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Association of Secondhand Tobacco Smoke and Abdominal Adiposity in the United States Population

Omar T. Tahtamooni
Wright State University - Main Campus

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Association of Secondhand Tobacco Smoke and Abdominal Adiposity

in the United States Population

Omar T. Tahtamooni, MBBS

Wright State University

2016

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Table of Contents

Abstract.....	4
Introduction.....	5
Purpose Statement.....	5
Literature Review.....	6
Morbidity and Mortality Associated with SHS	6
Temporal Trends to SHS Exposure in the US	7
SHS Exposure by Race/Ethnicity and Gender.....	7
Global SHS Prevalance.....	9
SHS Exposure Assessment	9
SHS Exposure and Metabolic Syndrome.....	10
SHS Exposure and Obesity.....	10
SHS Exposure and Type 2 Diabetes Mellitus (DM).....	11
Methods.....	12
Statistical Analysis.....	13
Results.....	16
Discussion.....	23
Conclusion.....	26
References.....	27
Appendix A: List of Competencies Met in CE.....	32

Abstract

Background: Secondhand smoke (SHS) exposure occurs when people inhale smoke from tobacco products or inhale smoke that has been expelled by other smokers. Exposure to SHS increases the risk of many metabolic and chronic diseases, such as cardiovascular disease.

Purpose: To evaluate the association between SHS and abdominal adiposity in US adults.

Methods: Data from 2011-2012 National Health and Nutrition Examination Survey (NHANES) was used to examine the relationship between SHS exposure and abdominal adiposity, Sagittal Abdominal Diameter [SAD]), after adjusting for covariates (age, gender, ethnicity, and income) (n=4,012). Univariate linear regression analyses were used to predict association of smoking exposure, SAD and each of the covariates in separate models.

Multivariable linear regression analyses were used to assess independent association of smoking categories with SAD, after adjusting for covariates.

Results: Overall, males, non-Hispanic Blacks, and individuals with low income constituted the highest prevalence of those exposed to SHS. A positive significant association was found between SHS exposure and increasing SAD ($p<0.001$).

Conclusion: The highest exposure to SHS was among males, non-Hispanic Blacks, and individuals with low and middle income. Also, there was a significant association between SHS exposure and increasing SAD.

Keywords: exposure, chronic disease, metabolic, outcome, cardiovascular disease

Association of Secondhand Tobacco Smoke and Abdominal Adiposity in the United States Population

Secondhand smoke (SHS) exposure occurs when people inhale smoke from tobacco products or inhale smoke that has been expelled by other smokers. SHS may occur in homes, workplaces, and public places (restaurants, bars, in cars, or other vehicles) (Centers for Disease Control and Prevention (CDC), 2016). Tobacco smoke contains more than 4,000 chemicals, of which at least 250 are known as harmful or carcinogenic, such as hydrogen cyanide, carbon monoxide, ammonia, cadmium, arsenic, benzene, and beryllium (National Cancer Institute, 2011). Globally, more than 600,000 premature deaths per year were reported as a result of SHS in 2011 (National Cancer Institute, 2011).

Not only are adults exposed to SHS, but children can also breathe smoke from burning tobacco products. A study shows that about 40% of children are exposed to SHS in their homes (Pagani, Nguyen, & Fitzpatrick, 2016). In 2004, 28% of child deaths were related to SHS (World Health Organization [WHO], 2016).

Many studies have provided evidence regarding the association between SHS and chronic diseases, such as cardiovascular diseases (Pagani et al., 2016). The published literature shows that breathing SHS has harmful effects on the heart and blood vessels. Between 2005 and 2009 SHS exposure caused more than 7,300 lung cancer deaths among adult nonsmokers in the United States (US) (CDC, 2016).

Purpose Statement

The purpose of this study was to evaluate the association between SHS and abdominal adiposity in adult US residents.

Literature Review

Morbidity and Mortality Associated with SHS

Exposure to SHS increases the risk of all-cause death. According to the CDC, about 41,000 deaths were related to the SHS exposure in the United States in 2016. These deaths primarily resulted from lung cancer and heart disease. Out of these 41,000 deaths, about 7,000 deaths were from lung cancer, and 34,000 deaths resulted from heart disease per year (CDC, 2016).

Exposure to SHS increases the risk of lung cancer by 20% to 30% among nonsmokers (U.S. Department of Health and Human Services, 2014). Also, people who have coronary heart disease are at higher risk of heart attack as a result of continuous exposure to SHS (U.S. Department of Health and Human Services, 2016).

Among children and adolescents, exposure to SHS increases the respiratory illness in the United States. A watershed study conducted by United States Environmental Protection Agency (EPA) in 1993 stated that SHS “present a serious and substantial public health impact” (Anonymous, 1993, p. 71). It showed that exposure to SHS, especially indoor exposure, increased the risk of lower respiratory tract infection such as bronchitis and pneumonia (EPA, 1993). In addition, exposure to SHS increased the prevalence of other diseases such as chronic otitis media and increased the risk of having asthmatic symptoms (EPA, 1993). A review of available data showed that about 150,000 to 300,000 respiratory cases per year were in infants and children aged up to two years of age (EPA, 1993). Further, approximately 200,000 to one million children had asthma exacerbation when exposed to SHS (EPA, 1993). In a more recent study, Reh, Higgins, and Smith (2012) noted that

exposure to SHS increased the risk of chronic rhinosinusitis that is associated with chronic headache.

Temporal Trends in SHS Exposure in the US

Cotinine measurement shows that exposure to SHS has gradually decreased between 1988 and 2012 (CDC, 2016). Between 1988 and 1991 (Figure 1), among American aged 20 years, about 87.9% showed evidence of tobacco smoke exposure; that number fell to 40.1% during 2007 and 2008, and to 25.3% during 2011 and 2012 (CDC, 2016).

