMI

Alexandra Lawson

*Wright State University - Main Campus*

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The Association between Secondhand Smoke Exposure and Metabolic Syndrome in Children  
with Elevated BMI  
Alexandra Lawson  
Wright State University

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### Abstract

**Background & Purpose:** The childhood obesity epidemic is associated with increased metabolic syndrome (MetS) and cardiovascular morbidity. The purpose of this study was to determine whether secondhand tobacco smoke exposure (SHSe) is associated with MetS in children with elevated body mass index (BMI).

**Method:** A retrospective chart review was conducted on children aged six to 11 years with elevated BMI seen in a pediatric lipid clinic from 2008-2014. Data included age, sex, race, BMI, and five MetS criteria. MetS was defined as BMI  $\geq$ 85th percentile and  $\geq$ two of the following: systolic or diastolic blood pressure  $\geq$ 90th percentile, HDL  $<$ 35 mg/dL, triglycerides  $>$ 150 mg/dL, fasting glucose  $>$ 100 mg/dL, or fasting insulin  $\geq$ 17  $\mu$ IU/mL. Parents/guardians self-reported the number of smokers in the household; children were classified as SHSe+ if  $\geq$ one smokers resided in the household. Comparisons between exposed (SHSe+) and unexposed (SHSe-) were made with chi-square and Mann-Whitney tests. Adjusted odds ratios were determined with multiple logistic regression.

**Results:** Of 1,027 eligible patients, 514 had information for SHSe and MetS. Two hundred fifty-five patients (49.6%) lived with  $\geq$ one smokers. The prevalence of MetS was significantly higher in the SHSe+ group (54.1% vs. 31.3% SHSe- group,  $P < 0.001$ ) with an adjusted odds ratio of 2.2 (95% CI: 1.5-3.3). SHSe+ children also had significantly higher systolic blood pressure, levels of triglycerides, fasting insulin, and lower HDL levels compared to SHSe- children.

**Conclusion:** MetS is associated with secondhand smoke exposure in children. More efforts are needed to decrease SHSe, including increased physician screening pediatric patient exposure.

*Keywords:* tobacco, obesity, insulin resistance syndrome, Syndrome X, pediatrics

The Association between Secondhand Smoke Exposure and Metabolic Syndrome in Children  
with Elevated BMI

The increasing rate of childhood obesity has dramatically impacted the health of American youth. Between 1980 and 2012, the obesity rate in children aged six to 11 years has increased from 7% to 18% (Centers for Disease Control and Prevention [CDC], 2014). In turn, obesity-related diseases like diabetes have become more common in childhood and can lead to severe health problems earlier in adulthood than the average person. One of the emerging conditions of particular concern in children is metabolic syndrome, a disease process that involves abnormal insulin and glucose levels, abnormal cholesterol levels, and high blood pressure, all of which result from excess weight (Lee & Sanders, 2012). Metabolic syndrome increases the chances of developing heart disease, especially if it develops in childhood (Weitzman et al., 2005).

In addition to the growing obesity problem in the United States (U.S.), smoking and secondhand smoke exposure remain significant public health concerns. It is well known that use of tobacco products can lead to detrimental health outcomes like lung cancer, heart disease, and stroke (CDC, 2015a). Research suggests that individuals who have regular exposure to secondhand smoke may develop health issues similar to those seen in active smokers (Mason, Wheeler, & Brown, 2015).

The effects of secondhand smoke may be more pronounced in children. Secondhand smoke exposure in children has been linked to high rates of ear infections, asthma, and allergies (Mason et al., 2015). Sudden infant death syndrome (SIDS), a preventable cause of infant mortality, has also been associated with secondhand smoke exposure (Aligne & Stoddard, 1997; University of Nebraska Cooperative Extension in Lancaster County, 2003; Winickoff et al.,

2003). The realization of this effect on children has led health care providers to advocate for children and encourage parents to quit smoking, but efforts thus far have not been sufficient enough to stop the problem. Secondhand smoke exposure in children who are obese may result in greater risk of cardiovascular disease in the future, particularly if these children also suffer from metabolic syndrome.

### **Statement of Purpose**

The purpose of this study was to determine if an association exists between metabolic syndrome and secondhand smoke exposure in children, so that the mechanism of secondhand smoke may be better understood and to better inform parents about the harmful effect their smoking has on their children.

### **Literature Review**

Excessive weight in childhood, both in the forms of being overweight and obese, are recognized public health concerns. Nearly 18% of children aged six to 11 in the U.S. were considered obese according to 2005-2008 estimates. The objective as outlined in Healthy People 2020 is to reduce obesity in children aged six to 11 by 10% through better nutrition and physical activity programs (Healthy People 2020, 2016a). Diminishing the childhood obesity problem is one of the best options for preventing metabolic syndrome from developing in childhood.

Secondhand smoke also imposes a health risk to children. Healthy People 2020 (2016b) estimated that roughly 52% of children 3 to 11 years old were exposed to secondhand smoke between 2005 and 2008. Exposure to secondhand smoke can increase infection and disease in children, including but not limited to ear infections, respiratory infections, and asthma (Quinto, Kit, Lukacs, & Akinbami, 2013; Singh, Siahpush, & Kogan, 2010; Yi et al., 2012). Reducing

secondhand smoke exposure in children by 10% thus is one of the Healthy People 2020 goals, and can reduce infection and disease in young children (Healthy People 2020, 2016b).

**Metabolic Syndrome**

Metabolic syndrome is the term for a collection of metabolic abnormalities including elevated body mass index (BMI), elevated blood pressure, dyslipidemia, hyperinsulinemia, and impaired glucose tolerance (Jessup & Harrell, 2005). To be diagnosed with metabolic syndrome as an adult, one must have at least three of these criteria, as outlined by the Third Report of the National Cholesterol Education Program Adult Treatment Panel (NCEP-ATPIII), the International Diabetes Federation (IDF), and the World Health Organization (WHO), displayed in Table 1 (National Heart, Lung, and Blood Institute [NHLBI], 2004; Yadav et al., 2013).

Table 1

*Criteria for Metabolic Syndrome in Adults*

	<b>NCEP-ATPIII</b>	<b>IDF</b>	<b>WHO</b>
Diagnostic Parameters	≥3 risk factors	Central Obesity and two additional risk factors	Glucose Intolerance and two additional risk factors
Obesity	Waist circumference: ≥102 cm in men ≥88 cm in women	Waist circumference: ≥90 cm in men ≥80 cm in women	Waist-to-hip ratio: >0.90 for men >0.85 for women
Blood Pressure	≥130/85 mmHg	Systolic ≥130 mmHg or Diastolic ≥85 mmHg	≥140/90 mmHg
Triglycerides	≥150mg/dL	≥150mg/dL or treatment for high TG	≥150 mg/dL
HDL	<40 mg/dL for men <50 mg/dL for women	<40 mg/dL for men <50 mg/dL for women or treatment for low HDL	<35 for men <39 for women
Glucose Intolerance	Fasting blood glucose ≥110 mg/dL	Fasting blood glucose ≥100 mg/dL or existing diabetes mellitus	Known diabetes mellitus, fasting plasma glucose ≥6.1 mmol/L, or insulin resistance

Source: Yadav et al., 2013

Note: The WHO definition also includes microalbuminuria, which is either a urinary albumin excretion rate ≥20 µg/min or an albumin: creatinine ratio ≥30 mg/g.

Each of these factors individually is considered a risk for cardiovascular disease; however, when they are in combination, they present a greater risk of disease, including atherosclerotic cardiovascular disease and type 2 diabetes mellitus (Jessup & Harrell, 2005; Lee & Sanders, 2012; NHLBI, 2011). A systematic review completed in 2007 determined that metabolic syndrome had a relative risk of 1.54 (95% CI: 1.32 to 1.79) of cardiovascular events and death when adjusted for usual cardiovascular risk factors. Metabolic syndrome in women had a stronger association of morbidity and mortality with a relative risk of 2.63 compared to 1.98 for men ( $p=0.09$ ) (Gami et al., 2007).

