



HHS Public Access

Author manuscript

J Adolesc Health. Author manuscript; available in PMC 2019 April 01.

Published in final edited form as:

J Adolesc Health. 2018 April ; 62(4): 463–470. doi:10.1016/j.jadohealth.2017.10.001.

Tobacco Smoke Exposure Association with Lipid Profiles and Adiposity Among U.S. Adolescents

Ashley L. Merianos, PhD, CHES^a, Roman A. Jandarov, PhD^b, Jane C. Khoury, PhD^c, and E. Melinda Mahabee-Gittens, MD, MS^d

^aSchool of Human Services, University of Cincinnati, Cincinnati, OH, USA

^bDivision of Biostatistics and Bioinformatics, Department of Environmental Health, College of Medicine, University of Cincinnati, 162 Kettering Lab Building, 160 Panzeca Way, Cincinnati, OH, 45267-0056, USA, Phone: 513-558-7975

^cDivision of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center, College of Medicine, University of Cincinnati, 3333 Burnet Avenue, Cincinnati, OH, 45229, USA, Phone: 513-636-3690

^dDivision of Emergency Medicine, Cincinnati Children's Hospital Medical Center, College of Medicine, University of Cincinnati, 3333 Burnet Avenue, MLC 2008, Cincinnati, OH, 45229, USA, Phone: 513-636-7966

Abstract

Purpose—We investigated the association between tobacco smoke exposure (TSE) as measured by serum cotinine and lipoprotein cholesterols and adiposity in adolescents.

Methods—We performed a secondary analysis of 1999–2012 NHANES data including participants 12–19 years old. We examined TSE: unexposed (<0.05ng/mL), passively exposed (0.05–2.99ng/mL), and actively exposed (≥3ng/mL); lipid profiles: total cholesterol, HDL-C, non-HDL-C, LDL-C, and triglycerides; and adiposity: body mass index z-score (BMIZ), waist circumference (WC), and waist-to-height ratio (WHtR). Covariates were age, sex, race/ethnicity, income, diet, and physical activity. Multiple regression models were used to assess the association between TSE and lipid profile variables separately, and then TSE and adiposity measures separately, adjusting for covariates. We performed logistic regression to examine the association of TSE with BMI and WHtR classifications.

Results—Of the 11,550 participants, 41.7% were unexposed to tobacco smoke, 40.5% were passively exposed, and 17.8% were actively exposed. Compared to unexposed: participants with

Address correspondence to: Ashley L. Merianos, School of Human Services, University of Cincinnati, P.O. Box 210068, Cincinnati OH, 45221, USA, ashley.merianos@uc.edu, Phone: +01 513-556-6753, Fax: +01 513-556-3898.

Conflict of Interest: The authors have no potential conflicts of interest to disclose.

Implications and Contribution: Participants who were actively exposed to tobacco smoke had lower cholesterol and HDL-C, and higher triglycerides, BMIZ, WC, and WHtR. Participants who were passively exposed had lower HDL-C, and higher cholesterol, LDL-C, BMIZ, WC, and WHtR. TSE prevention efforts should start as early as childhood and continue throughout adolescence.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

active TSE had lower total cholesterol, lower HDL-C, and higher triglycerides; higher BMIZ, higher WC, and higher WHtR; participants with passive TSE had lower HDL-C, higher total cholesterol, and higher LDL-C; higher BMIZ, higher WC, and higher WHtR. Participants actively exposed were at increased odds of being obese or WHtR 0.65, and those passively exposed were at increased odds of being overweight, obese, or WHtR 0.65.

Conclusions—Active TSE and passive TSE are differentially associated with factors within the lipid profile and adiposity, independent of covariates. TSE prevention efforts should start as early as childhood and continue throughout adolescence and adulthood.

Keywords

smoking; tobacco smoke exposure; lipoprotein cholesterols; adiposity; adolescence

Tobacco smoke exposure (TSE) is a modifiable risk factor of cardiovascular disease and the leading cause of death and disability in the U.S. [1]. TSE includes active smoking which is smoking tobacco products and passive smoking which is inhalation of smoke from lit tobacco products. Nearly 90% of active adult smokers initiate smoking before 18 years of age [1,2]; of these young smokers, one-third will die from a tobacco-related cause including cardiovascular disease [2]. One-third (33.8%) of 12-19 year olds are passively exposed to tobacco smoke [3]. A review of case-control and cohort studies indicate passive TSE, as measured by self-report and biomarkers, increases cardiovascular disease risk by approximately 30% on average [4], leading to 33,951 heart disease-specific deaths annually [1].

There is an increasing emphasis being placed on adolescent cardiovascular health promotion with focus on primordial and primary prevention [5]. The American Heart Association [6] defines ideal cardiovascular health by four health behaviors (smoking, body mass index [BMI], physical activity, diet) and four health factors (smoking, total cholesterol, blood pressure, glycemia). Smoking is treated as both a health behavior and health factor due to the impact smoking can have on one's health [6]. Even with priority placed on avoiding the development of risk factors altogether [5], a growing body of evidence suggests cardiovascular disease begins in childhood and measurements of lipoprotein cholesterols and adiposity can predict this negative health consequence in adulthood [7]. It is important to focus on promoting ideal cardiovascular health in prevention programs that address multiple domains since risk factors for poor health are often related and ideally addressed through combined programs [8]. Further, prior work suggests assessing the relationships between ideal cardiovascular health metrics and outcomes to gain a better understanding of individual metrics that create one single index [5].