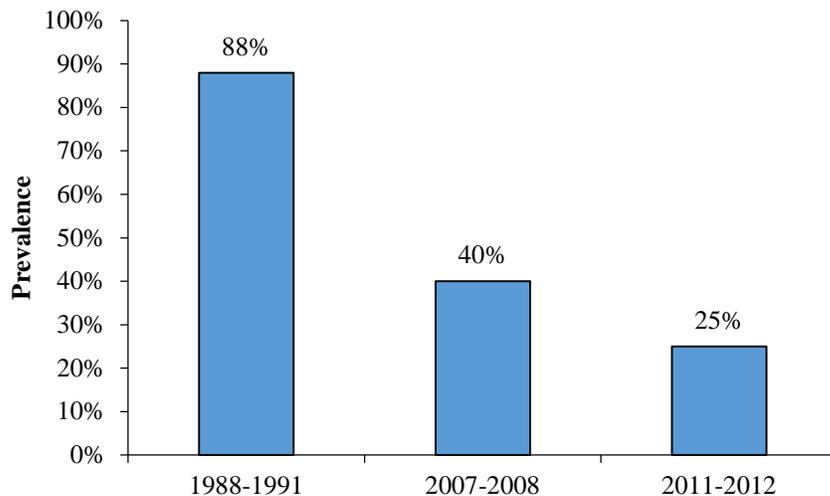


Figure 1. Secondhand smoke exposure rate for US, 1988–2012.

SHS Exposure by Race/Ethnicity and Gender

In the US, SHS exposure varies by race: African-Americans have the highest rate of SHS exposure (47%), followed by Mexican-Americans (22%), and Caucasians (22%) (CDC, 2016). A 2015 report by CDC described the average cotinine serum level by age group and race/ethnicity (Figure 2). The report demonstrated that between the year 2011 and 2012 the cotinine serum level was the highest (78.4%) among Non-Hispanic Black children aged three to 11 years, intermediate among Non-Hispanic White children (44.4%), and the lowest among

Mexican-American children (39.4%). For the age group of 12 to 19 years old, the pattern of mean cotinine serum levels was the same: 66.2% among Non-Hispanic Black children, 43% among Non-Hispanic White children, and 26.9% among Mexican-American children. For the age group of 20 years old and older, the serum level was again highest for Non-Hispanic Blacks (46.6%), lowest among Non-Hispanic Whites (21.9%), and intermediate for Mexican-Americans (31.4%) (CDC, 2015b). The report did not mention any potential reasons for this change of pattern.

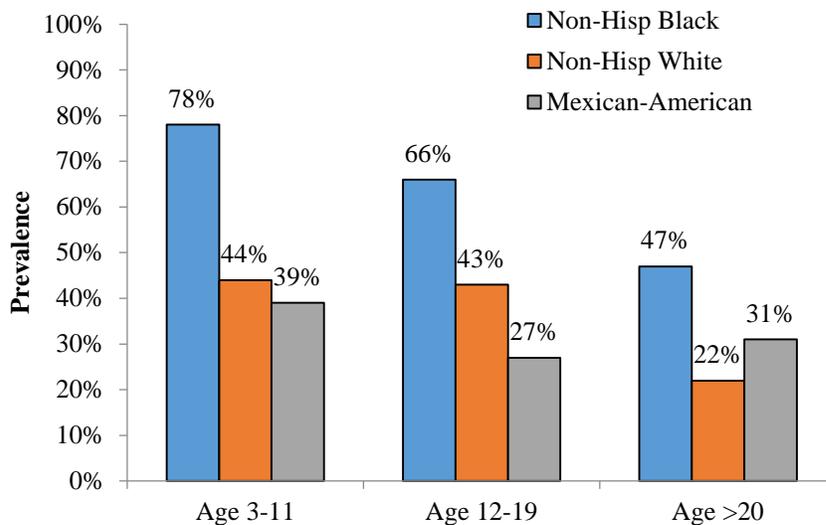


Figure 2. Serum cotinine level (ng/ml) by age and ethnicity.

SHS exposure also varies by gender, with higher prevalence rates of SHS in males than females. Women's Health USA (2011) reported that SHS exposure among men in the United States was 41.6%, while in women it was 33.4%. However, women are more susceptible to more serious illness and have a higher risk of death due to SHS exposure (Women's Health USA, 2011). According to the World Health Organization (2010), 46% of deaths due to SHS exposure were among women.

Global SHS Prevalence

SHS exposure is a major global public health concern. For instance, Collins and Lapsley (2005) claimed that SHS is a major factor contributing to ischemic heart disease, which is responsible for approximately 90% of Australia's deaths. Their study shows that between 2004 and 2005, exposure to SHS caused the death of 141 Australians (113 cases among adults and 28 death cases among infants) (Collins & Lapsley, 2005). The WHO (2004) stated that prevalence of SHS exposure for children ranges of low to 12% in Africa to high 68% in Western Pacific.

SHS Exposure Assessment

According to Whirl-Carrillo and colleagues (2012), nicotine in bloodstream is metabolized into different identifiable metabolites in the liver. One of the most important metabolites is cotinine because about 70% to 80% of nicotine is converted to cotinine (Whirl-Carrillo et al., 2012). For smokers, tobacco smoke is inhaled into the lungs where the gas exchanged and the nicotine goes to the bloodstream and it is metabolized into cotinine. Smokers also exhale nicotine to the environment and other people can inhale it again. For non-smokers nicotine can enter the body by inspiring polluted oxygen, into the lungs where the gas is exchanged and the nicotine is metabolized into cotinine (CDC, 2010).