Being overweight or obese, is the core problem of metabolic syndrome. Poor diet and a sedentary lifestyle are the primary contributors of metabolic syndrome, and they contribute to the mechanism of insulin resistance, a key component of metabolic syndrome (Gami et al., 2007). Family history of metabolic syndrome or obesity-related diseases or a personal history of childhood obesity also increase the likelihood of developing metabolic syndrome (NHLBI, 2011; Steinberger et al., 2009). The most recent estimate for the prevalence of metabolic syndrome for adults 20 years old and older in the U.S. is 34.7% (95% CI, 33.5%-36.0%) in 2011-2012, which is an increase from the 2003-2004 estimate of 32.9% (95% CI, 31.6%-34.2%). Hispanics were found to have the greatest prevalence of metabolic syndrome at 35.4% (95% CI, 34.2%-36.6%), followed next by Whites at 33.4% (95% CI, 32.6%-34.2%), then Blacks at 32.7% (95% CI, 31.5%-33.9%) (Aguilar, Bhuket, Torres, Liu, & Wong, 2015).

While family history can increase the chances of developing metabolic syndrome, the primary cause remains to be lifestyle. As mentioned before, people with a history of childhood obesity are more likely to develop metabolic syndrome (Steinberger et al., 2009). A study conducted in Finland surveyed individuals to determine the association of childhood lifestyle

with metabolic syndrome in adulthood. The researchers were specifically assessing diets and frequency of fruit, vegetable, fish, and butter on bread consumption, as well as physical activity. The study began in 1980 with over 2000 individuals aged three to 18 years, and proceeded to conduct follow-up surveys periodically over a period of 27 years. The study found that adults with metabolic syndrome had elevated BMIs as children compared to those without metabolic syndrome, and generally ate fewer fruits and vegetables. The researchers also found that those with less vegetable consumption in childhood had higher blood pressure and triglyceride levels than individuals with greater vegetable consumption in childhood. It also noted that the fruit and vegetable consumption trends stayed true into adulthood for both those with and without metabolic syndrome (Jaaskelainen et al., 2012).

Metabolic syndrome has traditionally been a disease of adults, but it is well established that atherosclerotic cardiovascular disease begins in childhood (Kavey et al., 2003), and with the increasing rate of childhood obesity, the components of metabolic syndrome are seen more and more in children. As stated before, the obesity rate in 2012 for children aged six to 11 was almost 18% and nearly 21% for adolescents aged 12 to 19 (CDC, 2014). Research demonstrates that children will experience more abnormal metabolic processes with more severe obesity, including low high-density lipoprotein (HDL) cholesterol, elevated triglycerides levels, elevated glycated hemoglobin, and elevated blood pressures (Skinner, Perrin, Moss, & Skelton, 2015). However, to this date, there still is not a concrete definition of metabolic syndrome in children.

There has yet to be a concrete definition of metabolic syndrome in children because the metabolic processes are always changing, and what is deemed a normal value depends predominantly on the age of the child (Steinberger et al., 2009). In adults, it is fairly simple to create an absolute value for normal and abnormal cholesterol levels, blood pressure, weight,

glucose, and insulin levels. These values continuously change in childhood because a child is constantly growing. Therefore, it is extremely difficult to reach consensus on one set of criteria. Instead, there must be a set criteria for the different age ranges during childhood. The International Diabetes Federation (2015) established criteria for ages six to 10, 10 to 16 and over 16 years old, but even in these criteria, the waist circumference limits depend on the region of the world and children aged six to 10 are not supposed to be diagnosed with metabolic syndrome. Unfortunately, because there are varying sets of criteria, it is difficult to determine the true prevalence rate of metabolic syndrome in children, because it largely depends on which definitions or criteria are used. Recent estimates place the prevalence of metabolic syndrome between 2% to 9% for the pediatrics population as a whole, and between 12% to 44% in obese children (Lee & Sanders, 2012).

Many studies across the world have established their own criteria of metabolic syndrome for the pediatric population, examples of which are given in Table 2. It is more than apparent that the variances between different criteria present a challenge to establishing a uniform set of standards.

A key challenge is using waist circumference as a measurement of body fat. Multiple studies have demonstrated that waist circumference is better associated with visceral fat than BMI (Steinberger et al., 2009), and because BMI relies solely on a person's height and weight, it cannot take into account muscle mass (Barlow & Expert Committee, 2007). Waist circumference is also associated with high blood pressure and abnormal cholesterol levels, and it can serve as a predictor for insulin resistance (Lee & Sanders, 2012).

Table 2

*Examples of Metabolic Syndrome Criteria for the Pediatric Population*

<b>Study</b>	<b>Diagnostic Parameters</b>	<b>Obesity</b>	<b>Blood Pressure</b>	<b>Triglycerides</b>	<b>HDL</b>	<b>Glucose Intolerance</b>
International Diabetes Federation, 2007*	Central obesity and 2 additional risk factors	≥10-16 years: WC ≥90 <sup>th</sup> percentile or adult cutoff if lower >16 years: Adult criteria	≥10-16 years: systolic BP ≥130 or diastolic BP ≥85 mm Hg >16 years: systolic BP ≥130 or diastolic BP ≥85 mm Hg or treatment of hypertension	≥10-16 years: ≥150 mg/dL >16 years: ≥150 mg/dL or specific treatment for high TG	≥10-16 years: <40 mg/dL >16 years: <40 mg/dL in males and <50 mg/dL in females, or specific treatment for HDL	FPG ≥100 mg/dL or known type II diabetes mellitus
de Ferranti et al., 2004	≥3 risk factors	WC ≥75 <sup>th</sup> percentile (specific for age, gender, ATPIII)	≥90 <sup>th</sup> percentile (age, gender and height specific, NHBPEP)	≥97 mg/dL (Lipid Research Clinics)	<50 mg/dL (Lipid Research Clinics)	FPG ≥110 mg/dL
Cruz et al., 2004	≥3 risk factors	WC ≥90 <sup>th</sup> percentile (specific for age, gender, race NHANES III)	≥90 <sup>th</sup> percentile (age, gender and height specific, NHBPEP)	≥90 <sup>th</sup> percentile (specific for age and gender, NHANES III)	≤10 percentile (specific for age and gender, NHANES III)	2-hour glucose >140 mg/dL in OGTT

Table 2 (continued)

*Examples of Metabolic Syndrome Criteria for the Pediatric Population*

<b>Study</b>	<b>Diagnostic Parameters</b>	<b>Obesity</b>	<b>Blood Pressure</b>	<b>Triglycerides</b>	<b>HDL</b>	<b>Glucose Intolerance</b>
Weiss et al., 2004	≥3 risk factors	>97 <sup>th</sup> percentile BMI or BMI z score ≥ 2 (age and gender specific)	>95 <sup>th</sup> percentile (specific for age, gender and height, NHBPEP)	>95 <sup>th</sup> percentile (age, gender and race specific, NGHS)	<5 <sup>th</sup> percentile (age, gender and race specific, NGHS)	2-hour glucose >140 mg/dL in OGTT
Cook et al., 2003; Ford et al., 2005*	≥3 risk factors	WC ≥90 <sup>th</sup> percentile (age and gender specific, NHANES III)	≥90 <sup>th</sup> percentile (specific for age, gender and height, NHBPEP)	≥110 mg/dL (specific for age, NCEP)	≤40 mg/dL	FPG ≥110 mg/dL or 2-hour glucose >140 mg/dL in OGTT
Jessup & Harrell, 2005	≥3 risk factors	≥95 <sup>th</sup> percentile BMI	≥90 <sup>th</sup> percentile	>110 mg/dL	≤35 mg/dL	FPG >100 mg/dL Insulin >15 μU/L
Loureiro et al., 2015	All factors	WC >90 <sup>th</sup> percentile (NHANES)	≥90 <sup>th</sup> percentile (specific for age and gender)	>110 mg/dL	≤40 mg/dL	FPG >100 mg/dL
Weitzman et al., 2005	≥3 risk factors	WC ≥90 <sup>th</sup> percentile (age and gender specific)	≥90 <sup>th</sup> percentile	≥110 mg/dL	≤40 mg/dL	FPG ≥100 mg/dL

Note: WC = waist circumference; ATP III = Adult Treatment Panel III of the National Cholesterol Education Program (NCEP); NHANES III = third National Health and Nutrition Survey; NHBPEP = National High Blood Pressure Education Program; NGHS = National Growth and Health Survey; FPG = fasting plasma glucose; OGTT = oral glucose tolerance test. \*Criteria were given in (Lee & Sanders, 2012).