Though active and passive TSE have been causally associated with the development of cardiovascular disease in adulthood [1], recent research shows variations in this relationship among adolescents. Specifically, regarding serum lipoprotein cholesterols, studies among adolescents indicate that TSE is associated with higher low-density lipoprotein cholesterol (LDL-C) [9,10] and triglycerides [9,10], and lower high-density lipoprotein cholesterol (HDL-C) [9,11-14] and total cholesterol [10]. Conversely, some research reports no association between TSE and HDL-C [10], non-HDL-C [10], LDL-C [15], triglycerides

[15], and total cholesterol [10,15,16] in adolescents. Active TSE has also been linked to increased adiposity as measured by BMI and body fat [16]. Similarly, passive TSE has been associated with BMI percentiles indicative of obesity in adolescents [17]. More research is needed to elucidate the relationship between TSE, lipid profiles, and adiposity in adolescents to better inform prevention programs that focus on ideal cardiovascular health and address multiple related risk factors.

The present study investigates the association between TSE as measured by serum cotinine and serum lipoprotein cholesterol (total cholesterol, HDL-C, non-HDL-C, LDL-C, and triglycerides) in adolescents. We also sought to examine the association between TSE and adiposity (BMI, WC, and WHtR) as childhood adiposity strongly predicts cardiovascular risk in adulthood [7].

Methods

Study Design

We performed a secondary analysis of 1999-2012 National Health and Nutrition Examination Survey (NHANES) data including participants 12-19 years old. NHANES is a nationally representative survey conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics that continuously observes the health and nutrition status of U.S. adults and children. A university-based IRB deemed this study as exempt from review with a "not human subjects' research" determination.

Data Source and Population

The seven continuous NHANES cycles used a stratified, multistage probability design to select and recruit participants before completing household interviews and medical examinations encompassing laboratory measurements (e.g., blood samples collected using venipuncture) and physical examination components (e.g., weight). Prior to participation, informed consent was received from adolescents 18 years. For those <18 years, parental/guardian permission and child assent was received.

The present study included 11,550 participants who were interviewed and examined. Information was available on sociodemographics, diet, physical activity, total cholesterol, and adiposity measures for these participants. Since only a random subsample of participants completed the fasting lipid panel, we excluded participants missing data on HDL-C ($n=7,335$), non-HDL-C ($n=7,335$), LDL-C ($n=6,191$), and triglycerides ($n=6,190$), excluding a total of 9,728 participants; leaving 1,822 for analyses including lipid biomarkers. However, there were 11,517 available with total cholesterol. We compared the sociodemographics, diet, and physical activity of the participants included in all the lipids analyses and those who were not included (Table 1).

Independent and Dependent Variables

TSE, our independent variable, was assessed using the optimal biomarker serum cotinine [18], which was measured from the participants' blood collected by a phlebotomist using an isotope-diluted high performance liquid chromatography method. The detection limits of

cotinine have changed in NHANES over time. The limit was 0.05 ng/mL from 1999-2000. Both 0.05 ng/mL and 0.015 ng/mL were used in 2001-2002. The limit of detection was 0.015 ng/mL from 2003-2012. Thus, we used the higher detection limit of 0.05 ng/mL as the cut point for detectable cotinine. Based on prior recommendations [19,20], we used the following cotinine levels to classify participants: (1) no detectable cotinine (<0.05 ng/mL) as unexposed; (2); detectable cotinine of 0.05-2.99 ng/mL as passively exposed; and (3) detectable cotinine \geq 3 ng/mL as actively exposed with levels consistent with active smoking. The cut point of \geq 3 ng/mL is sensitive enough to detect nondaily or light smokers.

We assessed several continuous lipid profile outcome measures, which were measured using the CDC's Lipid Standardization Program for accuracy purposes. We examined total cholesterol, HDL-C, non-HDL-C, LDL-C, and triglycerides. Total cholesterol was measured from blood samples. A subsample of participants was selected at random with a specified sampling fraction to give a fasting blood sample in the morning after at least a nine-hour fast. HDL-C, non-HDL-C, LDL-C, and triglycerides were measured from these fasting blood samples. We calculated non-HDL-C by subtracting HDL-C from total cholesterol. Non-HDL-C is the biomarker for atherogenic apoB-containing lipoprotein concentration and is an understudied measure that may be a key predictor of cardiovascular risk in adulthood [21]. We used lipid concentrations defined by NHLBI [22] to interpret our findings. Acceptable lipid levels are defined as: <170 mg/dL for total cholesterol; >45 mg/dL for HDL-C; <120 mg/dL for non-HDL-C; <110 mg/dL for LDL-C; and <90 mg/dL for triglycerides.

During the physical examination, children's height, weight, and WC were measured. We used several measures to examine adiposity in children; BMI z-score (BMIZ) which was calculated using the LMS method and CDC's sex-specific BMI-for-age reference [23], WC, and WHtR. For secondary analyses, we categorized BMI as percentiles: (1) <85th percentile—underweight and normal weight; (2) 85th to <95th percentile—overweight; and (3) \geq 95th percentile—obese; and WHtR values: (1) <0.65 and (2) \geq 0.65 [24]. We selected these measures of adiposity as research indicates that BMI in childhood is a strong predictor of cardiovascular risk factors in adulthood [7]. Additionally, other research indicates that WC and WHtR are simple and effective proxy measures of abdominal adiposity [25], and are better predictors of cardiovascular disease risk in children than BMI [26]. Particularly in adolescents, BMI is a strong measure of subcutaneous adipose tissue, whereas WC is a strong measure of visceral adipose tissue [27].

Covariates

We selected covariates to include in the analysis; age, sex, race/ethnicity, annual household income level, diet, and physical activity. Racial/ethnic groups were: white, black, Hispanic, or other race. Income level was self-reported as: <\$20,000/year, \$20,000-\$44,999/year, \$45,000-74,999/year, and \geq \$75,000/year. Diet information was obtained from the day one dietary recall interview conducted during the medical examination portion. We only used data from day one for consistency since not all NHANES years included data on day two. We adjusted for the following diet variables that are used to determine intakes of calories, energy, saturated fat, and sodium: gram weight of food, energy, total fat, total saturated fatty

acids, total monounsaturated fatty acids, total polyunsaturated fatty acids, sodium, and alcohol. Physical activity levels included: none/light, moderate, and vigorous activity.

