Jurnal Promkes: The Indonesian Journal of Health Promotion and Health Education
Vol. 13 No. 1 (2025) 47-55 *doi: 10.20473/jpk.V13.I1.2025.47-55*

# *The Effects of Cigarette Smoke Exposure on Anthropometric Measurements, Lipid Profile, Fasting Blood Glucose, Fasting Insulin, and Blood Pressure in Overweight/Obese Adolescents*

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## ABSTRACT

***Background:*** *Cigarette smoke exposure increases the risk of obesity, metabolic syndrome, hypertension, hyperglycemia, and dyslipidemia. The nicotine content in cigarettes can acutely increase energy expenditure, decrease appetite, and several other chronic diseases such as cardiovascular disease, lung disease, cancer, and so on.* ***Objectives:*** *This study aims to determine the effect of cigarette smoke exposure on anthropometric measurements, lipid profiles, fasting blood glucose, fasting insulin, and blood pressure in overweight/obese adolescents.* ***Methods:*** *This was a cross-sectional study involving overweight/obese adolescents conducted from September to October 2019 to evaluate the effects of tobacco exposure on anthropometric measurements, lipid profile, fasting blood glucose (FBG), fasting insulin, and blood pressure.* ***Results:*** *This study showed no significant differences in the age distribution, body weight, body height, height-for-age z-score (HAZ), hip circumference, body mass index (BMI), BMI-for-age z-score, fasting insulin, FBG, lipid profile, and blood pressure between low, moderate, and high cigarette smoke exposure in overweight/obese adolescents, but the high exposure group had a bigger waist circumference than the moderate exposure group (107,45 + 11,25 vs 99,35 + 11,36 cm, p = 0,019) and low exposure (107,45 + 11,25 vs 99,04 + 12,08 cm, p = 0,015).* ***Conclusion:*** *The degree of exposure to heavy cigarette smoke has a greater waist circumference than exposure to light and moderate cigarette smoke. Cigarette smoke exposure is significantly associated with body weight and hip circumference, and heavy cigarette smoke exposure is prevalent in adolescent boys.*

***Keywords:*** *Adolescents, Cigarette smoke exposure, Metabolic syndrome, Obesity*

## INTRODUCTION

Exposure to tobacco smoke, both secondhand and active smoking, is one of the modifiable risks of obesity, and one of the public health concerns in the pediatric population, with an exposure of about 40% (Miyamura et al., 2023). Secondhand smoke exposure affects two-thirds of China's child population (Li et al., 2020). Cigarette smoke exposure is an independent factor associated with insulin resistance and may lead to the development of atherosclerosis (Weitzman et al., 2005). Tobacco use in cigarettes is known to correlate with body mass index (BMI), both in decreasing and increasing ways, in a dose- and sex-dependent manner (Saarni et al., 2009). However, the relationship between obesity and smoking remains unclear. It is suggested that nicotine acutely increases energy expenditure, leading to decreased appetite, so smokers have low body weight. But heavy smokers have a heavy body weight (Chiolero et al., 2008).

Smoking is one of the lifestyle factors that can increase the risk of metabolic syndrome and has a negative influence on the incidence of central obesity (abdominal obesity), blood pressure, blood sugar concentration, and lipid profile (Behl et al., 2023). There is growing evidence that secondhand smokers (SHS) are associated with greater


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

Vol. 13 No. 1, March 2025, 47-55 *doi: 10.20473/jpk.V13.I1.2025.47-55*

BMI in children through inflammation, oxidative stress, and endocrine disruption, leading to the development of insulin resistance (IR) and metabolic syndrome (Koyanagi et al., 2020). Cigarette smoke exposure plays a very substantial role in the pathogenesis of several chronic diseases, such as cardiovascular disease (CVD), cancer, lung disease, and so on (Kolovou et al., 2016). In obese children, exposure to cigarette smoke increases the risk of NAFLD by 44.5 times (Lin et al., 2014). Cigarette smoke exposure during conception in infants influences adipocyte accretion from birth to 5 years of age, and this influence is associated with exposure time. Exposure in infancy led to a 1.7% increase in adipocytes at 5 years of age, while exposure in mid-pregnancy and early childhood did not affect adipocytes (Moore et al., 2022). This study was conducted to determine the effect of cigarette smoke exposure on anthropometric measurements, lipid profile, fasting blood glucose, fasting insulin, and blood pressure in overweight/obese adolescents.