According to CDC (2016), exposure to SHS can be measured by analysis of cotinine levels in body fluids, such as saliva, urine, or blood. Measuring cotinine is considered the most reliable assay to determine the degree of exposure to nicotine in tobacco smoke among smokers and nonsmokers who are exposed to SHS (CDC, 2016). Serum cotinine level of less than 1 ng/mL is used to identify for non-smokers; levels of 1-10 ng/mL are found in people exposed to SHS, and level of more than 10 ng/ml are found for smokers (Hukkanen, Jacob, &

Benowitz 2005). In addition, cotinine as a blood biomarker is preferred in measurement than nicotine because it remains in the human body for about 16 hours (CDC, 2016).

SHS Exposure and Metabolic Syndrome

Exposure to SHS may increase the risk of metabolic syndrome (MetS), a cardiometabolic condition of great public health concern. MetS is a combination of risk factors including high blood pressure, raised blood glucose, high cholesterol levels, and deposition of abdominal fat. MetS increases a person's chance of heart disease, diabetes and stroke. More than 34% of Americans have MetS (Pagani et al., 2016).

MetS has been shown to influence many biomarkers of cardiovascular inflammation and early disease. Specifically, exposure to SHS is associated with adverse cardio-metabolic profile. Pagani, Nguyen, and Fitzpatrick (2016) showed that SHS exposure among German children aged 10 years increased their level of plasma of leptin, C-reactive protein, fibrinogen, triglycerides, and interleukin-6. A study by Barnoya and Glantz (2005) showed similar effect of SHS on the cardiovascular system. These effects were comprised of inflammation biomarkers, plaque instability, endothelial dysfunction, atherosclerosis, decreased HDL levels, increased oxidized low density lipoprotein (LDL), increased oxidative stress, decreased energy metabolism, increased insulin resistance, decreased heart rate, increased arterial stiffness, increased risk of coronary disease events, and endocrine disruption.

SHS Exposure and Obesity

There is a strong body of evidence that shows that exposure to SHS is associated with metabolic effects including developing obesity (Barnoya & Glantz, 2005; Moore et al., 2015). This effect is noted in adults and also in children. More recent research focuses on additional

variables that affect this association. Moore et al. (2015) built upon previous findings of an association between SHS exposure and obesity in children by studying the effect of diet on the association between SHS exposure and obesity. The study showed that in the United States, children and adolescents aged six to 19 years of age who were exposed to SHS with low measure of dietary quality were more obese than children with SHS exposure and higher dietary quality. Moreover, Capul-Uicab et al. (2012) found that exposure to SHS in utero increased the risk of being obese among adolescent and adult women aged 14 to 47 years.

Body mass index (BMI) is a measure of body size based on weight and height. However, the BMI is not always an accurate indicator of obesity (Pagani et al., 2016). For that reason, many studies use waist circumference (WC) and/or Sagittal Abdominal Diameter (SAD) to measure the distribution of body fat (Pagani et al., 2016). According to De Souza and de Oliveira (2013), SAD is a valid measure of visceral obesity (the amount of fat in the gut area). WC and SAD are commonly used as abdominal adiposity measurement due to many benefits, such as lower costs, ease of measurement, non-invasiveness, and easier to implement in clinical practice.

Long term SHS exposure among young children (≤ 10 years) was related to increased waist circumference and BMI (Pagani et al., 2016). In addition, children who were continuously exposed to SHS had WC between three to five inches greater than children who were not exposed to SHS; their BMIs were also between 0.48 kg/m^2 and 0.81 kg/m^2 higher by the age of 10 years (Pagani et al., 2016).

SHS Exposure and Type 2 Diabetes Mellitus (DM)

It has been consistently shown that there is association between smoking and diabetes mellitus (Willi, Bodenmann, Ghali, Faris, & Cornuz, 2007). As interest in SHS grew,

researchers also investigated potential associations between SHS and diabetes in non-smokers. Eze and colleagues (2014, Abstract, Background) state that “only few recent studies have shown environmental tobacco smoke (ETS) to be associated with DM and never-smokers”. The literature shows that cotinine from long term exposure to SHS has a harmful impact on vital systems, including the endocrine system (Pagani et al., 2016). According to Eze et al. (2014), exposure to SHS associated with insulin resistance, reduced insulin sensitivity, and glucose intolerance. They reported that SHS exposure increased DM risk by 50% in never-smokers. Another study supported this proposed relationship between exposure to SHS and DM type 2: Lajous et al. (2013) showed that exposure to SHS is associated with chronic pancreatitis (inflammation of the pancreas), glucose metabolism disorder, and insulin resistance. Their analysis of a sample of (37,343) women non-smokers who were exposed to SHS in childhood concluded that the risk of DM was 18% higher for women who had childhood SHS exposure.

There are many studies that explain the mechanism that could be involved in the effect of SHS on diabetes. Xie, Liu, Wu, and Waku (2009) study showed that exposure to SHS may cause insulin resistance by affecting insulin action. Also, Bruin et al. (2008) mentioned that exposure to SHS was associated with loss of pancreatic β -cells based on animal studies. Chuang et al. (2011) found that exposure to SHS might have a chronic toxic impact on the pancreas, especially among children, which increases the risk of pancreatic cancer and associated with DM.

Methods

Data were obtained from National Health and Nutrition Examination Survey (NHANES) for the years 2011 and 2012. Data were used to examine the relationship between

the exposure to SHS and SAD after adjusting for covariates; age, gender, body mass index (BMI), ethnicity, and income.

Serum cotinine (ng/ml) the metabolite of nicotine was measured by an isotope dilution-high performance liquid chromatography. Based on serum levels of cotinine participants were categorized into three groups non-smokers (<1 ng/ml), SHS (1-10 ng/ml), and smokers (>10 ng/ml).