Despite the advantages of waist circumference, the American Academy of Pediatrics (AAP) recommends that BMI be used to indicate an individual's level of body fat. While the BMI measurement is not perfect, the AAP has found that the BMI sensitivity for identifying body fat above the 85<sup>th</sup> percentile is actually quite strong. Therefore, the designation for being overweight begins at the 85<sup>th</sup> percentile, and the designation for being obese begins at the 95<sup>th</sup> percentile. BMI is also much easier to calculate, because height and weight are routinely and accurately measured (Barlow & Expert Committee, 2007), whereas waist circumference is subject to bias due to a lack of clear guidelines for measuring waist circumference (Lee & Sanders, 2012). BMI also has a better correlation with blood pressure than waist circumference (Weiss et al., 2004).

Fortunately, there was much more congruency for the criteria of blood pressure. The vast majority of studies listed in Table 2 specified greater than or equal to the 90<sup>th</sup> percentile of systolic or diastolic pressure as the cutoff for metabolic syndrome. Because blood pressure is dependent on age, sex, and height, blood pressure tables for children created by the National Heart, Lung, and Blood Institute were referenced (NHLBI, 2004).

The criteria for triglycerides, HDL, and glucose intolerance had much more variation. The limit for triglyceride levels could be argued to be 110 mg/dL or 150 mg/dL based on the studies in Table 2. In a recent publication, the criteria for hypertriglyceridemia in children and adolescents was deemed to be greater than 150 mg/dL. It was determined that risk for cardiovascular disease and pancreatic disease was seen above the 150 mg/dL level (Shah & Wilson, 2015). The criteria for HDL also varied from study to study and varies depending on the levels of the other forms of cholesterol (Horsley, 2009). In general, an HDL below 35 mg/dL is

found to be the level that cardiovascular risk increases in children and adolescents (Stanford University, 2016).

Glucose intolerance was much more difficult to define. Table 2 shows that the majority of studies used a fasting plasma glucose limit of 100 or 110 mg/dL, while other studies chose to use the two-hour glucose level from an oral glucose tolerance test, also known as impaired glucose tolerance. Abnormal fasting plasma glucose does not occur frequently in childhood, therefore the two-hour glucose level from an OGTT is more accurate (Weiss et al., 2004). However, OGTTs are not frequently done in the pediatric population and mostly conducted on children with multiple symptoms and signs of insulin intolerance. Requiring OGTT data can create a bias in patient selection. In addition, insulin resistance occurs more often than abnormal glucose levels in childhood (Weiss et al., 2004).

### **Secondhand Smoke Exposure and Patient Advocacy**

The various effects of secondhand smoke are still being discovered, but it is apparent that secondhand smoke exposure impacts morbidity and mortality. Secondhand smoke exposure in adults leads to asthma, lung cancer, and heart disease, as well as other diseases (Mason et al., 2015). Children are especially sensitive to secondhand smoke exposure. Because their bodies' defense mechanisms are still developing, children can absorb more of the toxins from the same amount of exposure as an adult (Mason et al., 2015). Unfortunately, most of a child's secondhand smoke exposure occurs in the home (Aligne & Stoddard, 1997; Mason et al., 2015; Winickoff et al., 2003). It is estimated that roughly 41.3% of children aged three to 11 were exposed to secondhand smoke from 2009 to 2012 (Healthy People 2020, 2015), and disparities exist in which children of Hispanic decent, as well as children from lower socioeconomic classes are more likely to have secondhand smoke exposure than children of different race or higher

socioeconomic status (Singh et al., 2010; Yi et al., 2012). As a result, there are increased rates of respiratory tract infections, asthma, ear infections, and urinary dysfunction in all children, especially younger children (Aligne & Stoddard, 1997; Emmons et al., 2001; Mason et al., 2015; Winickoff et al., 2003); almost all of these diseases are more associated with maternal smoking than paternal smoking (Aligne & Stoddard, 1997). Smoking while pregnant results in low-birth weight, and infants with secondhand smoke exposure are more likely to die from SIDS (Aligne & Stoddard, 1997; University of Nebraska Cooperative Extension in Lancaster County, 2003; Winickoff et al., 2003).

Secondhand smoke exposure has also been found to have an association with metabolic syndrome. A study conducted in 2009 found that secondhand smoke exposure in adults was not only associated with metabolic syndrome (odds ratio [OR]=2.58,  $p = 0.01$ ), but also associated with central obesity (OR=2.70,  $p<0.001$ ), hypertriglyceridemia (OR=2.10,  $p=0.02$ ), decreased levels of HDL (OR=1.90,  $p=0.02$ ) and increased levels of fasting insulin ( $p<0.01$ ) (Xie et al., 2010). An association between metabolic syndrome and secondhand smoke also exists in adolescents. In a study conducted in 2005, adolescents 12 to 19 years old were questioned about their secondhand smoke exposure and smoking habits. They subsequently were tested for serum cotinine, a byproduct of tobacco products, to determine the level of secondhand smoke exposure and smoking, and were tested for metabolic syndrome (see criteria in Table 2) (Weitzman et al., 2005). Researchers found that secondhand smoke exposure increased the odds of having metabolic syndrome by 4.7 (95% CI: 1.7 to 12.9). To compare, the adolescents that actively smoked had an odds ratio of 6.1 (95% CI: 2.8 to 13.4) (Weitzman et al., 2005).

Secondhand smoke exposure is clearly an important environmental risk factor in a child's health. It has been linked to multiple disease processes, and more are being discovered every

day. A recent study even found that prenatal and postnatal exposure to secondhand smoke increased the likelihood of having insulin resistance in ten-year-olds (Thiering et al., 2011). Unfortunately, secondhand smoke exposure is not frequently addressed in doctor appointments. A study conducted in 2002 surveyed over 900 parents whose child was seen by a pediatrician or a family practitioner in the last year. The parents were asked if the pediatrician or family practitioner inquired about the smoking status of household members. If the pediatrician or family practitioner had asked about smoking status, parents were subsequently asked if the physician informed them about the dangers of smoking and secondhand smoke to the child's health, and if the physician counseled the parent on how to quit. Fifty-two percent of parents who visited a pediatrician and 42% who visited a family physician said they had been asked about smoking status. For the parents who did smoke, 41% were advised by a pediatrician and 33% were advised by a family physician about the dangers of secondhand smoke to their children. Lastly, only 36% and 45% of parents who smoke said they were advised to quit by a pediatrician and family physician, respectively (Winickoff et al., 2003).

Despite being nationally recommended, assessment of a child's risk for secondhand smoke exposure is relatively low for both pediatricians and family physicians. These physicians have a great opportunity to address smoking cessation because children have much more frequent appointments than do parents (American Academy of Pediatrics, 2016). It is imperative that pediatricians and family physicians routinely address secondhand smoke for both the child's and the parent's health.

### **Methods**

A retrospective chart review was completed examining patients six to 11 years old who were referred to the Lipid Clinic at Dayton Children's Hospital (Dayton, OH, USA) between

March 8, 2008 and December 31, 2014. The data were de-identified and received from Dayton Children's Hospital. The project was granted permission by the Dayton Children's Hospital Institutional Review Board (IRB), and the Wright State University Office of Research and Sponsored Programs determined the study did not need separate IRB approval (see appendices A and B).

Data collected included height, weight, blood pressure, BMI, BMI percentile, race, sex, and age at the initial visit. The secondhand smoke exposure data was self-reported by the parent/guardian, as they are asked about household member smoking status during the first visit to the lipid clinic. The secondhand smoke questions specifically ask if mom, dad, or anyone else (e.g. grandparent or sibling) in the house smokes. Lastly, the laboratory data necessary for determining metabolic syndrome status (see Table 3) was collected.

Table 3

*Metabolic Syndrome Criteria*

<b>Risk Factor</b>	<b>Criteria</b>
BMI	$\geq 85^{\text{th}}$ percentile
Systolic and/or Diastolic Blood Pressure	$\geq 90^{\text{th}}$ percentile
Fasting Glucose	>100 mg/dL
Fasting Insulin	$\geq 17 \mu\text{IU/mL}$
Triglycerides	>150 mg/dL
HDL	<35 mg/dL

Note: BMI and two other risk factors qualifies for metabolic syndrome.

Laboratory data was only included if it had been collected within six months of the initial appointment. All labs performed were collected as part of the medical evaluation at the Lipid Clinic. The criteria of metabolic syndrome is a BMI at or above the 85<sup>th</sup> percentile, and any two of the additional five risk factors listed in Table 3. Patients who were missing any of the data necessary for secondhand smoke exposure or metabolic syndrome were excluded from the study.