## METHODS

### Study Design

A cross-sectional study involving healthy overweight/obese adolescents was conducted from September to October 2019 to evaluate the effects of tobacco exposure on anthropometric measurements, lipid profile, fasting blood glucose, fasting insulin, and blood pressure.

### Subject

This study recruited healthy overweight/obese adolescents aged 13-18 years who attended schools in Surabaya and Sidoarjo. Subjects involved in this study must not use illegal drugs or alcohol and have no infectious diseases or chronic and congenital diseases. Exclusion criteria were taking dyslipidemia medications or hormonal therapy for six months before the study, taking antibiotics or steroids, or medications that affect body weight by asking the subjects they were taking medicine during at least three months when the screening was conducted, and what medicine they were taking. When they could not explain the medicine, we asked them to bring it when the study was conducted.

### Anthropometric Measurements

Anthropometric measurements were taken by two researchers by asking the subjects to wear minimal clothing, without accessories or pigtails (for women), hats, watches, shoes or sandals, belts, etc. Body weight was measured using a digital scale (Seca Robusta 813), by having the subject step on the scale after the "on mode" was activated for 30 seconds, and then the weight displayed on the screen was recorded. For height measurement, the subject was asked to step on the stadiometer floor plate (Seca 213 portable stadiometer) in a straight position, then the head positioner was pulled down until it touched the head. Height was then recorded according to the scale position. To measure waist circumference, the researchers looped a measuring tape (Seca 201) around the subject's abdomen in a standing position, above the hip bone and parallel to the navel, making sure the tape measure was not looped, not too tight or too loose. The 0 end of the tape was placed at the belly button, while the rest of the tape was wrapped around the abdomen, making sure the tape was not twisted. Waist measurement was taken after the subject exhaled. Hip circumference was measured by wrapping the measuring tape around the largest part of the hip in a standing position, making sure the tape was not twisted. The hip measurement is located at the end of the tape that meets the zero on the tape measure wrapped around the body.

### Blood Check

Blood was drawn through the cubitus vein by the laboratory staff as much as 5 ml and then put into a non-EDTA tube. Name, gender, date of birth, and school ID were recorded using labeled paper and wrapped around the tube for identification. After that, the tubes were put into an ice box containing ice blocks and then taken to the laboratory for further analysis, which included fasting blood sugar, fasting insulin, and lipid profile.

### Blood Pressure Measurements

To measure blood pressure with a digital tensimeter, the subjects were asked to sit comfortably in a chair in a relaxed position. Making sure the measuring arm was level with the heart, the blood pressure cuff was placed around the upper arm above the elbow, making sure it fitted snugly without being too tight or too loose. The blood pressure tube was connected to a monitor or blood pressure readout. The blood pressure monitor was


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

turned on and waited for instructions or warnings. After pressing the button to start the measurement, the cuff will inflate to compress the arm and measure blood pressure. There is a need to be quiet and avoid talking during the measurement. The monitor shows the systolic and diastolic pressure results after the process is completed.

**Determination of Metabolic Syndrome (MetS)**

Metabolic syndrome (MetS) criteria were determined based on the International Diabetes Foundation (IDF) criteria, namely central obesity (abdominal obesity; waist circumference > 90th percentile or adult cut-off value) accompanied by at least two other signs: glucose intolerance (fasting glucose > 100 mg/dL), triglycerides > 150 mg/dL, and HDL-c (high-density lipoprotein cholesterol) < 40 mg/dL, high blood pressure (systole blood pressure > 130 mmHg or diastole blood pressure > 85 mmHg) (Magge et al., 2017; Zimmet et al., 2007).

**Statistical Analysis**

Statistical analysis carried out in this study includes: the normality test (Kolmogorov-Smirnorv) and homogeneity test, followed by one-way ANOVA or Kruskal Wallis test (depending on the normality and homogeneity of variables) to determine the real difference between groups (divided into low exposure, medium exposure, and high exposure), Pearson correlation test or Spearman Rho, and Pearson chi-square or Fischer exact test. Statistical tests were declared significant if the p-value was <0.05.

**Cigarette Smoke Exposure Assessment**

Cigarette smoke exposure was assessed using the World Health Organization-Global School Student Health Survey (WHO-GSHS), as it is an easy and inexpensive tool. The results of the assessment are shown in Table 1. We divided cigarette smoke exposure into three groups: low (<7), medium (8-11) and high (>12). The mean cigarette smoke exposure was 8.53 + 2.51 (min-max 6 - 23).