Average SAD (cm) was measured for participants eight years and older using the following protocol. In supine position (participant lying down), an abdominal caliper was used to measure the external distance between the front of the abdomen and the small of the back at the iliac level line. Age at the time of participant screening ranged from zero to 80 years. Ethnicity was classified as Non-Hispanic White, Non-Hispanic Black, Non-Hispanic Asian, and Others (Mexican-American, other Hispanic, other Multi-Racial). Gender was categorized as male or female. Annual family income was collected as a range value in dollars and categorized as low income (<25,000), middle income (25,000-54,999), and high income (\geq 55,000).

Statistical Analysis

Descriptive Statistics

To characterize the study participants, descriptive statistics were calculated, including means and standard deviation (SD) for continuous variables and counts and percentages for categorical variables. Comparisons among smoking exposure groups were completed using ANOVA for the continuous variables and Chi-square (χ^2) tests for the categorical variables.

Linear Regression

Univariate linear regression analyses were used to measure association of SAD and smoking status for each individual risk factor in separate models (Figure 3). This was done in order to test for statistically significant association between each covariate and SAD: only significant co-factors will be included in the multivariate model. Categorical variables were dummy-coded to create reference groups as shown in Table 1. Dummy variables allowed for multiple comparisons in a single multivariate model while controlling experimentwise error.

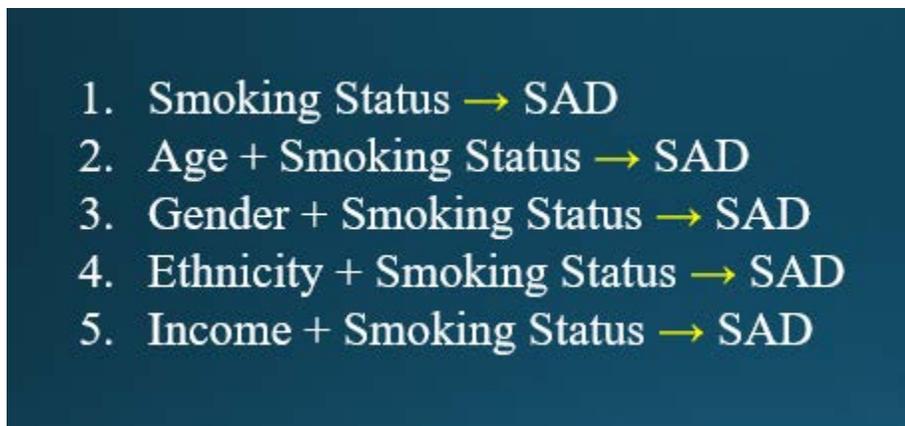
- 
1. Smoking Status → SAD
 2. Age + Smoking Status → SAD
 3. Gender + Smoking Status → SAD
 4. Ethnicity + Smoking Status → SAD
 5. Income + Smoking Status → SAD

Figure 3. Individual univariate models.

Table 1

Dummy Coding and Reference Groups for Linear Regression

Dummy Variable Name	Dummy Variable Value	Reference Groups
Non-Hispanic White	1=White	Others: (Mexican American, other Hispanic, and other Multi Racial)
	0=Others	
Non-Hispanic Black	1=Black	
	0=Others	
Non-Hispanic Asian	1=Asian	
	0=Others	
Males	1=Males	Females
	0=Females	
Low income	1=Low income	High income
	0=Others	
Middle income	1=Middle income	
	0=Others	
SHS	1=SHS	Non-Smokers
	0=Others	
Smokers	1=Smokers	
	0=Others	

Multivariate linear regression analysis was used to measure independent association of SAD with smoking exposure categories for the significant covariates identified in the univariate models. A single multivariate model was constructed. This model utilized the dummy coded variables used in the univariate models. Statistical analyses were performed using SPSS software. (SPSS: IBM Corp. Released 2013 IBM SPSS Statistics for Windows,

Version 22.0. Armonk, NY). Statistical significance for hypothesis tests were set at $\alpha = 0.05$ (two tailed).

Results

Descriptive Statistics

Descriptive statistics were generated for demographic variables overall and by smoking status groups (Table 2). Statistical differences among smoking categories were tested using χ^2 analysis for categorical variables and ANOVA for continuous variables. Significant differences across smoking status group were found for all variables listed in Table 2.

Table 2

Characteristics of 2011-2012 NHANES Participants, Overall, and by Smoking Status

Characteristic, n (%), mean \pm SD	Overall n=4,013	Non-SMK n=2,907	SHS n=152	SMK n=954	p-value
Gender					
Male	2,030 (50.6)	1,334 (45.9)	94 (61.8)	602 (63.1)	<0.001
Female	1,983 (49.4)	1,573 (54.1)	58 (38.2)	352 (36.9)	
Ethnicity					
NH White	1,557 (38.8)	1,065 (36.6)	51 (33.6)	441 (46.2)	<0.001
NH Black	987 (24.6)	641 (22.1)	49 (32.2)	297 (31.1)	
NH Asian	541 (13.5)	481 (16.5)	12 (7.9)	48 (5.0)	
Others	928 (23.1)	720 (24.8)	40 (26.3)	168 (17.6)	
Annual household income					
<25,000	1,277 (31.8)	788 (27.1)	58 (38.2)	431 (45.2)	<0.001
25,000-54,999	1,172 (29.2)	829 (28.5)	52 (34.2)	291 (30.5)	
\geq 55,000	1,564 (39.0)	1,290 (44.4)	42 (27.6)	232 (24.3)	
Sagittal Abdominal Diameter (cm)	22.78 \pm 4.41	22.69 \pm 4.38	23.95 \pm 5.06	22.85 \pm 4.34	0.003
Age (years)	48.54 \pm 16.91	49.93 \pm 17.25	41.36 \pm 15.91	45.45 \pm 15.25	<0.001

^a p-values for difference between smoking status using ANOVA (continuous variables) or chi-square test (categorical variables)

Gender, Ethnicity, and Income by Smoking Status Categories

Overall, males and females each constituted approximately half of sample. When comparing smoking status by gender, more males reported SHS or active smoking than females: more females reported non-smoking status ($p < 0.001$) (Figure 4).