The inclusion criteria for metabolic syndrome were determined by examining best practices, comparing previous metabolic syndrome studies in children, and determining levels which increased cardiovascular disease risk. The criteria for each risk factor are described below:

- **BMI:** As previously stated, waist circumference has a better association with visceral fat than BMI (Steinberger et al., 2009). However, BMI has a strong sensitivity for identifying body fat above the 85<sup>th</sup> BMI percentile (Barlow & Expert Committee, 2007). Additionally, waist circumference measurement has potential for many biases because there is no clear guideline as to how to measure it (Lee & Sanders, 2012), whereas BMI is more easily and frequently calculated. Therefore, BMI at the 85<sup>th</sup> percentile or greater was used as the criterion, as according to the growth charts set by the Centers for Disease Control and Prevention (2010).
- **Blood Pressure:** The majority of studies examining metabolic syndrome used the 90<sup>th</sup> percentile or greater as the criterion for blood pressure.
- **Glucose Intolerance:** Both fasting plasma glucose and fasting insulin were used to determine glucose intolerance. Because of the low frequency of elevated fasting plasma glucose, the criterion was set to greater than 100 mg/dL. The criterion for fasting insulin was set to equal or greater than 17  $\mu$ IU/mL (Sabato, 2011).
- **Triglycerides:** The criterion for triglycerides was set to greater than 150 mg/dL as cardiovascular disease risk is seen above this level (Shah & Wilson, 2015).
- **HDL:** The criterion for HDL was set to less than 35 mg/dL as cardiovascular disease risk is seen below this level (Stanford University, 2016).

Each patient was assessed to determine if they qualified for metabolic syndrome, as well as being classified into secondhand smoke exposure and no secondhand smoke exposure groups. A patient was classified as having exposure to secondhand smoke if at least one member of the household was a smoker. In order to be classified as no secondhand smoke exposure, at least mom and dad reported being non-smokers, and the other family member(s) were listed as non-smokers or it was left blank, indicating no other smoker lived in the house.

### **Data Analysis**

BMI percentiles were calculated using a SAS Institute, Inc., program (2015) for the 2000 CDC Growth Charts ages zero to <20 years (CDC, 2015b). Mean values for the BMI relative to the 50<sup>th</sup> percentile (rBMI50) were calculated using the BMI-for-age tables provided by the Centers for Disease Control and Prevention (2001). The mean values for fasting glucose, fasting insulin, triglycerides, HDL, and blood pressure were also calculated, both for the entire group as well as rBMI50 subgroups. Systolic and diastolic blood pressure were measured at the initial visit, and the blood pressure percentiles were determined using blood pressure tables for children and adolescents from the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescent (NHLBI, 2004).

Comparisons were made between the group with secondhand smoke exposure and group without secondhand smoke exposure for sex, race, metabolic syndrome, and each of the metabolic syndrome criterion. The p-values for these differences were calculated either using chi-square test, or Fisher's exact test, with  $\alpha=0.05$ . The continuous variables, including age, height, weight, all metabolic syndrome criteria, BMI z-score and rBMI50 were also compared between secondhand smoke exposure and no secondhand smoke exposure. The p-values were calculated using one of listed tests as appropriate: two sample t-test with equal variances, two

sample t-test with unequal variances, or Mann-Whitney test for skewed data. In order to determine any confounding factors and to assess for an independent association between metabolic syndrome and secondhand smoke exposure, a multiple logistic regression analysis was also conducted. Analyses were completed using SPSS.

### **Results**

There were 1,094 patients aged six to 11 who were referred to the Lipid Clinic at Dayton Children's Hospital between March 8, 2008 and December 31, 2014. Of those 1,094 patients, 514 patients had complete data on secondhand smoke exposure and the lab data for metabolic syndrome. Fifty-eight percent of the 514 patients were female, and the majority of patients were White (59.1%). In order to be included for the study, patients had to be equal or greater than the 85<sup>th</sup> percentile for BMI. However, the majority of patients (62.6%) were at or above the 99<sup>th</sup> percentile, meaning the majority of patients were considered obese.

Of the 514 patients included in the study, 255 patients (49.6%) were classified as having secondhand smoke exposure. The median age for all patients was  $9.5 \pm 2.0$  years, and the median BMI percentile for all patients was  $99.3 \pm 1.1$ . The median age, median BMI percentile, BMI range, mean BMI z-score, and mean rBMI50 are given in Table 4, in the total sample and with distinction for patients without secondhand smoke exposure (SHSe-) and patients with secondhand smoke exposure (SHSe+). Age and BMI percentile were found to be left-skewed; therefore median and interquartile range is given.

Table 4

*Sample Median Age and BMI Characteristics, and By Secondhand Smoke Exposure*

	<b>All Patients</b>	<b>SHSe-</b>	<b>SHSe+</b>	<b>p-value</b>
Median Age	9.5 ± 2.0	9.5 ± 2.0	9.6 ± 2.1	0.654 <sup>†</sup>
Median BMI Percentile	99.3 ± 1.1	99.2 ± 1.4	99.4 ± 0.9	<0.001 <sup>†</sup>
BMI Percentile Range	89.4 – 100.0	90.4 – 99.9	89.4 – 100.0	
Mean BMI z-score	2.40 ± 0.36	2.34 ± 0.37	2.46 ± 0.35	<0.001 <sup>‡</sup>
Mean rBMI50	1.79 ± 0.31	1.74 ± 0.31	1.85 ± 0.30	<0.001 <sup>‡</sup>

Note: <sup>†</sup>p-value calculated using Mann-Whitney test. <sup>‡</sup>p-value calculated using two-sample t-test with equal variances.

The distribution of race between all patients, as well as the patients with secondhand smoke exposure and without secondhand smoke exposure is given in Table 5, and it is shown in Figure 1. It should be noted that 15 patients were missing information about race; therefore, the information provided in Table 5 and Figure 1 applies to 499 patients of the total 514 patients.

Table 5

*Distribution of Race in All Patients and By Secondhand Smoke Exposure*

	<b>All Patients (n=499) No. (%)</b>	<b>SHSe- (n=249) No. (%)</b>	<b>SHSe+ (n=250) No. (%)</b>	<b>p-value</b>
White	295 (59.1)	126 (50.6)	169 (67.6)	
Black	153 (30.7)	92 (36.9)	61 (24.4)	
Hispanic	36 (7.2)	25 (10.0)	11 (4.4)	<0.001
Asian	1 (0.2)	1 (0.4)	0 (0.0)	
Other	14 (2.8)	5 (2.0)	9 (3.6)	

Note: p-value calculated using Fisher's exact test.

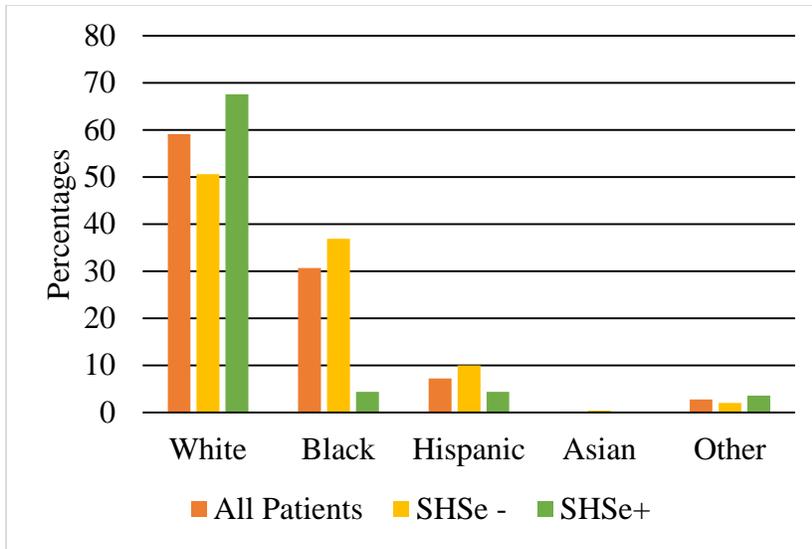


Figure 1. Distribution of race in all patients and by secondhand smoke exposure.

Note:  $p < 0.001$ , calculated using Fisher’s exact test.

In the total sample, 42.6% of patients met the criteria of metabolic syndrome. The characteristics of age and BMI were also assessed depending on whether or not the patient qualified for metabolic syndrome. The median age as well as BMI characteristics for those with metabolic syndrome (MetS+) and those without (MetS-) are given in Table 6. The patients without metabolic syndrome were found to be younger than the patients with metabolic syndrome, and BMI percentile was found to be greater for those with metabolic syndrome. All of the factors were found to be statistically significant between patients with and without metabolic syndrome.