Statistical Analysis

We conducted statistical analyses using R version 3.3.0. Using NHANES analytical guidelines [28], we combined seven successive cycles. We applied appropriate examination sampling weights to obtain estimates generalizable to the U.S. adolescent population, while accounting for the complex NHANES design, survey non-response, and post-stratification. First, we examined differences between covariates (i.e., age, sex, race/ethnicity, income level, diet, physical activity) and TSE categories (i.e., unexposed, passively exposed, actively exposed) by performing χ^2 tests for categorical variables and ANOVAs for continuous variables. Categorical variables are presented as raw sample sizes with the weighted percent in parentheses and continuous variables are presented as weighted mean \pm weighted standard error (SE). We performed multiple linear regression modeling to assess the association between the categories of TSE and lipid profile variables separately. Least square means (LS Means) and SE are provided. We adjusted for covariates in most models: age, sex, race/ethnicity, income level, diet, and physical activity. We were unable to adjust for physical activity in the models that assessed HDL-C and non-HDL-C since all participants included were in the none/light physical activity level; but adjusted for all other covariates. To adjust for diet, we performed principal component analysis (PCA) on the scaled and centered dietary intake data before running each model. PCA is a multivariate data analysis method to reduce the dimensionality of the data by considering a smaller number of important variables, principle components scores. In PCA, principal components are constructed as linear combinations of the original variables in the data in such a way that they 1) are uncorrelated to each other, and 2) account for as much of the variability in the observed data as possible. We calculated the first two principal component scores of diet. We found these two scores explained 81% of the variability in the original diet data. These scores were then used in each model to represent the potentially confounding dietary patterns.

We performed multiple regression modeling to examine the relationship between TSE and adiposity measures (i.e., BMIZ, WC, WHtR), while adjusting for covariates. In secondary analyses, we built a multinomial logistic regression model to assess the association of TSE with BMI-percentile and a multivariable logistic regression model to examine the association of TSE with WHtR classifications. Adjusted odds ratios and 95% confidence intervals are presented. Analyses were two-sided and we used a *p*-value of $<.05$ to indicate statistical significance. In all analyses, incomplete cases were removed before applying the models.

Results

Of the 11,550 adolescents from 12-19 years old, mean age was 15.48 years (SE=0.02) and 51.2% ($n=5,902$) were males (Table 2). Over half (59.8%; $n=3,043$) were white and nearly one-third (31.8%; $n=2,228$) reported an annual income of \leq \$75,000/year. TSE levels indicated 41.7% ($n=4,571$) were unexposed to tobacco smoke (cotinine <0.05 ng/mL), 40.5% ($n=5,104$) were passively exposed (cotinine 0.05-2.99 ng/mL), and 17.8% ($n=1,875$)

were actively exposed (cotinine = 3 ng/mL). Participants had an average energy intake of 2,523.77kcal (SE=12.82), total fat intake of 84.20gm (SE=0.45), and sodium intake of 3,756.05mg (SE=18.96). Most participants engaged in none or light physical activity (81.6%), followed by moderate activity (12.8%), and vigorous activity (5.6%). Results indicated that age, sex, race/ethnicity, income level, gram weight of food intake, energy intake, sodium intake, alcohol intake, and physical activity level differed significantly (all $p < .001$) by categorized TSE level (see Table 2). The association between age and TSE is mostly driven by the higher age of adolescents in the actively exposed group. Sex differences show that there is a higher proportion of males in both the actively and passively exposed groups compared to the unexposed group. Comparing race/ethnicity, there is a high proportion of black children in the actively and passively exposed groups. An inverse relationship was found between income level and TSE; lower income participants were more likely to be actively exposed, and higher income participants in the unexposed group. Regarding diet, participants in the actively and passively exposed groups reported higher gram weight of food, energy, and alcohol intake than the unexposed group. Adolescents in the actively and passively exposed groups had relatively higher rates of none/light physical activity.

Adolescents in each TSE group had mean total cholesterol (<170 mg/dL), HDL-C (<45 mg/dL), and LDL-C (<110 mg/dL) within acceptable levels. Triglycerides were acceptable (<90 mg/dL), with the exception of those with active TSE who had a mean value within the borderline triglyceride levels (90-129 mg/dL). Table 3 presents multiple regression models of the association between TSE and lipid biomarkers. We found a significant difference between TSE levels for total cholesterol, while adjusting for the covariates. Participants with active TSE had lower total cholesterol ($\beta = -3.41$, $SE = 1.19$, $p = .004$), but participants with passive TSE had higher total cholesterol ($\beta = 2.09$, $SE = 0.89$, $p = .02$) compared with unexposed participants. HDL-C differed based on TSE exposure; participants with active TSE ($\beta = -2.29$, $SE = 0.81$, $p = .005$) and passive TSE ($\beta = -2.08$, $SE = 0.65$, $p = .001$) had lower HDL-C than those who were unexposed. Results also indicated that participants who were passively exposed to TSE ($\beta = 4.13$, $SE = 1.15$, $p < .001$) had higher LDL-C than unexposed participants. Participants with active TSE ($\beta = 7.87$, $SE = 3.53$, $p = .03$) had higher triglycerides than participants who were unexposed (see Table 3).

We found significant differences for adiposity measures between TSE groups (Table 4). Participants with active TSE ($\beta = 0.13$, $SE = 0.04$, $p = .002$) and passive TSE ($\beta = 0.22$, $SE = 0.03$, $p < .001$) had higher BMIZ compared with participants who were unexposed. Participants with active TSE ($\beta = 1.87$, $SE = 0.54$, $p < .001$) and passive TSE ($\beta = 2.51$, $SE = 0.40$, $p < .001$) had higher WC than unexposed participants. Results also indicated that participants with active TSE ($\beta = 0.013$, $SE = 0.003$, $p < .001$) and passive TSE ($\beta = 0.017$, $SE = 0.002$, $p < .001$) had higher WHtR than those unexposed to tobacco smoke.