**Research Ethics**

This study has been ethically tested and declared ethically passed by the Health Research Ethics Committee of the Faculty of Medicine, Airlangga University, Surabaya, Indonesia, No. 141/EC/KEPK/FKUA/2020.

**Table 1**. Assessment of Cigarette Smoke Exposure Using the Global School-based Student Health Survey (GSHS).

| Variable | Low exposure (n=73) | Moderate exposure (n=74) | High exposure (n=14) | p |
|---|---|---|---|---|
| Age, months-old | 190.22 ± 16.93 | 190.38 ± 16.14 | 194.14 ± 16.54 | 0.707[3] |
| Smoking frequency per month | | | | 0.000[1] |
| - 0 day | 73 (100%) | 70 (94.59%) | 7 (50%) | |
| - 1-2 day | 0 | 4 (5.41%) | 2 (14.28%) | |
| - 3-4 day | 0 | 0 | 3 (21.42%) | |
| - In a whole month | 0 | 0 | 2 (14.28%) | |
| Age of first smoking | | | | 0.000[1] |
| - Never tried | 70 (95.89%) | 64 (86.48%) | 4 (28.57%) | |
| - 7 years or younger | 0 | 0 | 2 (14.28%) | |
| - 10-11 year | 1 (1.36%) | 3 (4.05%) | 3 (21.42%) | |
| - 12-13 year | 1 (1.36%) | 4 (5.40%) | 2 (14.28%) | |
| - 14-15 year | 1 (1.36%) | 1 (1.35%) | 3 (21.42%) | |
| - 16-17 year | 0 | 2 (2.70%) | 0 | |
| Use of other tobacco products except cigarettes (such as betelnut, cigar, electrical cigarette) | | | | 0.002[1] |
| - 0 day | | | | |
| - 1-2 day | 73 (100%) | 73 (98.64%) | 11 (78.57%) | |
| - 3-4 day | 0 | 1 (1.35%) | 1 (71.42%) | |
| - In a whole month | 0 | 0 | 1 (71.42%) | |
| | 0 | 0 | 1 (71.42%) | |


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

Vol. 13 No. 1, March 2025, 47-55 *doi: 10.20473/jpk.V13.I1.2025.47-55*

| Variable | Low exposure (n=73) | Moderate exposure (n=74) | High exposure (n=14) | p |
|---|---|---|---|---|
| The frequency of smoke exposure from the environment or people around | | | | 0.000[2] |
| - 0 day | | | | |
| - 1-2 day | 44 (60.27%) | 4 (5.41%) | 8 (57.14%) | |
| - 3-4 day | 28 (38.35%) | 18 (24.32%) | 0 | |
| - 5-6 day | 1 (1.36%) | 19 (25.67%) | 3 (21.42%) | |
| - 7 full day | 0 | 7 (9.45%) | 1 (71.42%) | |
| | 0 | 28 (37.83%) | 10 (71.42%) | |
| Efforts to quit smoking | | | | 0.000[1] |
| | 73 (100%) | 63 (85.13%) | 1 (71.42%) | |
| - Never smoked | 0 | 3 (4.05%) | 3 (21.42%) | |
| - No smoking during | 0 | 7 (9.45%) | 8 (57.14%) | |
| | 0 | 1 (1.35%) | 2 (14.28%) | |
| - 12 month | | | | |
| - Yes. | | | | |
| - No. | | | | |
| Smokers in the family | | | | 0.000[1] |
| - None | | | | |
| - Father | 60 (82.19%) | 25 (33.78%) | 4 (28.57%) | |
| - Don't know | 12 (16.43%) | 47 (63.51%) | 10 (71.43%) | |
| | 1 (1.36%) | 2 (2.70%) | 0 | |

[1]Fischer exact test;[2] Pearson chi square; [3]One way ANOVA.

## RESULTS AND DISCUSSION

A total of 161 subjects were involved in this study, with a male-to-female ratio of 87/74. Subject characteristics are shown in **Table 2.** There were no significant differences in the distribution of age, weight, height, HAZ, hip circumference, BMI, BMI-for-age z-score, fasting insulin, FBG, lipid profile, and blood pressure between low, medium, and high cigarette smoke exposure in overweight/obese adolescents, but the high exposure group had a larger waist circumference compared to the medium exposure group. (107.45 ± 11,25 vs 99,35 ± 11,36 cm, p = 0,019) and low exposure (107,45 ± 11,25 vs 99,04 ± 12,08 cm, p = 0,015).