Table 6

*Sample Median Age and BMI Characteristics, and By Metabolic Syndrome*

	<b>All Patients</b>	<b>MetS-</b>	<b>MetS+</b>	<b>p-value</b>
Median Age	9.5 ± 2.0	9.4 ± 2.1	9.7 ± 1.9	0.002 <sup>†</sup>
Median BMI Percentile	99.3 ± 1.1	99.1 ± 1.5	99.5 ± 0.6	<0.001 <sup>†</sup>
BMI Percentile Range	89.4 – 100.0	89.4 – 100.0	90.4 – 100.0	
Mean BMI z-score	2.40 ± 0.36	2.32 ± 0.38	2.50 ± 0.32	<0.001 <sup>‡</sup>
Mean rBMI50	1.79 ± 0.31	1.72 ± 0.31	1.89 ± 0.29	<0.001 <sup>*</sup>

Note: <sup>†</sup>p-value calculated using Mann-Whitney test. <sup>‡</sup>p-value calculated using two-sample t-test with unequal variances. <sup>\*</sup>p-value calculated using two-sample t-test with equal variances.

The distribution of race according to presence of metabolic syndrome was also examined. Once again, the patients with metabolic syndrome were predominantly White. The distribution of race between those with metabolic syndrome and those without metabolic syndrome follows a similar trend to the distribution of race between those with secondhand smoke exposure and those without, respectively. The data regarding race is given in Table 7, and its distribution is shown in Figure 2.

Table 7

*Distribution of Race in All Patients and By Metabolic Syndrome*

	<b>All Patients (n=499)</b>	<b>MetS- (n=284)</b>	<b>MetS+ (n=215)</b>	<b>p-value</b>
	<b>No. (%)</b>	<b>No. (%)</b>	<b>No. (%)</b>	
White	295 (59.1)	153 (53.9)	142 (66.0)	0.012
Black	153 (30.7)	103 (36.3)	50 (23.3)	
Hispanic	36 (7.2)	21 (7.4)	15 (7.0)	
Asian	1 (0.2)	1 (0.4)	0 (0.0)	
Other	14 (2.8)	6 (2.1)	8 (3.7)	

Note: p-value was calculated using Fisher's exact test

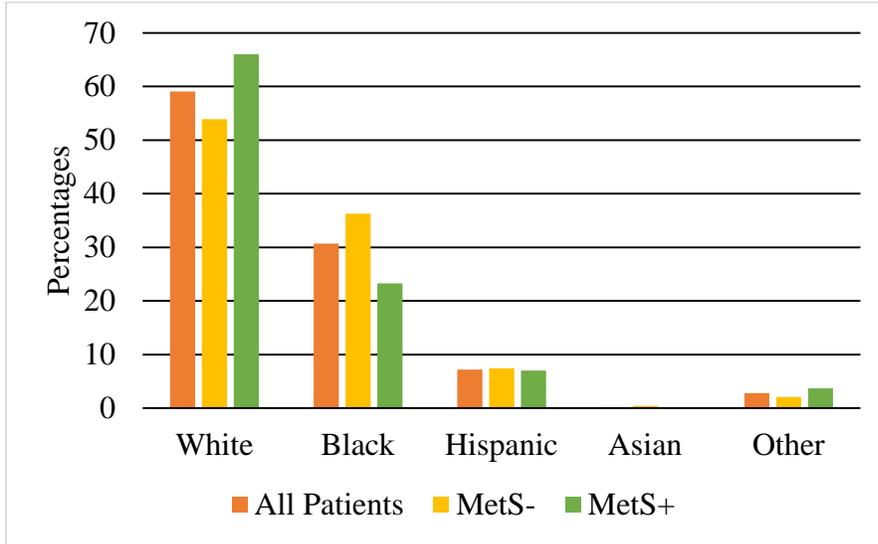


Figure 2. Distribution of race in all patients and by metabolic syndrome.

Note:  $p=0.012$ , calculated using Fisher’s exact test

It was determined that metabolic syndrome was greater in patients with secondhand smoke exposure (54.1%) than those without secondhand smoke exposure (31.3%, chi-square  $p$ -value  $<0.001$ ). In order to control for confounding factors, multiple logistic regression was performed in order to predict metabolic syndrome, the result of which is given in Table 8. Again, because 15 patients had missing information about race, the multiple logistic regression was completed with 499 patients.

Table 8

*Multiple Logistic Regression Predicting Metabolic Syndrome*

<b>Variable</b>	<b>Adjusted Odds Ratio (95% CI)</b>	<b>p-value</b>
Secondhand smoke exposure (reference = no exposure)	2.2 (1.5-3.3)	<0.001
Female sex (reference = male sex)	2.4 (1.6-3.5)	<0.001
White Race (reference = all non-White races)	1.6 (1.0-2.3)	0.031
Age (years)	1.3 (1.1-1.5)	0.001
BMI Percentile	1.5 (1.3-1.8)	<0.001

Note: For age, the odds ratio is for a one year increase. For BMI, odds ratio is for a one percentile increase.

As can be seen in Table 8, age and BMI had separate associations with predicting metabolic syndrome, but more importantly, so did secondhand smoke exposure. Having secondhand smoke exposure increased the odds of having metabolic syndrome by a factor of 2.2 (95% CI: 1.5-3.3). It is important to note that age and BMI are strongly associated with metabolic syndrome, despite having a lesser odds ratio as compared to secondhand smoke. In Table 8, the odds ratio is for an increase of age by one year and increase in BMI by one percentile. Table 9 gives the adjusted odds ratio for age when the increase is by two or more years, and the adjusted odds ratio for BMI percentile by two, five, or 10 percentile increase is given in Table 10.

Table 9

*Adjusted Odds Ratio for 2-5 Year Increases in Age*

<b>Increase</b>	<b>Adjusted Odds Ratio (95% Confidence Interval)</b>
2 years	1.7 (1.3-2.3)
3 years	2.2 (1.4-3.5)
4 years	2.9 (1.6-5.3)
5 years	3.8 (1.8-8.0)

Table 10

*Adjusted Odds Ratio for 2, 5 and 10 Percentile Increases in BMI Percentile*

<b>Increase</b>	<b>Adjusted Odds Ratio (95% Confidence Interval)</b>
2 percentiles	2.3 (1.6-3.3)
5 percentiles	7.8 (3.1-19.6)
10 percentiles	61.4 (9.8-384.0)

The number of risk factors (as defined in Table 3), excluding a BMI  $\geq 85^{\text{th}}$  percentile which was found in all patients, is given in Table 11, and it is distributed by secondhand smoke exposure classification. Patients with two or more risk factors were more likely to have secondhand smoke exposure, and the difference of the number of risk factors for those with and without secondhand smoke exposure was significant ( $p < 0.001$ ). The distribution of risk factors for all patients as well as secondhand smoke exposure classification is shown in Figure 3.

Table 11

*Distribution of Metabolic Syndrome Risk Factors in all Patients and by Secondhand Smoking Exposure*

	<b>All Patients (%)</b>	<b>SHSe – (%)</b>	<b>SHSe+ (%)</b>	<b>p-value</b>
0	18.3	20.8	15.7	<0.001
1	39.1	47.9	30.2	
2	27.2	22.0	32.5	
3	10.3	5.4	15.3	
4	3.5	2.7	4.3	
5	1.6	1.2	2.0	

*Note:* p-value was calculated with Fisher’s exact test.

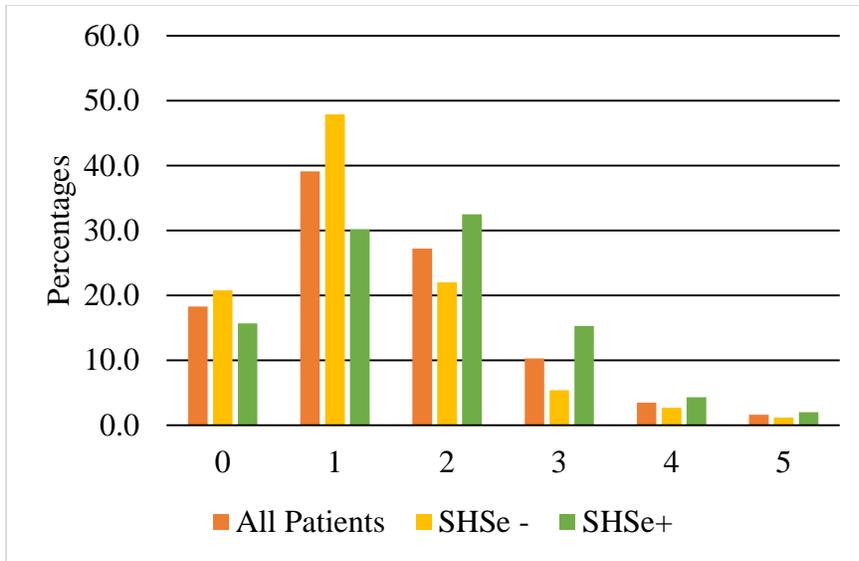


Figure 3. Distribution of metabolic syndrome risk factors in all patients and by secondhand smoking exposure.