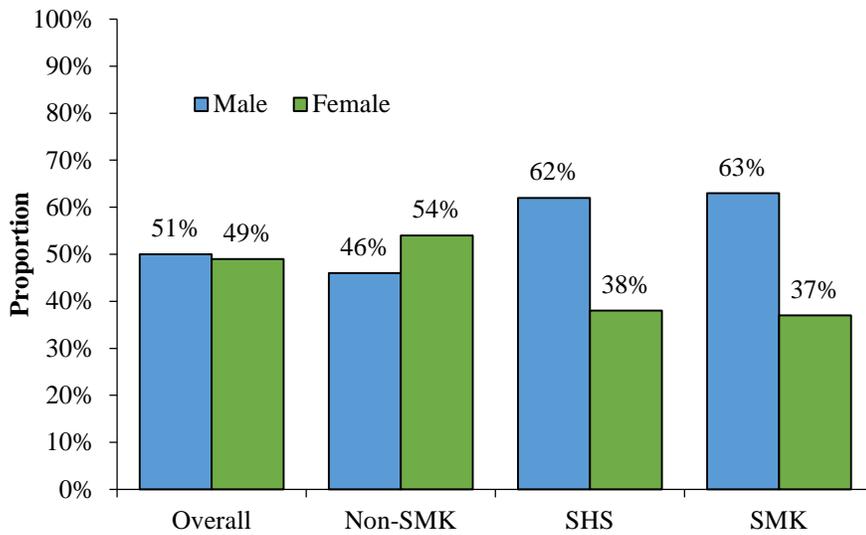


Figure 4. Smoking status by gender (p<0.001).

When comparing the prevalence of smoking status by gender, the prevalence of SHS and smoking was higher in males: females had higher prevalence of non-smoking status (p<0.001) (Figure 5).

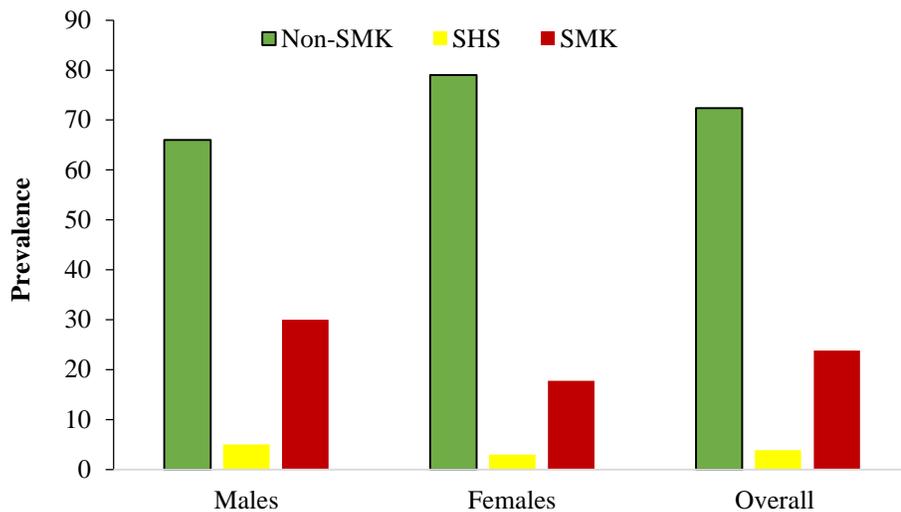


Figure 5. Prevalence of smoking by gender (p<0.001).

In overall analysis, non-Hispanic White participants constituted the highest proportion of the sample by ethnicity (39%). Additionally, by smoking status the same ethnic group

constituted the highest proportion among non-smokers, active smokers, and those exposed to SHS (Figure 6).

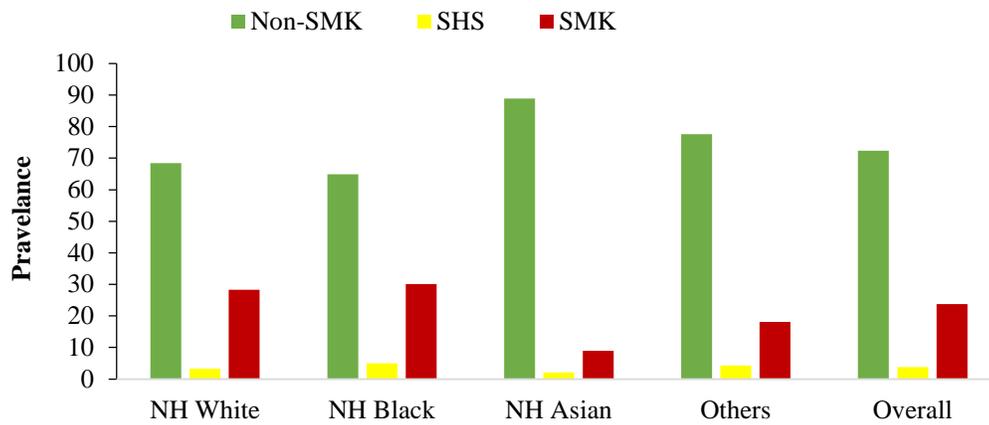


Figure 6.

Non-Hispanic Black participants had the highest prevalence of SHS exposure (5.0%) followed by Others (4.3%). Non-Hispanic Blacks and non-Hispanic Whites had the highest prevalence of active smoking (30% and 28%, respectively) (Figure 7).