**Table 2.** Subjects' Characteristics.

| Variable | Low exposure (n=73) | Moderate exposure (n=74) | High exposure (n=14) | P value |
|---|---|---|---|---|
| Age | 190.22 ± 16.93 | 190.38 ± 16.14 | 194.14 ± 16.54 | 0.707[1] |
| Body weight, kg | 89.98 ± 15.39 | 90.79 ± 14.97 | 99.02 ± 17.36 | 0.130[1] |
| Body height, cm | 143.84 ± 26.76 | 145.88 ± 25.16 | 144.48 ± 24.42 | 0.811[2] |
| HAZ | -0.48 ± 1.11 | -0.66 ± 0.96 | -0.88 ± 0.87 | 0.327[1] |
| Waist circumference, cm | 99.35 ± 11.36 | 99.04 ± 12.08 | 107.45 ± 11.25 | 0.043[1] |
| Hip circumference, cm | 111.23 ± 9.01 | 112.20 ± 11.72 | 114.61 ± 8.65 | 0.518[1] |
| BMI | 33.96 ± 4.52 | 34.84 ± 5.79 | 36.85 ± 4.99 | 0.144[1] |
| BMI for age | 2.89 ± 0.58 | 2.96 ± 0.68 | 3.30 ± 0.63 | 0.090[1] |
| Fasting insulin | 23.25 ± 13.59 | 26.89 ± 17.77 | 26.00 ± 12.67 | 0.344[2] |
| FBG | 87.51 ± 7.68 | 86.78 ± 6.95 | 86.14 ± 4.97 | 0.791[2] |
| Total cholesterol | 171.22 ± 28.53 | 179.81 ± 37.80 | 170.07 ± 23.24 | 0.240[1] |
| HDL-c | 42.68 ± 6.95 | 44.20 ± 8.10 | 40.50 ± 6.93 | 0.179[1] |
| LDL-c | 112.77 ± 24.53 | 119.01 ± 32.22 | 118.86 ± 25.22 | 0.386[1] |
| Triglycerides | 117.37 ± 55.25 | 115.87 ± 68.13 | 113.57 ± 47.10 | 0.704[2] |
| Systolic-BP | 126.84 ± 13.97 | 127.12 ± 12.78 | 125.21 ± 14.94 | 0.874[2] |
| Diastolic-BP | 82.20 ± 9.30 | 82.39 ± 9.66 | 81.64 ± 8.78 | 0.905[2] |

[1]One way ANOVA;[2] Kruskal Wallis

Cigarette smoke exposure includes both active and passive smoking (Florescu et al., 2009). The definition of passive


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

smoking is inhaled cigarette smoke, also known as second-hand smoke (SHS) and environmental tobacco smoke (ETS), which refers to people who do not actively smoke, but get exposure to cigarette smoke from relatives, friends, and other family members who actively smoke (Ebrahimi et al., 2019). Cigarette smoke exposure is one of the environmental factors that may play an important role in the development of childhood obesity (Srivastava et al., 2024). Parental cigarette smoke exposure increases the risk of overweight/obesity by 2.10 times in adolescents (Wang et al., 2014). The relationship between cigarette smoke exposure and obesity and central obesity is not entirely clear. However, it is known that individuals who are obese and smoke have a shorter lifespan of 13 years than normal people who do not smoke (Tuovinen et al., 2016). Active smokers who increased their cigarette consumption by one cigarette per day had a 0.14% increase in waist circumference and a smaller hip circumference (Morris et al., 2015). Research on female smokers showed that active smokers had a larger waist circumference than non-smoking women (p=0.004), and increased levels of androstenedione and extradiol (Ellberg et al., 2018). Morris et al (2015) stated that every increase in consumption of 1 cigarette per day will be accompanied by an increase of 0.14% in waist circumference, due to the redistribution of adipocytes towards central obesity. Adult studies show that people who have never smoked have a smaller waist-hip ratio, waist circumference, and hip circumference than people who have smoked and are currently smoking (Morris et al., 2015). Similar evidence was seen in the level of exposure to cigarette smoke in this study.