Secondary analysis using logistic regression examining overweight and obesity as measured by BMI percentile revealed that participants with active TSE were more likely (OR=1.86, 95%CI=[1.51,2.30], $p < .001$) to be in the obese group compared to the underweight/normal weight group; no difference was found between the overweight and underweight/normal weight groups (Table 5). Participants who were overweight (OR=1.55; 95%CI=[1.38,1.74],

$p < .001$) and obese ($OR = 1.49$; $95\% CI = [1.26, 1.77]$, $p < .001$) were more likely to be passively exposed. We also found significant associations between TSE and WHtR. Participants with active TSE ($OR = 1.93$; $95\% CI = [1.59, 2.35]$, $p < .001$) and passive TSE ($OR = 2.01$; $95\% CI = [1.72, 2.36]$, $p < .001$) were at increased odds of having WHtR 0.65.

Discussion

Among adolescents in this study, TSE was associated with serum lipid biomarkers and adiposity measures that could be concerning for future cardiovascular risk. Although studies of the relationship between TSE and lipid profiles have yielded mixed findings [9-16], we found notable differences in lipid biomarkers for adolescents who were actively and passively exposed to tobacco smoke compared to those unexposed. Similar to prior research [9,11], those with active TSE had higher triglycerides, but lower total cholesterol and HDL-C. Adolescents with passive TSE had higher total cholesterol and LDL-C, and lower HDL-C [13,14]. No difference was found based on non-HDL-C, which paralleled prior work using NHANES 2005-2010 data [10]. It is important to note that all adolescents in each TSE group on average had acceptable lipid profile values, based on NHLBI [22] guidelines, with the exception of those with active TSE who had borderline high triglyceride levels. However, the differences revealed in the present study could give us more information into future trends of lipid profiles that could become clinically concerning and significant in adulthood. The present study highlights the importance of investigating metrics of ideal cardiovascular health in adolescence that may track into adulthood [5,6].

Our research largely expands on previous findings by using objective measurements of TSE and lipid profiles, while delineating risk between active TSE and passive TSE in a national sample. The study's findings suggest that active TSE and passive TSE influence lipid profiles in different ways, suggesting a complex relationship in adolescence. One possible explanation is the different toxins found in active TSE and passive TSE. Passive TSE consists of a mixture of mainstream smoke (i.e., smoke inhalation from cigarette butt) and sidestream smoke (i.e., between puffs) with higher concentration of toxic gases and small particles, which may have a more negative impact on cardiovascular health than mainstream smoke alone [29]. TSE is an important component to consider for ideal cardiovascular health as this concept treats TSE as both a modifiable health behavior and health factor [6], and the present study adds to the literature by using an objective measure and assessing both active and passive smoking. It is of importance to study both levels of exposure since active TSE and passive TSE are causally related to cardiovascular disease in adulthood [1,30], which remains the leading cause of death and disability in the U.S. [31]. A review of the literature revealed that brief passive TSE negatively influences the cardiovascular system (e.g., arterial stiffness, oxidative stress, heart rate variability, atherosclerosis) at rates as high (around 80%-90%) as those from chronic active TSE in adults [32]. The prevention and reduction of both active TSE and passive TSE, which are modifiable risk factors of cardiovascular risk, should be a key target of prevention programming for the adolescent population.

Of note, the current study found high rates of passive TSE (40.5%) and active TSE (17.8%) in adolescents, suggesting that nearly 60% had been exposed at detectable levels. Similar to national research conducted by Homa et al. [3], we found that as age increased, TSE levels

increased. We found that males had higher rates of active TSE compared to females. Passive TSE rates of males (40.5%) and females (40.4%) were similar. There was an inverse relationship between TSE and income. As income level increased, rates of active TSE and passive TSE decreased, which parallels prior literature [33]. Regarding diet, energy and sodium intake increased from the unexposed to actively exposed group. The estimated energy (2,931.36kcal) and sodium (3,815.01mg) in the active TSE group greatly exceeded the 2015-2020 Dietary Guidelines for Americans, which recommends energy intake up to 2,400kcal, depending on sex and physical activity, and 2,300mg of sodium. Similar to prior research [34], we found physical activity was inversely related to TSE.

Our findings indicate that elevated BMI, WC, and WHtR placed adolescents at increased risk of being actively and passively exposed to tobacco smoke. Examining the relationships between these metrics helps to assess overall cardiovascular health so that recommendations can be made to position adolescents to modify behaviors that will allow them to achieve ideal cardiovascular health. Prior research has typically investigated this relationship in adolescents by using BMI as the sole predictor of adiposity [17] or using clusters of cardiovascular risk [35,36]. For example, Camhi et al. [36] indicated that adolescents who are at increased risk of cardiometabolic risk factor clustering have a higher BMIZ, and that overweight active smokers are at increased likelihood of risk factor clustering. Other studies found that active smokers tend to have lower BMI but greater WC and increased abdominal fat compared with nonsmokers [37]. Our findings suggest it is important to consider WC and WHtR as indicators, especially since both adiposity measures may be better predictors of cardiovascular disease than BMI [26]. Our work emphasizes the importance of continuing early prevention efforts to decrease active TSE and passive TSE in adolescents [38]. Overall, the study's findings align with national recommendations [39] that underscore the need for tobacco control programs and obesity prevention programs targeting adolescents nationwide.

There are several limitations that should be considered while interpreting findings. The NHANES is cross-sectional and causal relationships cannot be established. Although serum cotinine is an objective TSE measure, random measurements can only identify TSE within a short period of time as cotinine has a half-life of approximately 16-17 hours [18]. Although we used cotinine cut points that are sensitive enough to distinguish between active smoking including light smoking and passive smoking [19,20], adolescents who were passively exposed could have potentially been active, nondaily smokers. We adjusted for potential confounders, but residual confounding may have occurred and unexpectedly biased our results.