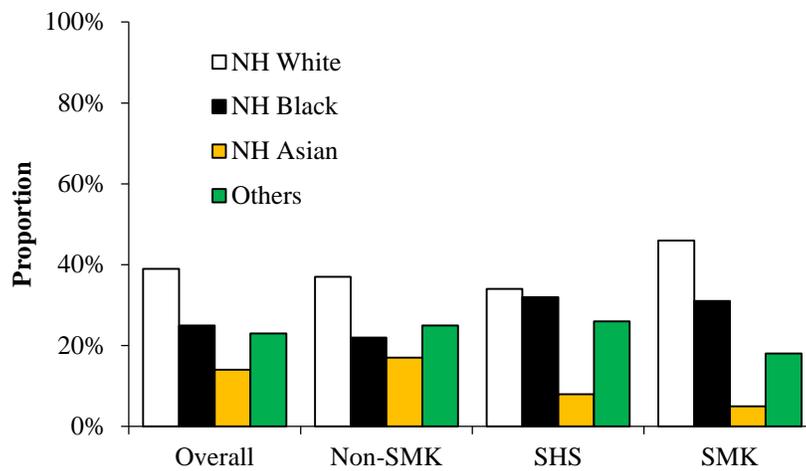


Figure 7. Prevalence of smoking by ethnicity (p<0.001).

Overall, based on annual household income, 39% of participants had high income (\$55,000 or more per year). By smoking status, more individuals with high income were non-smokers. However, among those exposed to SHS and active smokers the highest proportion was constituted by individuals with less than 25,000 per year income (Figure 8).

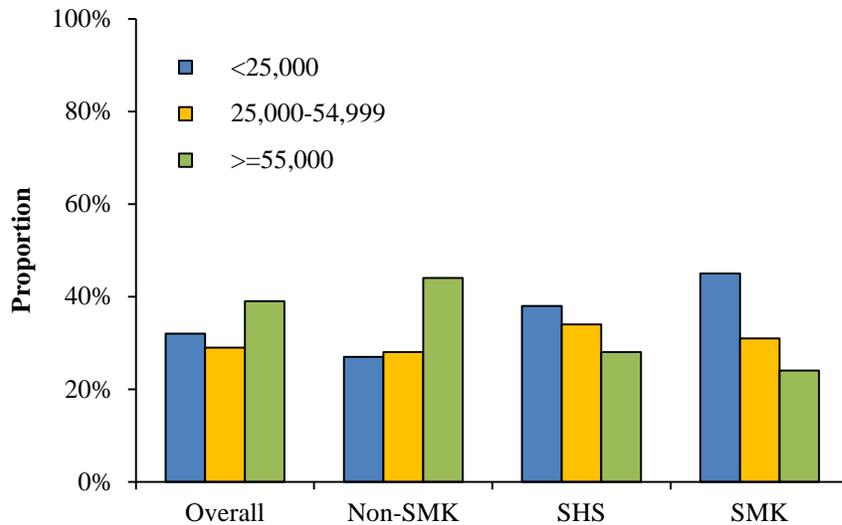


Figure 8. Smoking status by income ($p < 0.001$).

The highest prevalence of SHS exposure was among low (4.5%) and middle income (4.4%) groups. The highest prevalence of smoking was exhibited by individuals with less than 25,000 per year income (Figure 9).

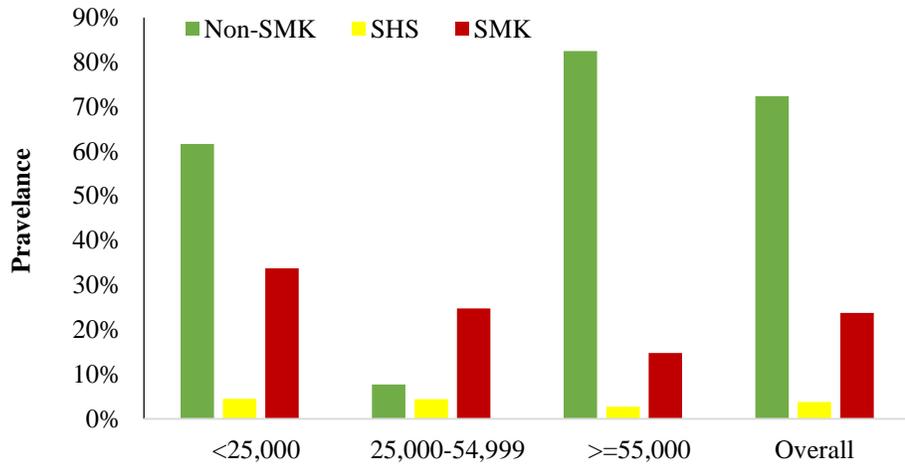


Figure 9. Prevalence of smoking by income (p<0.001).

Sagittal Abdominal Diameter and Age by Smoking Categories

Overall, the mean sagittal abdominal diameter was 22.78±4.41 centimeters. By smoking status, the highest mean SAD was seen among SHS exposed (p=0.003): this was four percent (4%) higher than active smokers and five percent (5%) higher than non-smokers (Figure 10).

Overall, mean age in years was 48.54±16.91. The lowest mean age was seen among SHS exposed (p<0.001).