While research on parental cigarette smoke exposure to children shows it is positively associated with the prevalence of overweight/obesity, both preconception and post-conception exposure, although only dominant in males. This relationship was influenced by the quantity of smoking, duration, and age of exposure (Srivastava et al., 2024). Another study also found a positive association between cigarette smoke exposure and the prevalence of central obesity in boys (Jaakkola et al., 2021), which is also in line with other findings of increased prevalence of central obesity and larger waist circumference. However, passive exposure to cigarette smoke did not affect the incidence of prediabetes, insulin resistance, and visceral fat (Davis et al., 2016). However, one study showed insignificant results, with parental cigarette smoke exposure not affect BMI, and even having a lower BMI (Yaghoubi et al., 2015), which is in line with the results of this study. The pediatric study showed an increase in waist circumference of 0.93 and 1.56 cm in children with transient and continuous exposure, and this was accompanied by an increase in BMI of 0.48 and 0.81 points 23. The in vitro study showed a modified body fat distribution due to chronic cigarette smoke exposure in mice, especially in visceral fat reserves (Dubois-Deruy et al., 2020).

Correlations between tobacco exposure and anthropometric measurements, lipid profile, fasting blood glucose, fasting insulin, and blood pressure are summarized in **Table 3**. Tobacco exposure score was weakly correlated with body weight (r=-0.162, p=0.040) and hip circumference (r=-0.163, p=0.039), while it showed no correlation with other parameters. It almost showed a significant correlation with waist circumference.

**Table 3.** Correlation between Tobacco Exposure Scores and Anthropometric Measurements, Lipid Profile, Fasting Blood Glucose, Fasting Insulin, and Blood Pressure.

| Variable | R | P |
|---|---|---|
| HAZ | -0.0554 | 0.479[1] |
| Body weight | 0.162 | 0.040[1] |
| Body height | -0.052 | 0.515[2] |
| Waist circumference | 0.151 | 0.055[1] |
| Hip circumference | 0.163 | 0.039[1] |
| BMI | 0.135 | 0.087[1] |
| Score z BMI-for-age z-score | 0.144 | 0.057[1] |
| Fasting insulin | 0.058 | 0.465[2] |
| FBG | -0.012 | 0.796[2] |
| Total cholesterol | -0.061 | 0.444[1] |
| HDL-c | 0.003 | 0.968[1] |
| LDL-c | 0.072 | 0.366[1] |
| TG | 0.014 | 0.864[2] |
| Systolic-BP | 0.019 | 0.808[2] |
| Diastolic-BP | 0.057 | 0.472[2] |

[1]Pearson Correlation; [2] Spearman Rho Correlation.

Cigarette smoke exposure leads to decreased pre- and postnatal weight and length in children, resulting in poor growth


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

Vol. 13 No. 1, March 2025, 47-55 *doi: 10.20473/jpk.V13.I1.2025.47-55*

parameters (increased risk of being underweight, wasting, and stunting), but increased BMI in children (Nadhiroh et al., 2020). In contrast to the results of this study, which found a positive correlation between cigarette smoke exposure score and body weight, it showed a positive correlation between exposure weight and weight gain. Exposure to cigarette smoke through active smoking mothers' children's weight showed a negative effect, whereas exposed children experienced a weight loss of about 0.53 kg (Dasgupta et al., 2018). An in vitro study in rats showed a 23% reduction in caloric intake (Chen et al., 2007). However, when studies on adolescent active smokers were conducted, the results were contradictory, adolescents in France, Suriname, and Indonesia had a 72% risk of being underweight, but cigarette users in Uganda, Nigeria, and Namibia had 2.30 times the risk of being overweight/obese (Wang, 2021). The same study also found that while smoking was associated with weight loss and BMI, smoking intensity did not affect body weight, and there was no dose-response relationship (Jitnarin et al., 2014). The positive association of cigarette smoke exposure with weight is thought to be related to the eating preferences of subjects who like to consume unhealthy foods (Srivastava et al., 2024).