Conclusion

We investigated the relationship between TSE and both serum lipoprotein cholesterol and adiposity measures in a national sample of adolescents. We report that active and passive exposure to tobacco smoke was uniquely associated with lipid profiles and adiposity, independent of covariates. Health promotion and prevention intervention efforts should start as early as childhood and continue throughout adolescence and adulthood in order to promote ideal cardiovascular health. Initiatives could be implemented in multiple venues including communities, schools, healthcare settings, and faith-based institutions [5].

Families should be extensively included in these efforts as they can play a central role in promoting ideal cardiovascular health. For example, caregivers can be educated on the importance of protecting their adolescents from passive smoking by implementing smoking bans inside and outside of the home and car. Programs could teach caregivers skills on how to effectively discuss the dangers of smoking with their youth. Further, families and adolescents could be educated on how to maintain a healthy weight to work towards ideal cardiovascular health. Though cardiometabolic risk factors in adults have been well defined over time [40], more research is still needed on the impact of active TSE and passive TSE on cardiovascular risk in adolescence. Longitudinal research starting in childhood may provide insight into how, at what level, and when active TSE and passive TSE influence cardiovascular risk over time.

Acknowledgments

Funded, in part, by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NIH Grant Number R01HD083354). The corresponding author affirms that she has listed everyone who contributed significantly to the work.

References

1. U.S. Department of Health and Human Services. The health consequences of smoking - 50 years of progress: a report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services Office of the Surgeon General; 2014.
2. U.S. Department of Health and Human Services. Preventing tobacco use among youth and young adults: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health; 2012.
3. Homa DM, Neff LJ, King BA, et al. Vital signs: Disparities in nonsmokers' exposure to secondhand smoke--United States, 1999-2012. *MMWR*. 2015; 64(4):103-108. [PubMed: 25654612]
4. Law MR, Wald NJ. Environmental tobacco smoke and ischemic heart disease. *Prog Cardiovasc Dis*. 2003; 46(1):31-38. [PubMed: 12920699]
5. Ford ES. Ideal cardiovascular health: start young, finish strong. *Circulation*. 2012; 125(16):1955-1957. [PubMed: 22452833]
6. Lloyd-Jones DM, Hong Y, Labarthe D, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic impact goal through 2020 and beyond. *Circulation*. 2010; 121(4):586-613. [PubMed: 20089546]
7. Srinivasan SR, Frontini MG, Xu J, Berenson GS. Utility of childhood non-high-density lipoprotein cholesterol levels in predicting adult dyslipidemia and other cardiovascular risks: the Bogalusa Heart Study. *Pediatrics*. 2006; 118(1):201-206. [PubMed: 16818566]
8. Werch CC, Moore MJ, DiClemente CC, Bledsoe R, Jobli E. A multihealth behavior intervention integrating physical activity and substance use prevention for adolescents. *Prev Sci*. 2005; 6(3):213-226. [PubMed: 16133900]
9. Craig WY, Palomaki GE, Johnson AM, Haddow JE. Cigarette smoking-associated changes in blood lipid and lipoprotein levels in the 8- to 19-year-old age group: a meta-analysis. *Pediatrics*. 1990; 85(2):155-158. [PubMed: 2136949]
10. Zakhari J, Amrock SM, Weitzman M. Passive and active tobacco exposure and children's lipid profiles. *Nicotine Tob Res*. 2016; 18(5):982-987. [PubMed: 26187391]
11. He B, Zhao S, Peng Z. Effects of cigarette smoking on HDL quantity and function: Implications for atherosclerosis. *J Cell Biochem*. 2013; 114(11):2431-2436. [PubMed: 23852759]
12. Le-Ha C, Beilin LJ, Burrows S, et al. Gender difference in the relationship between passive smoking exposure and HDL-cholesterol levels in late adolescence. *J Clin Endocrinol Metab*. 2013; 98(5):2126-2135. [PubMed: 23633198]