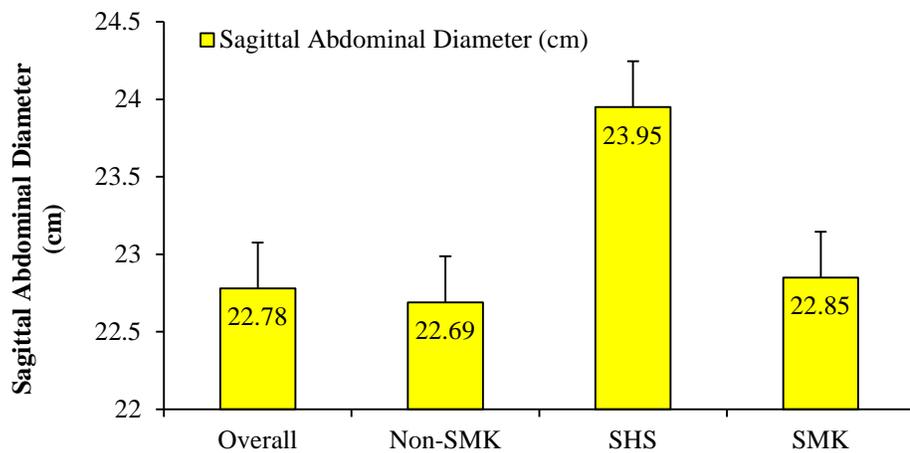


Figure 10. Sagittal abdominal diameter (cm) by smoking status (p=0.003).

Univariate Linear Regression

The univariate regression analysis evaluated the relationship between SAD and smoking status using separate models adjusting for a single covariate (age, gender, ethnicity, and income).

A positive significant association was found between SHS exposure and SAD ($p < 0.001$) (Table 3). The un-adjusted mean SAD was 1.25 cm greater among individuals who were exposed to SHS than active smokers. The association between active smoking status and SAD was not statistically significant ($p = 0.350$).

Table 3

*Univariate Model Showing Association between Sagittal Abdominal Diameter (cm), Smoking Exposure Categories, and other Covariates among 2011-2012 NHANES Participants Overall, and by Smoking Status**

Variables		B	SE B	β	p-value	95% CI
Smoking	Constant	22.700	0.082		<0.001	(22.54, 22.86)
	SHS	1.252	0.367	0.054	0.001	(0.53, 1.97)
	Smokers	0.154	0.164	0.015	0.350	(-0.16, 0.47)
Age in years at screening	Constant	19.823	0.218		<0.001	(19.39, 20.25)
	SHS	1.746	0.359	0.076	<0.001	(1.04, 2.45)
	Smokers	0.412	0.161	0.040	0.011	(0.09, 0.72)
Gender	Constant	22.227	0.103		<0.001	(22.02, 22.43)
	SHS	1.088	0.365	0.047	0.003	(0.37, 1.80)
	Smokers	-0.023	0.165	-0.002	0.887	(-0.34, 0.30)
Ethnicity	Constant	22.989	0.142		<0.001	(22.71, 23.26)
	SHS	0.841	0.350	0.036	0.016	(0.15, 1.52)
	Smokers	-0.330	0.159	-0.032	0.038	(-0.64, -0.01)
Income	Constant	22.100	0.114		<0.001	(21.87, 22.32)
	SHS	1.062	0.365	0.046	0.004	(0.34, 1.77)
	Smokers	-0.089	0.166	-0.009	0.593	(-0.41, 0.23)

*Note: Nonsmokers were the reference group; their values are shown on the constant row.

Age was significantly associated with SAD in both SHS exposed and active smokers ($p < 0.001$). For gender, there was a significant association between being male and SAD in the SHS exposure group ($p = 0.003$). In contrast, in male active smokers, this relationship was not significant ($p = 0.887$). When we compared the dummy coded ethnicity variables (non-Hispanic White, non-Hispanic Black, and non-Hispanic Asian) with our reference group (others), SAD was significantly associated with SHS exposure ($p = 0.016$). In addition, SAD was also significantly associated with active smoking status after adjusting for ethnicity ($p = 0.038$) ($p = 0.004$). SAD and income were significantly associated for the non-smoking group ($p < 0.001$) and SHS group ($p = 0.004$). No significant association between SAD and income was seen for active smokers ($p = 0.593$).

Multivariate Linear Regression

Multivariate regression analysis evaluated relationship between SAD and smoking status adjusting for all covariates (age, gender, ethnicity, and income). As shown in Table 4, there was a significant positive association between SAD and exposure to SHS ($p = 0.004$). In contrast, a negative association between SAD and active smoking was noted ($p = 0.009$) after adjusting for all covariates. No significant association between smoking status and SAD was found for non-Hispanic Whites ($p = 0.339$). A negative association was found between smoking status and SAD for non-Hispanic Asians.

Table 4

*Multivariate Model Showing Association between Sagittal Abdominal Diameter (cm), Smoking Exposure Categories, and other Covariates, 2011-2012 NHANES Participants**

Variables		B	SE B	β	P-value	95% CI
Independent variable	Constant	19.635	0.251		<0.001	(19.14, 20.12)
	Smoking					
	SHS	0.982	0.343	0.043	0.004	(0.31, 1.65)
	Smokers	-0.418	0.161	-0.040	0.009	(-0.73, -0.10)
Covariates						
	Gender					
	Male	1.127	0.130	0.128	<0.001	(0.87, 1.38)
	Income					
	Low-Income	0.812	0.159	0.086	<0.001	(0.50, 1.12)
	Middle-Income	0.634	0.160	0.065	<0.001	(0.32, 0.94)
	Ethnicity					
	NonHisp-White	-0.164	0.171	-0.018	0.339	(-0.50, 0.17)
	NonHisp -Black	1.164	0.188	0.114	<0.001	(0.79, 1.53)
	NonHisp -Asian	-3.024	0.223	-0.234	<0.001	(-3.46, -2.58)
	Age in years at screening	0.049	0.004	0.188	<0.001	(0.04, 0.05)

*Note: Reference groups were as follows: Smoking: nonsmokers; Gender: female; Income: high income; Ethnicity: Others (Mexican-American, other Hispanic, and other Multi-racial).