Note:  $p < 0.001$ , calculated using Fisher’s exact test.

In addition to Table 11 and Figure 3, the number of patients who qualified for each of the metabolic syndrome risk factors is given in Table 12, and the distribution is shown in Figure 4. The most common abnormalities were systolic blood pressure and fasting insulin, according to the metabolic syndrome criteria defined in Table 3. Abnormal metabolic levels were more likely to occur in the patients with secondhand smoke exposure, and the difference between secondhand smoke exposure and no secondhand smoke exposure was significant for HDL levels ( $p=0.088$ ), triglyceride levels ( $p=0.050$ ), and fasting insulin levels ( $p<0.001$ ), as reported in Table 12.

Table 12

*Metabolic Abnormalities in all Patients and by Secondhand Smoke Exposure*

	<b>All Patients No. (%)</b>	<b>SHSe – No. (%)</b>	<b>SHSe+ No. (%)</b>	<b>p-value</b>
Systolic Blood Pressure $\geq 90^{\text{th}}$ percentile	285 (55.4)	134 (51.7)	151 (59.2)	0.088
Diastolic Blood Pressure $\geq 90^{\text{th}}$ percentile	42 (8.2)	21 (8.1)	21 (8.2)	0.958
HDL $< 35$ mg/dL	110 (21.4)	42 (16.2)	68 (26.7)	0.004
Triglyceride $> 150$ mg/dL	103 (20.0)	43 (16.6)	60 (23.5)	0.050
Fasting Glucose $> 100$ mg/dL	62 (12.1)	26 (10.0)	36 (14.1)	0.156
Fasting Insulin $\geq 17$ $\mu\text{IU/mL}$	184 (35.8)	73 (28.2)	111 (43.5)	$< 0.001$

Note: p-values were calculated using chi-square test

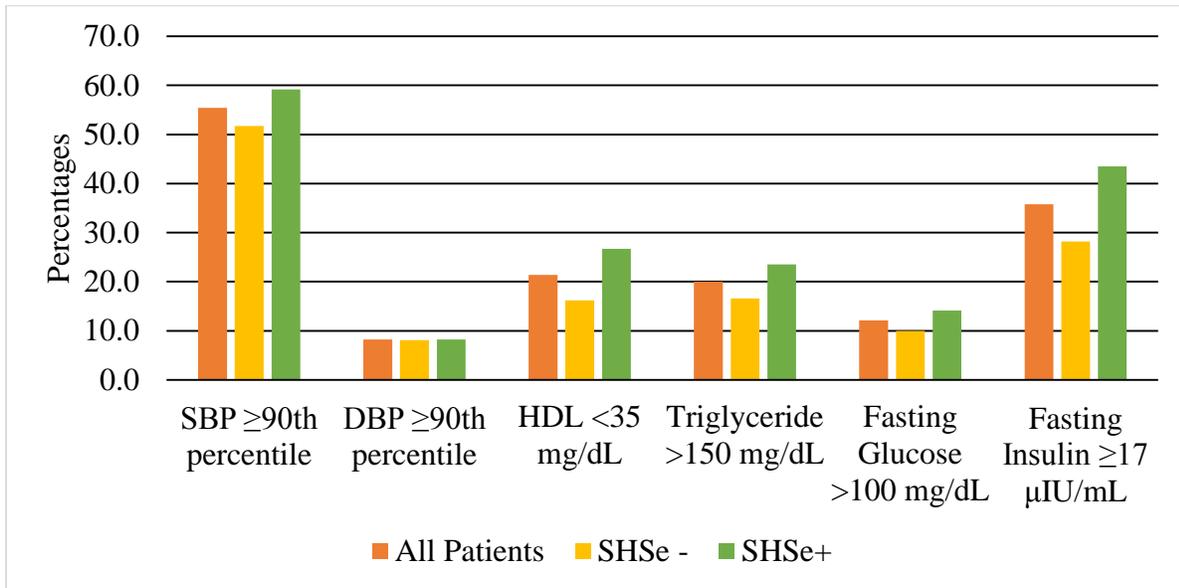


Figure 4. Metabolic abnormalities in all patients and by secondhand smoke exposure.

In addition to examining the percentage of patients who met each of the defined risk factors (Table 3), the mean or median (depending on distribution) for each value was calculated.

Table 13 shows the mean or median for each value for all patients, as well as by secondhand

smoke exposure classification. Fasting insulin and triglycerides were right skewed. For these values, the median and interquartile range are given. The metabolic levels were more abnormal for all risk factors, with the exemption of diastolic blood pressure, for the patients with secondhand smoke exposure. There is a statistically significant difference between secondhand smoke exposure and no exposure for systolic blood pressure ( $p=0.018$ ) and percentile ( $p=0.016$ ), fasting insulin ( $0.001$ ), triglycerides ( $p=0.010$ ), and HDL ( $p=0.016$ ).

Table 13

*Continuous Components in all Patients and by Secondhand Smoke Exposure*

	All Patients	SHSe-	SHSe+	p-value
Systolic Blood Pressure (mmHg)	118 ± 11	117 ± 11	119 ± 12	0.018*
Systolic Blood Pressure Percentile	92.2 ± 20.1	90.6 ± 20.3	93.8 ± 19.6	0.016 <sup>‡</sup>
Diastolic Blood Pressure (mmHg)	67 ± 7	67 ± 7	67 ± 7	0.488*
Diastolic Blood Pressure Percentile	67.8 ± 21.0	65.8 ± 19.4	69.1 ± 21.7	0.169 <sup>‡</sup>
Fasting Glucose (mg/dL)	92 ± 7	92 ± 6	93 ± 8	0.377 <sup>†</sup>
Fasting Insulin (μIU/mL)	13.6 ± 11.7	12.7 ± 10.1	15.5 ± 12.5	0.001 <sup>‡</sup>
Triglycerides (mg/dL)	93 ± 81	85 ± 72	100 ± 86	0.010 <sup>‡</sup>
HDL (mg/dL)	43 ± 10	44 ± 10	41 ± 11	0.016*

Note: \*p-value calculated using t-test with equal variances; <sup>†</sup>p-value calculated using t-test with unequal variances; <sup>‡</sup>p-value calculated using Mann-Whitney test.

### Discussion

The results of the study show that secondhand smoke exposure is associated with metabolic syndrome in young children. To the best of the researcher's knowledge, this is the first study demonstrating this association, increasing the odds of having metabolic syndrome by

2.2 (95% CI: 1.5-3.3). Age and BMI also had an association with secondhand smoke exposure, but a one year or one percentile increase did not have the same effect as the presence of secondhand smoke (Table 8). A one year increase in age increased the odds of metabolic syndrome by 1.13 (95% CI: 1.1-1.5), and a one percentile increase in BMI increased the odds by 1.5 (95% CI: 1.3-1.8). However, when age was increased by two or more years, or BMI by two or more percentile, those associations with metabolic syndrome became stronger than secondhand smoke (see Tables 9 and 10).

Secondhand smoke has a metabolic impact on children, even at this young age, but the mechanism as to how secondhand smoke has the effect is yet to be fully understood. Previous studies have shown a link between secondhand smoke exposure and insulin resistance. As previously mentioned, secondhand smoke exposure has been associated with insulin resistance in adults (Xie et al., 2010). However, a study conducted in 2011 found that children with frequent secondhand smoke exposure had a 24% increase in insulin resistance compared to children without secondhand smoke exposure, and the rate of insulin resistance increased with the increasing number of cigarettes smoked in the home (Thiering et al., 2011).