13. Hirata K, Yamano Y, Suzuki H, Miyagawa S, Nakadate T. Passive smoking is associated with lower serum HDL-C levels in school children. *Pediatrics Int.* 2010; 52(2):252–256.
14. Moskowitz WB, Schwartz PF, Schieken RM. Childhood passive smoking, race, and coronary artery disease risk: the MCV Twin Study. *Arch Pediatr Adolesc Med.* 1999; 153(5):446–453. [PubMed: 10323623]
15. Neufeld EJ, Mietus-Snyder M, Beiser AS, Baker AL, Newburger JW. Passive cigarette smoking and reduced HDL cholesterol levels in children with high-risk lipid profiles. *Circulation.* 1997; 96(5):1403–1407. [PubMed: 9315524]
16. Flouris AD, Faught BE, Klentrou P. Cardiovascular disease risk in adolescent smokers: evidence of a `smoker lifestyle. *J Child Health Care.* 2008; 12(3):221–231. [PubMed: 18678584]
17. Wang L, Mamudu HM, Alamian A, Anderson JL, Brooks B. Independent and joint effects of prenatal maternal smoking and maternal exposure to second-hand smoke on the development of adolescent obesity: a longitudinal study. *J Paediatr Child Health.* 2014; 50(11):908–915. [PubMed: 24920104]
18. Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev.* 1996; 18(2):188–204. [PubMed: 9021312]
19. Benowitz NL, Bernert JT, Caraballo RS, Holiday DB, Wang J. Optimal serum cotinine levels for distinguishing cigarette smokers and nonsmokers within different racial/ethnic groups in the united states between 1999 and 2004. *Am J Epidemiol.* 2009; 169(2):236–248. [PubMed: 19019851]
20. Merianos AL, Hossain MM, Khoury JC, Matt GE, Mahabee-Gittens EM. Serum cotinine and hemoglobin A1c among a national sample of adolescents without known diabetes. *Nicotine Tob Res.* 2017 Epub ahead of print.
21. Boekholdt SM, Arsenault BJ, Mora S, et al. Association of LDL cholesterol, Non-HDL cholesterol, and apolipoprotein B levels with risk of cardiovascular events among patients treated with statins: a meta-analysis. *JAMA.* 2012; 307(12):1302–1309. [PubMed: 22453571]
22. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents National Heart, Lung, and Blood Institute. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics.* 2011; 128(5):S213–S256. [PubMed: 22084329]
23. Kuczumski RJ, Johnson CL, Ogden CL, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat.* 2002; 11(246)
24. Saydah S, Bullard KM, Imperatore G, Geiss L, Gregg EW. Cardiometabolic risk factors among US adolescents and young adults and risk of early mortality. *Pediatrics.* 2013; 131(3):e679–e686. [PubMed: 23420920]
25. Lo K, Wong M, Khalechelvam P, Tam W. Waist-to-height ratio, body mass index and waist circumference for screening paediatric cardio-metabolic risk factors: a meta-analysis: screening cardio-metabolic risk factors. *Obes Rev.* 2016; 17(12):1258–1275. [PubMed: 27452904]
26. Savva SC, Tornaritis M, Savva ME, et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int J Obesity.* 2000; 24(11):1453–1458.
27. Brambilla P, Bedogni G, Moreno LA, et al. Crossvalidation of anthropometry against magnetic resonance imaging for the assessment of visceral and subcutaneous adipose tissue in children. *Int J Obes.* 2006; 30(1):23–30.
28. Johnson CL, Paulose-Ram R, Ogden CL, et al. National Health and Nutrition Examination Survey: analytic guidelines, 1999-2010 National Center for Health Statistics. *Vital Health Stat.* 2013; 2(161)
29. Raupach T, Schäfer K, Konstantinides S, Andreas S. Secondhand smoke as an acute threat for the cardiovascular system: a change in paradigm. *Eur Heart J.* 2006; 27(4):386–392. [PubMed: 16230308]
30. Venn A, Britton J. Exposure to secondhand smoke and biomarkers of cardiovascular disease risk in never-smoking adults. *Circulation.* 2007; 115(8):990–995. [PubMed: 17296856]
31. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics—2016 update: a report from the American Heart Association. *Circulation.* 2016; 133(4):e38–e360. [PubMed: 26673558]

32. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation*. 2005; 111(20):2684–2698. [PubMed: 15911719]
33. Hiscock R, Bauld L, Amos A, Fidler JA, Munafò M. Socioeconomic status and smoking: a review. *Ann N Y Acad Sci*. 2012; 1248(1):107–123. [PubMed: 22092035]
34. Ali MM, Amialchuk A, Heller LR. The influence of physical activity on cigarette smoking among adolescents: evidence from Add Health. *Nicotine Tob Res*. 2015; 17(5):539–545. [PubMed: 25187062]
35. Huntington-Moskos L, Turner-Henson A, Rice M. Tobacco exposure, weight status, and elevated blood pressure in adolescents. *J Community Health*. 2014; 39(4):653–659. [PubMed: 24519179]
36. Camhi SM, Katzmarzyk PT. Prevalence of cardiometabolic risk factor clustering and body mass index in adolescents. *J Pediatr*. 2011; 159(2):303–307. [PubMed: 21429506]
37. Canoy D, Wareham N, Luben R, et al. Cigarette smoking and fat distribution in 21,828 british men and women: a population-based study. *Obesity*. 2005; 13(8):1466–1475.
38. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014; 311(8):806–814. [PubMed: 24570244]
39. American Academy of Pediatrics. [Accessed May 1, 2017] Bright futures: Prevention and health promotion for infants, children, adolescents, and their families. <https://brightfutures.aap.org/Pages/default.aspx> Updated 2017
40. Gregg EW, Cheng YJ, Cadwell BL, et al. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA*. 2005; 293(15):1868–1874. [PubMed: 15840861]

Abbreviations

TSE	Tobacco smoke exposure
LDL-C	low-density lipoprotein cholesterol
HDL-C	high-density lipoprotein cholesterol
BMI	body mass index
BMIZ	body mass index z-score
WHtR	waist-to-height ratio
NHANES	National Health and Nutrition Examination Survey
LS mean	least square mean
SE	standard error
aOR	adjusted odds ratio
CI	confidence interval

Table 1
Characteristics of Participants 12-19 Years Old: Comparing those Included and Excluded from Lipid Analyses, NHANES 1999-2012

Participant Characteristics	Included in All Lipids Analyses (<i>n</i> = 1,822) ^a	Excluded from Lipids Analyses (<i>n</i> = 9,728)	<i>P</i> Value
Age, mean ± SE	15.52 ± 0.05	15.47 ± 0.02	.46
Sex			.29
Male	928 (52.5)	4,974 (51.1)	
Female	894 (47.5)	4,754 (48.9)	
Race/Ethnicity			.01
White	487 (62.8)	2,556 (59.4)	
Black	497 (13.5)	2,964 (14.7)	
Hispanic	762 (16.5)	3,632 (19.2)	
Other Race	76 (7.2)	576 (6.8)	
Annual Household Income Level			<.001
< \$20,000/y	448 (20.0)	2,369 (18.2)	
\$20,000-\$44,999/y	540 (27.6)	3,119 (28.3)	
\$45,000-\$74,999/y	329 (25.2)	1,721 (21.1)	
> \$75,000/y	301 (27.2)	1,927 (32.5)	
Dietary Intake, mean ± SE			
Grams	2,226.59 ± 27.75	2,565.38 ± 14.19	<.001
Energy (kcal)	2,430.79 ± 26.64	2,261.94 ± 11.30	<.001
Total fat (gm)	87.49 ± 1.15	83.75 ± 0.50	<.001
Total Saturated Fatty Acids (gm)	30.78 ± 0.44	28.81 ± 0.18	<.001
Total Monounsaturated Fatty Acids (gm)	33.51 ± 0.46	30.69 ± 0.19	<.001
Total Poly unsaturated Fatty Acids (gm)	16.57 ± 0.26	17.28 ± 0.12	.01
Sodium (mg)	3,549.06 ± 45.43	3,579.77 ± 20.80	.54
Alcohol (gm)	3.56 ± 0.47	2.27 ± 0.19	.01
Physical Activity Level			<.001
None/light	1,792 (100.0)	8,197 (79.1)	
Moderate	0 (0.0)	829 (14.6)	
Vigorous	0 (0.0)	329 (6.3)	
Total Cholesterol, mean ± SE	161.62 ± 0.71	160.18 ± 0.31	.06
TSE^b			<.001
Unexposed	663 (37.0)	3,908 (42.4)	
Passively Exposed	846 (41.7)	4,258 (40.3)	
Actively Exposed	313 (21.4)	1,562 (17.3)	