Discussion

Our study sought to evaluate the association between SHS and abdominal adiposity in adult US residents. Our findings show that more males, non-Hispanic Whites, and people with low income were exposed to SHS than their counterparts. The highest prevalence of SHS exposure (number exposed relative to sample size) was among males, non-Hispanic Black individuals, and those with low and middle income. Multivariate analyses showed a significant positive association was seen between SHS exposure and SAD. In fact, the highest mean SAD

was seen in SHS-exposed individuals. We discuss our other findings with regard to specific covariates below:

Gender

This study shows that more males (61.8%) than females (38.2%) exhibited SHS exposure. This exposure was higher than the that reported in the Women`s Health USA report (2011), which found that (41.6%) of males were exposed to SHS in the United States. More males (63.1%) smoked than females (36.9%). In this study, using cotinine serum levels to categorize individuals with regard to smoking status created discrete separation between smokers and people expose to SHS. In everyday life smokers are not just smoke, but also they expose to SHS from their own and from other smoke because smokers often smoke with other.

This high SHS exposure among males needs to be addressed by implementing strategies to change smoking behavior among males and further reduce the routes of exposure to SHS as well.

Ethnicity

This study shows that the highest prevalence of SHS exposure was among Non-Hispanic Blacks (5.0%) followed by Others (4.3%), and the highest prevalence of active smoking was among Non-Hispanic Blacks and non-Hispanic Whites (30% and 28%, respectively).

By smoking status, non-Hispanic White participants constituted the highest proportion (33.6%) of those exhibiting SHS exposure. However, according to the CDC report (2015a), the highest SHS exposure (46.8%) is seen among non-Hispanic Black. This difference might be explained by the fact that the CDC report (2015a) includes all ages group of ≥ 3 years old (CDC, 2015b).

The multivariate linear regression showed no association between SAD and smoking status in non-Hispanic Whites; this might be explained by the racial differences in functional

activity of CYP2A6 enzyme. According to Tanner et al. (2016), the functional CYP2A6 enzyme allele metabolizes nicotine and cotinine and removes them from the system. Functional CYP2A6 enzyme allele frequencies for non-Hispanic Whites range from zero to four percent (0-4%), and for non-Hispanic Blacks the range from zero to two (0-2%) (PharmGKB, 2001-2016). Therefore, the cotinine in blood in non-Hispanic White removed faster than in non-Hispanic Blacks. This means that there was not seen an association between SHS exposure and SAD among non-Hispanic Whites. Nicotine and cotinine do not disrupt the metabolism as much in Whites because it remains in the system for a short time.

Income

Individuals with low income showed a significant association with increased SAD among SHS exposure ($p < 0.001$). The same result was also found in the CDC report (2015a), which demonstrated that SHS exposure was higher among individuals with low incomes, and that about 43% of nonsmokers with below-poverty income were exposed to SHS.

SAD and SHS

Our results show that the highest mean SAD was seen among individuals exposed to SHS. This result was also reported by Pagani et al., 2016 and Moore et al., 2015, who found that the long term exposure to SHS was related to greater levels of SAD. Additionally, our univariate and multivariate linear regression analysis showed a significant positive association between exposure to SHS and mean SAD. This finding is in accordance with the prior research studies that concluded that exposure to SHS is associated with obesity (Barnoya & Glantz, 2005).

As shown in Figure 10, there was not a dose response pattern for cotinine level and SAD: Based on the higher mean SAD for SHS group compared to the non-smoking group, one would expect the smoking group would have a higher mean SAD than the SHS group. Instead, they have a

lower mean SAD the SHS group. This may be explained by patterns of endocrine response disruption. Endocrine system is operated by hormones. The small dose of exposure has an effect, even for low levels of cotinine exposure leads to endocrine disruption that reduces the breaking down of visceral fat. At the high levels of cotinine in blood for smokers, the receptors may stop working appropriately which results in lower SAD accumulation (Tweed, Hsia, Lutfy, & Friedman, 2012).

Strengths and Limitations

The strengths of the study are that NHANES data are a nationally representative sample and that their smoking status assessment was based upon the measurements of the serum levels of cotinine. Cotinine is considered the most reliable assay to determine the degree of exposure to nicotine in tobacco smoke among smokers and nonsmokers who are exposed to SHS (CDC, 2016). A limitation of this study was our sample selection, which omitted people with missing data. If they had been included, that could affect the outcomes of our analysis.

Conclusion

We conclude that the highest exposure to SHS was among males, non-Hispanic Blacks, and individuals with low and middle income. Also, there was a significant association between SHS exposure and SAD.

We recommend that these findings be used to increase the awareness among the highest risk groups, including males, non-Hispanic Blacks, and individuals with low and middle income. In addition, public health efforts should work to create new plans and policies that help to protect these groups from this negative health behavior.

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Appendix A

Wright State Program Public Health Competencies Checklist

Assess and utilize quantitative and qualitative data.
Apply analytical reasoning and methods in data analysis to describe the health of a community.
Describe how policies, systems, and environment affect the health of populations.
Communicate public health information to lay and/or professional audiences with linguistic and cultural sensitivity.
Make evidence-informed decisions in public health practice.
Demonstrate ethical standards in research, data collection and management, data analysis, and communication.

Concentration Specific Competencies Checklist

Emergency Preparedness:
Use research and/or evaluation science methodologies and instruments to collect, analyze and interpret quantitative and qualitative data
Demonstrate an understanding of the protection of worker health and safety

Global Health:
Exhibit interpersonal skills that demonstrate willingness to collaborate, trust building abilities, and respect for other perspectives
Conduct evaluation and research related to global health
Apply systems thinking to analyze a diverse range of complex and interrelated factors shaping health at local, national, and international levels