Insulin resistance subsequently is associated with cardiovascular disease risk and atherosclerosis because it increases the inflammatory process. Normally, insulin is an inflammation inhibitor in the body, but when resistance develops, insulin is unable to suppress inflammatory molecules, which in turn furthers leads to the development of fatty streaks and atherosclerosis (Dandona, Aljada, Chaudhuri, Mohanty, & Garg, 2005; Steinberger et al., 2009; Ten & Maclaren, 2004). In addition, insulin resistance contributes to elevated blood pressure because the increased level of insulin causes more sodium to be retained (Ten & Maclaren, 2004). Though insulin resistance cannot account for all increases in metabolic syndrome in this

patient population, the most common risk factors were elevated systolic blood pressure and elevated fasting insulin levels, which correlate with the proposed mechanisms in the literature.

Secondhand smoke exposure has also been associated with decreased HDL levels. A study in 2005 examined the effects of secondhand smoke on cardiovascular disease and determined that nonsmokers with frequent exposure to secondhand smoke had similar decreases in HDL levels as compared to smokers. Even short-term exposures to secondhand smoke resulted in lower HDL levels (Barnoya & Glantz, 2005). Secondhand smoke has also been associated with increased lipid storage in the liver, which includes triglycerides. A study conducted in 2009 demonstrated that secondhand smoke cause increased lipid synthesis in the liver of mice, which led to increased atherosclerosis and non-alcoholic fatty liver disease in the mice (Yuan, Shyy, & Martins-Green, 2009).

### **Public Health Implications and Prevention**

Exposure to secondhand smoke affects young children's health, beyond just respiratory disease. If the process of atherosclerosis is instigated or accelerated in children due to secondhand smoke, it is entirely possible that the exposure of secondhand smoke in addition to metabolic syndrome would increase the risk of cardiovascular disease in adult life. Therefore, it is imperative that action be taken to prevent such outcomes in children, particularly young children.

Research suggests that only half of physicians, whether they are pediatricians or family physicians, actually ask about smoking habits of household members. That rate diminishes when examining how many practitioners educate parents/guardians about the harms of secondhand smoke and how many counsel parents/guardians about how to quit smoking (Winickoff et al., 2003). These rates are extremely unfortunate because it is estimated that practitioners have

direct contact with approximately 25% of U.S. smokers during their patient's appointments. A child has an average of ten visits during the first two years of life, providing ample opportunity for physicians to screen for secondhand smoke exposure, and counsel for smoking cessation (American Academy of Pediatrics, 2016).

A study conducted in 2002 examined the barriers pediatricians faced when screening for secondhand smoke exposure as well as parents' attitudes about secondhand smoke screening by pediatricians. The most common barriers given by pediatricians included insufficient time, insufficient knowledge or confidence on smoking cessation counseling, and concerns about negative reactions from parents about smoking cessation counseling. The authors subsequently interviewed 341 parents for their opinion on secondhand smoke screening at their child's appointments. The vast majority (89%) of parents, both nonsmokers and smokers, felt screening for secondhand smoke exposure was an important component of a child's health visit and screening. Approximately 81% of parents thought pediatricians should educate about the effects of secondhand smoke on their children. However, when asked about pediatricians providing information on smoking cessation, only 56% felt it was appropriate. When this question was posed to the parents who did smoke, 52% responded positively to receiving counseling on smoking cessation, and only 15% of parents actually said they would be angry if their pediatrician were to counsel them on smoking cessation (Cluss & Moss, 2002).

An unfavorable reaction from a parent is a likely reason many physicians do not screen for a child's secondhand smoke exposure. Fortunately, it is not a common reaction, according to the above study, and if done correctly, secondhand smoke screening and counseling can improve the health of the child. There are several resources available for physicians to learn about the best methods to approach such a screening and counseling with parents, including motivational

interviewing and the *Don't Be Silent About Smoking* movement (TalkToYourPatients.org, 2013). Generally, parents and guardians who receive education about secondhand smoke and advice about quitting from their child's physician have higher quit attempts (American Academy of Pediatrics, 2016).

In addition to encouraging physicians to screen for secondhand smoke exposure in their pediatric patients, it is important for physicians to understand the full effect of secondhand smoke exposure in the pediatric population. The most commonly listed concerns of secondhand smoke-related disease in children are often respiratory diseases: asthma, bronchiolitis, and otitis media, due to the ear canal being more directly connected to the respiratory tract in young age (Aligne & Stoddard, 1997). The results of this study demonstrate a metabolic effect in children from secondhand smoke, which may worsen cardiovascular disease risk. Physicians should be more aware of these metabolic effects as they can provide better education to parents/guardians and, hopefully, stronger motivation for the parents/guardians to quit.

Fortunately, there are many additional efforts being made in order to decrease secondhand smoke exposure in all populations. At this point in time, there are thirty-six states that have passed some form of smoking ban in public places including workplaces, restaurants, bars, or gambling establishments (American Nonsmokers' Rights Foundation, 2016a). In addition, seven states have also passed smoke-free car laws in order to protect passengers from secondhand smoke exposure in a confined space like a car (American Nonsmokers' Rights Foundation, 2016b). While it was previously mentioned that Healthy People 2020 has specific goals to reduce secondhand smoke exposure in children, there are goals to increase the smoke-free legislation in the U.S. Examples of such smoke-free legislations supported by Healthy People 2020 include daycare centers, public transportation, hotels, hospitals, college campuses,

and multiunit housing. Healthy People 2020 (2016a) is also promoting more smoke-free homes. As most secondhand smoke exposure for children occurs in the home, increasing smoke-free homes is imperative (Yi et al., 2012). All of these efforts will hopefully lead to Healthy People 2020 (2016a) surpassing its goal of a ten percent improvement in secondhand smoke exposure in children.

### **Limitations**

The selection of patients involved in this study were predominantly White and the subgroups remained predominantly White when divided by secondhand smoke exposure (see Figure 1) or presence of metabolic syndrome (see Figure 2). Approximately half of the patients were found to have secondhand smoke exposure (49.6%). The sample of patients for this study is not truly representative of the population in the U.S. because the study population had a greater proportion of patients with secondhand smoke exposure as compared to the national average, which is estimated at 41.3% for children aged three to 11 (Healthy People 2020, 2015). Additionally, Hispanic children are more likely to have exposure to secondhand smoke than White or Black children as according to national data (Singh et al., 2010), but Table 5 and Figure 1 shows the majority of studied patients with secondhand smoke exposure were White. The White patients were also found to have greater rates of metabolic syndrome, as shown in Table 7 and Figure 2. Because metabolic syndrome is so closely tied to elevated BMI, it would be predicted that Black or Hispanic children would have greater rates of metabolic syndrome as these children have greater rates of obesity compared to White children, according to national trends (Barlow & Expert Committee, 2007). Though the sample was not truly representative of the U.S. population, metabolic syndrome was found to have a significant association with

secondhand smoke exposure, which was found to be true even when accounting for confounding factors like BMI and age.

There are several limitations to this study, the most prominent being the secondhand smoke exposure data. The data was collected by asking if mother, father, or anyone else in the household smoked. In order to be classified as no smoking exposure, the responses for mother and father had to be “no”, and the answer for anyone could be “no” or left blank. In contrast, to be classified as having exposure to secondhand smoke, a minimum of one “yes” response was required. If the patient was missing responses for mother or father, and the remaining responses were “no”, the patient could not be classified. There is also, of course, the possibility that parents were not truthful in their responses. In order to improve upon this study, it would be best to evaluate secondhand smoke exposure via a biomarker such as cotinine levels in biological samples such as hair, saliva, blood or urine. Cotinine, a metabolite of nicotine, is a reliable indicator not only of exposure but also extent or quantification of exposure (Park et al., 2014).

In addition to improving the secondhand smoke data, it would be preferred to standardize the time in which the lab data is collected from the visit to the Lipid Clinic at Dayton Children’s Hospital. The lab data was only included if it was conducted within six months of the appointment date. While the majority of patients completed their lab data within two months of their initial appointment at the lipid clinic, it would be better if the lab samples were taken the day of the visit to the Lipid Clinic. This would ensure the most accurate results as the data would reflect the metabolism of the patient on the same day as the height, weight, BMI, and blood pressure were measured. Taking the laboratory samples that day would also prevent any influences from lifestyle changes in the time from the appointment to the lab sample collection.

Lastly, activity and diet were not assessed for in this study. Instead, it was presumed that patients at or above the 85<sup>th</sup> percentile likely had more sedentary lifestyles, and thus similar diet and exercise habits. Some would suggest, however, that parents who are smokers are less attentive to good health practices, thus children of these parents may have worse diets or exercise habits. Secondhand smoke exposure would therefore be a confounding factor to the lifestyle of the patient in determining the likelihood of metabolic syndrome. While this may be true, the literature suggests secondhand smoke would still have a negative metabolic effect on these children. However, in order to improve this study, it would be best to collect information on diet and exercise habits and quantify that information in order to determine the influence on metabolic syndrome.