**Table 4**. Comparison of Severity of Cigarette Smoke Exposure by Gender, MetS and MetS Components.

| Variable | Low exposure | Moderate exposure | High exposure | P |
|---|---|---|---|---|
| Gender, n (%) | | | | 0.005[1] |
| - Male | 40 (54.79%) | 34 (45.95%) | 13 (92.86%) | |
| - Female | 33 (45.21%) | 40 (54.05%) | 1 (7.14%) | |
| MetS, n (%) | 33 (45.21%) | 29 (39.18%) | 5 (35.71%) | 0.681[1] |
| Status BMI, n (%) | | | | 0.626[1] |
| - Overweight | 34 (46.57%) | 32 (43.24%) | 8 (57.14%) | |
| - Obesity | 39 (53.42%) | 42 (56.75%) | 6 (42.85%) | |
| Abdominal obesity, n (%) | 67 (91.78%) | 69 (93.24%) | 14 (100 | 0.730[2] |
| Hypertriglyceridemia, n (%) | 27 (36.98%) | 26 (35.13%) | 4 (28.57%) | 0.885[2] |
| Hyperglycemia, n (%) | 2 (2.73%) | 2 (2.70%) | 0 | 1.000[2] |
| Hypertension, n (%) | 39 (53.42%) | 38 (51.35%) | 6 (42.85%) | 0.700[2] |
| Hipo HDL-c, n (%) | 37 (50.68%) | 31 (41.89%) | 7 (50%) | 0.559[2] |

Table 4 shows that high exposure to cigarette smoke is more prevalent in males than females. There were no differences between the severity of exposure to tobacco smoke and incidence of MetS, overweight/obesity, and components of MetS. Health surveys using the World Health Organization Global Adult Tobacco Survey (WHO GATS) questionnaire showed men were exposed to tobacco from less than 18 years of age (Birmpili et al., 2012). In adolescent studies, males are more heavily exposed to secondhand smoke than females (Birmpili et al., 2012), which is in line with the results of this study.

Research in China showed an increase in leptin, retinol binding protein-4 (RBP-4), and a decrease in adiponectin in children exposed to cigarette smoke by 39.2%, 11.4%, and 4.6%, respectively (Li et al., 2020). The suspected influence of cigarette smoke exposure on the incidence of obesity through the leptin pathway, although population studies are still controversial. Active maternal smoking (6 mg/kg/day) from day two to 16 of lactation causes neonatal hyperleptinemia and primary hyperthyroidism. Nicotine causes an increase in body fat due to high levels of leptin (Lisboa et al., 2012). Other studies have also shown an increase in pro-inflammatory markers such as IL-6 and C-reactive protein (CRP) (Nagel et al., 2009). Cigarette exposure triggers the formation of free radicals that can interact with vascular homeostasis, increase inflammation/oxidation stress, and damage pancreatic β-cell function. In addition, exposure to cigarette smoke also has a positive influence on pro-inflammatory factors such as peroxisome proliferator-activated receptors (PPAR) and tumor necrosis factor-alpha (TNF-α) (Behl et al., 2023).

Research has also shown the effect of parental cigarette smoke exposure on elevated blood pressure (Li et al., 2020), by 0.44 mmHg systole and 0.26 mmHg diastole in females, by increasing the risk of hypertension by 1.11 times compared to


Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

girls who were not exposed (Zhang et al., 2019), However, the relationship between cigarette smoke exposure and blood pressure was not confirmed in this study. We suspect that the difference in results is due to differences in the determination of the exposure dose. Studies in adolescents using serum cotinine (>0.05 ng/mL in passive smokers) as a marker of exposure to cigarette smoke, showed a significant association between exposure to cigarette smoke and the incidence of metabolic syndrome (OR=4.7, 95% CI: 1.7 - 12.9), and the risk increased in active smokers (OR=6.1, 95% CI: 2.8 - 13.) 3. 3. A study in Iran also found increased triglyceride levels in children and adolescents of secondhand smoke, and its association with metabolic syndrome (OR=1.63, 95% CI: 1.17-2.29) (Ebrahimi et al., 2019). These results contradict the results of this study, possibly using different criteria for metabolic syndrome.

The weakness of this study is that it did not use cotinine as a marker of cigarette smoke exposure, so the effect of exposure on markers of metabolic syndrome has not been seen. Moreover, leptin and adiponectin levels were not examined.

## CONCLUSION

The degree of heavy cigarette smoke exposure had a larger waist circumference than light and moderate cigarette smoke exposure. Cigarette smoke exposure was significantly associated with body weight and hip circumference, and heavy smoke exposure was more prevalent among adolescent boys.

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Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

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Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025

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Received: 26-01-2024, Accepted: 28-05-2024, Published Online: 05-03-2025