Abbreviation: SE, standard error; TSE, tobacco smoke exposure; kcal, kilocalories; gm, grams; mg, milligrams.

^aData are presented as weighted mean ± weighted SE or raw *n* (weighted %).

^bUnexposed is defined as serum cotinine < 0.05 ng/mL; passively exposed is defined as serum cotinine 0.05-2.99 ng/mL; and actively exposed is defined as serum cotinine ≥ 3 ng/mL.

Table 2
Characteristics of Participants 12-19 Years Old According to Tobacco Smoke Exposure Group, NHANES 1999-2012

Participant Characteristics	Serum Cotinine				P Value
	All ^a (N = 11,550)	Unexposed ^b (n = 4,571)	Passively Exposed (n = 5,104)	Actively Exposed (n = 1,875)	
Age, mean ± SE	15.48 ± 0.02	15.02 ± 0.03	15.29 ± 0.03	16.97 ± 0.04	<.001
Sex					<.001
Male	5,902 (51.2)	2,141 (47.6)	2,578 (51.3)	1,183 (59.74)	
Female	5,648 (48.8)	2,430 (52.4)	2,526 (48.7)	692 (40.3)	
Race/Ethnicity					<.001
White	3,043 (59.8)	1,158 (59.65)	1,193 (56.4)	692 (67.9)	
Black	3,461 (14.6)	764 (8.78)	2,086 (21.0)	611 (13.6)	
Hispanic	4,394 (18.8)	2,344 (24.39)	1,580 (16.1)	470 (12.0)	
Other Race	652 (6.8)	305 (7.18)	245 (6.5)	102 (6.6)	
Annual Household Income Level					<.001
<\$20,000/y	2,817 (18.4)	740 (10.41)	1,444 (22.4)	633 (28.6)	
\$20,000-\$44,999/y	3,659 (28.2)	1,403 (23.44)	1,662 (31.4)	594 (32.3)	
\$45,000-\$74,999/y	2,050 (21.6)	872 (21.92)	905 (22.5)	273 (18.5)	
>\$75,000/y	2,228 (31.8)	1,268 (44.23)	722 (23.8)	238 (20.7)	
Dietary Intake, mean ± SE					
Gram Weight of Food	2,523.77 ± 12.82	2,442.64 ± 17.68	2,429.07 ± 18.87	2,931.38 ± 40.09	<.001
Energy (kcal)	2,282.67 ± 10.41	2,217.18 ± 14.58	2,261.01 ± 15.95	2,486.97 ± 30.59	<.001
Total fat (gm)	84.20 ± 0.45	82.03 ± 0.67	84.16 ± 0.70	89.44 ± 1.27	.30
Total Saturated Fatty Acids (gm)	29.06 ± 0.17	28.26 ± 0.24	29.16 ± 0.26	30.67 ± 0.46	.17
Total Monounsaturated Fatty Acids (gm)	31.03 ± 0.18	30.00 ± 0.26	31.15 ± 0.27	33.23 ± 0.50	.13
Total Polyunsaturated Fatty Acids (gm)	17.19 ± 0.11	17.03 ± 0.16	16.95 ± 0.17	18.15 ± 0.32	.54
Sodium (mg)	3,576.05 ± 18.96	3,518.31 ± 27.54	3,531.12 ± 29.07	3,815.01 ± 53.36	.20
Alcohol (gm)	2.43 ± 0.18	0.26 ± 0.06	1.22 ± 0.13	10.31 ± 0.98	<.001
Physical Activity Level					<.001
None/light	9,989 (81.6)	3,869 (78.5)	4,523 (84.5)	1,597 (82.6)	
Moderate	829 (12.8)	437 (16.6)	270 (10.0)	122 (10.2)	
Vigorous	329 (5.6)	117 (4.9)	131 (5.5)	81 (7.2)	

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Abbreviation: SE, standard error; y, year; kcal, kilocalories; gm, grams; mg, milligrams.

Data are presented as weighted mean \pm weighted SE or raw n (weighted %).

Unexposed is defined as serum cotinine < 0.05 ng/mL; passively exposed is defined as serum cotinine 0.05-2.99 ng/mL; and actively exposed is defined as serum cotinine \geq 3 ng/mL.