### **Future Research**

Additional studies are needed to truly understand the mechanism of secondhand smoke on the various metabolic processes.

### **Conclusion**

Obesity, tobacco use, and secondhand smoke continue to be public health concerns in the U.S, and methods to reduce these concerns have been outlined by Healthy People 2020 (2016a, 2016b). Childhood obesity has increased, more than doubling from 7% in 1980 to 18% in 2012 (CDC, 2014). Secondhand smoke exposure in children has declined in recent years, but estimates suggest roughly 40% of children continue to have regular exposure (Healthy People 2020, 2015; Quinto et al., 2013). These rates are of particular concern when examining metabolic syndrome in children. Metabolic syndrome is most associated with obesity, but associations with secondhand smoke exposure have also been established in adults and children (Weitzman et al., 2005; Xie et al., 2010).

This study determined that an association between metabolic syndrome and secondhand smoke exposure does exist in young children aged six to 11. Results suggest that secondhand smoke increases the odds of metabolic syndrome by 2.2 (95% CI: 1.5-3.3). Secondhand smoke increases inflammation, insulin resistance, triglyceride levels, blood pressure, and lower HDL (Barnoya & Glantz, 2005; Dandona et al., 2005; Steinberger et al., 2009; Thiering et al., 2011; Yuan et al., 2009), all of which are involved with metabolic syndrome. While the exact mechanism is unknown, it is predicted that secondhand smoke exposure would worsen the cardiovascular risk of metabolic syndrome in children aged six to 11. However, further studies are necessary to confirm this prediction.

It is crucial that health care providers be more proactive about limiting exposure to secondhand smoke in pediatric patients. Regardless of the presence of metabolic syndrome, this study found that patients exposed to secondhand smoke were more likely to have abnormal systolic blood pressure, fasting insulin, triglycerides, and HDL levels. In order to minimize their risk of cardiovascular disease, as well as other diseases, secondhand smoke screening, secondhand smoke education, and smoking cessation counseling need to be conducted more frequently.

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Appendix A: IRB Exemption Letter



**Office of Research and Sponsored Programs**  
201J University Hall  
3640 Col. Glenn Hwy.  
Dayton, OH 45435-0001  
(937) 775-2425  
(937) 775-3781 (FAX)  
e-mail: [rsp@wright.edu](mailto:rsp@wright.edu)

**DATE:** February 29, 2016

**TO:** Alexandra Lawson, PI, Student  
Public Health  
Sabrina Neeley, Ph.D., Faculty Advisor

**FROM:** Robyn Wilks  
Coordinator, IRB-WSU

A handwritten signature in black ink, appearing to read "RW", written over the printed name "Robyn Wilks".

**SUBJECT:** SC# 6116

*'Prevalence of Metabolic Syndrome and Second Hand Smoke Exposure in Children with Obesity'*

The above-listed project does not meet the Federal definition for human subjects research, specifically "a systematic investigation designed to contribute to generalizable knowledge". Therefore, the project does not require approval from the Wright State University Institutional Review Board.

If you have any questions or require additional information, please contact me at 775-4462.

Best wishes for a successful project.

## Appendix B: Statement of Permission from Dayton Children's Hospital

**Dayton Children's Hospital IRB**  
**One Children's Plaza**  
**Dayton, Ohio 45404-1815**  
(937) 641-4218

January 26, 2016

Ms. Alexandra Lawson  
MD/MPH Candidate  
Wright State University  
Boonshoft School of Medicine  
Dayton, Ohio 45435  
Email: lawson.124@wright.edu

RE: Your request dated 1/25/2016 / *Dayton Children's Hospital IRB Permission to Proceed*  
Dayton Children's reference number **2016-003**: Prevalence of Metabolic Syndrome and Second  
Hand Smoking Exposure in Children with Obesity

Dear Ms. Lawson:

This is in response to your request for permission to use the described data for the above-listed  
project.

Items reviewed:

- Memo request, dated 1/25/2016
- Protocol, submitted 1/25/2016
- HIPAA De-Identification Certification Form, dated 1/26/2016

You are granted permission to proceed with the use of the described de-identified existing data  
set from Dayton Children's Hospital for this project effective immediately.

Please contact Bev Comer (937-641-4218; fax 937-641-3201; email:  
ComerB@childrensdayton.org) if you have any questions or require further information.

Sincerely,

  
William Spohn, MD, CIP  
Chair, Institutional Review Board

Appendix C: List of Competencies Met in CE

**Tier 1 Core Public Health Competencies**

<b>Domain #1: Analytic/Assessment Skills</b>
Identifies quantitative and qualitative data and information (e.g., vital statistics, electronic health records, transportation patterns, unemployment rates, community input, health equity impact assessments) that can be used for assessing the health of a community
Applies ethical principles in accessing, collecting, analyzing, using, maintaining, and disseminating data and information
Uses information technology in accessing, collecting, analyzing, using, maintaining, and disseminating data and information
Selects valid and reliable data
Selects comparable data (e.g., data being age-adjusted to the same year, data variables across datasets having similar definitions)
Identifies gaps in data
Collects valid and reliable quantitative and qualitative data
Describes public health applications of quantitative and qualitative data
Uses quantitative and qualitative data
Describes assets and resources that can be used for improving the health of a community (e.g., Boys & Girls Clubs, public libraries, hospitals, faith-based organizations, academic institutions, federal grants, fellowship programs)
Describes how evidence (e.g., data, findings reported in peer-reviewed literature) is used in decision making
<b>Domain #2: Policy Development/Program Planning Skills</b>
Identifies current trends (e.g., health, fiscal, social, political, environmental) affecting the health of a community
Gathers information that can inform options for policies, programs, and services (e.g., secondhand smoking policies, data use policies, HR policies, immunization programs, food safety programs)
Describes implications of policies, programs, and services
<b>Domain #3: Communication Skills</b>
Solicits input from individuals and organizations (e.g., chambers of commerce, religious organizations, schools, social service organizations, hospitals, government, community-based organizations, various populations served) for improving the health of a community
Conveys data and information to professionals and the public using a variety of approaches (e.g., reports, presentations, email, letters)
Communicates information to influence behavior and improve health (e.g., uses social marketing methods, considers behavioral theories such as the Health Belief Model or Stages of Change Model)
Describes the roles of governmental public health, health care, and other partners in improving the health of a community
<b>Domain #4: Cultural Competency Skills</b>
Describes the diversity of individuals and populations in a community
Describes the ways diversity may influence policies, programs, services, and the health of a community
<b>Domain #5: Community Dimensions of Practice Skills</b>
Describes the programs and services provided by governmental and non-governmental organizations to improve the health of a community
Recognizes relationships that are affecting health in a community (e.g., relationships among health departments, hospitals, community health centers, primary care providers, schools, community-based organizations, and other types of organizations)
Supports relationships that improve health in a community
Provides input for developing, implementing, evaluating, and improving policies, programs, and services
<b>Domain #6: Public Health Sciences Skills</b>
Retrieves evidence (e.g., research findings, case reports, community surveys) from print and electronic sources (e.g., PubMed, Journal of Public Health Management and Practice, Morbidity and Mortality Weekly Report, The World Health Report) to support decision making
Recognizes limitations of evidence (e.g., validity, reliability, sample size, bias, generalizability)
Describes evidence used in developing, implementing, evaluating, and improving policies, programs, and services
Contributes to the public health evidence base (e.g., participating in Public Health Practice-Based Research Networks, community-based participatory research, and academic health departments; authoring articles; making data available to researchers)
Suggests partnerships that may increase use of evidence in public health practice (e.g., between practice and academic organizations, with health sciences libraries)

<b>Domain #7: Financial Planning and Management Skills</b>
Uses evaluation results to improve program and organizational performance
<b>Domain #8: Leadership and Systems Thinking Skills</b>
Incorporates ethical standards of practice (e.g., Public Health Code of Ethics) into all interactions with individuals, organizations, and communities
Describes the ways public health, health care, and other organizations can work together or individually to impact the health of a community
Contributes to development of a vision for a healthy community (e.g., emphasis on prevention, health equity for all, excellence and innovation)
Describes needs for professional development (e.g., training, mentoring, peer advising, coaching)