Table 3
Adjusted Mean Levels and Regression Coefficients for Lipid Biomarkers Among
Participants 12-19 years old According to Tobacco Smoke Exposure, NHANES 1999-2012

Lipid Biomarker	Weighted LS Mean \pm Weighted SE	β Coefficient (SE)	P Value ^a
Total Cholesterol (n = 10,905)			
Unexposed ^b	159.46 \pm 0.92	(Ref)	
Passively Exposed	161.55 \pm 0.93	2.09 (0.89)	.02
Actively Exposed	156.04 \pm 1.22	-3.41 (1.19)	.004
HDL-C (n = 4,142)^c			
Unexposed	51.20 \pm 0.60	(Ref)	
Passively Exposed	49.12 \pm 0.53	-2.08 (0.65)	.001
Actively Exposed	48.90 \pm 0.73	-2.29 (0.81)	.005
Non-HDL-C (n = 4,142)^c			
Unexposed	113.93 \pm 1.73	(Ref)	
Passively Exposed	114.74 \pm 1.55	0.81 (1.88)	.67
Actively Exposed	112.17 \pm 2.13	-1.76 (2.37)	.46
LDL-C (n = 5,085)			
Unexposed	88.336 \pm 1.30	(Ref)	
Passively Exposed	92.49 \pm 1.32	4.13 (1.15)	<.001
Actively Exposed	86.82 \pm 1.64	-1.53 (1.53)	.32
Triglycerides (n = 5,259)			
Unexposed	86.31 \pm 3.00	(Ref)	
Passively Exposed	89.29 \pm 3.04	2.98 (2.66)	.26
Actively Exposed	94.18 \pm 3.75	7.87 (3.53)	.03

Abbreviations: LS mean, least square mean; SE, standard error; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

^aAnalyses are adjusted for age (continuous), sex (male, female), race/ethnicity (white, black, Hispanic, or other race), household income level (< \$20,000/y, \$20,000-\$44,999/y, \$45,000-\$74,999/y, \$75,000/y), dietary intake (continuous; gram weight of food, energy, total fat, total saturated fatty acids, total monounsaturated fatty acids, total polyunsaturated fatty acids, sodium, and alcohol), and physical activity level (none/light, moderate, or vigorous).

^bUnexposed is defined as serum cotinine < 0.05 ng/mL; passively exposed is defined as serum cotinine 0.05-2.99 ng/mL; and actively exposed is defined as serum cotinine \geq 3 ng/mL.

^cDid not adjust for physical activity due to all participants included in the analyses reporting none/light physical activity.

Table 4
Adjusted Mean Levels and Regression Coefficients for Adiposity Measures in 12-19 Year Olds According to Tobacco Smoke Exposure, NHANES 1999-2012

Adiposity Variable	Weighted LS Mean \pm Weighted SE	β Coefficient (SE)	P Value ^a
BMIZ (n = 10,751)			
Unexposed ^b	0.43 \pm 0.03	(Ref)	
Passively Exposed	0.65 \pm 0.03	0.22 (0.03)	<.001
Actively Exposed	0.57 \pm 0.05	0.13 (0.04)	.002
WC (cm) (n = 10,714)			
Unexposed	79.67 \pm 0.42	(Ref)	
Passively Exposed	82.17 \pm 0.42	2.51 (0.40)	<.001
Actively Exposed	81.53 \pm 0.56	1.87 (0.54)	<.001
WHtR (n = 10,712)			
Unexposed	0.48 \pm 0.002	(Ref)	
Passively Exposed	0.50 \pm 0.002	0.017 (0.002)	<.001
Actively Exposed	0.49 \pm 0.003	0.013 (0.003)	<.001

Abbreviations: LS mean, least square mean; SE, standard error; BMIZ, body mass index z-score; WC, waist circumference; WHtR, waist-to-height ratio.

^aAnalyses are adjusted for age (continuous), sex (male, female), race/ethnicity (white, black, Hispanic, or other race), household income level (< \$20,000/y, \$20,000-\$44,999/y, \$45,000-\$74,999/y, \geq \$75,000/y), dietary intake (continuous; gram weight of food, energy, total fat, total saturated fatty acids, total monounsaturated fatty acids, total polyunsaturated fatty acids, sodium, and alcohol), and physical activity level (none/light, moderate, or vigorous).

^bUnexposed is defined as serum cotinine < 0.05 ng/mL; passively exposed is defined as serum cotinine 0.05-2.99 ng/mL; and actively exposed is defined as serum cotinine \geq 3 ng/mL.

Table 5
Logistic Regression Results for Adiposity Among Participants 12-19 years old According to Tobacco Smoke Exposure, NHANES 1999-2012

Adiposity Variable	n (weighted %)	n (weighted %)	n (weighted %)	aOR (CI) ^{a,c}	P Value ^{a,c}
BMI Weight Classification (n = 10,965)	Underweight/Normal weight	Overweight	Obese		
Unexposed ^d	3,793 (43.35)	396 (35.31)	176 (31.03)	(Ref)	(Ref)
Passively Exposed	4,026 (39.20)	552 (49.32)	267 (45.20)	1.55 (1.38, 1.74)	<.001
Actively Exposed	1,502 (17.45)	148 (15.37)	105 (23.78)	1.09 (0.93, 1.29)	.30
	n (weighted %)	n (weighted %)	aOR (CI) ^c	P Value ^c	
WHR Classification^b (n = 10,887)	WHR < 0.65	WHR 0.65			
Serum cotinine < 0.05 ng/mL	4,092 (42.84)	236 (27.59)	(Ref)		
Serum cotinine 0.05-2.99 ng/mL	4,475 (39.97)	342 (49.15)	2.01 (1.72, 2.36)	<.001	
Serum cotinine ≥ 3 ng/mL	1,613 (17.19)	129 (23.26)	1.93 (1.59, 2.35)	<.001	

Abbreviations: aOR = adjusted odds ratio; CI = confidence interval; WHR, waist-to-height ratio.

^aDifferences between underweight/normal weight and overweight groups.

^bDifferences between underweight/normal weight and obese groups.

^cAnalyses are adjusted for age (continuous), sex (male, female), race/ethnicity (white, black, Hispanic, or other race), household income level (< \$20,000/y, \$20,000-\$44,999/y, \$45,000-\$74,999/y, \$75,000/y), dietary intake (continuous; gram weight of food, energy, total fat, total saturated fatty acids, total monounsaturated fatty acids, total polyunsaturated fatty acids, sodium, and alcohol), and physical activity level (none/light, moderate, or vigorous).

^dUnexposed is defined as serum cotinine < 0.05 ng/mL; passively exposed is defined as serum cotinine 0.05-2.99 ng/mL; and actively exposed is defined as serum cotinine ≥ 3 ng/mL.