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Smoking and Metabolic Syndrome: A Review of the Double Impact on Cardiovascular Disease Risk

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Review Article

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Abstract

Metabolic syndrome (MetS) is a constellation metabolic changes that increase the risk of cardiovascular disease (CVD). Smoking associated with higher TG and low-density lipoprotein (LDL) levels, low HDL, and increased risk of developing insulin resistance, which may increase the risk of developing MetS. Smoking and MetS occurring together may further increase CVD risk. This paper reviewed the association between smoking and its exposure on development of MetS, as well as the combined effect of smoking and MetS on cardiovascular health. Current and former smoking increases the risk of MetS and its components, especially elevated TGs, lower HDL levels, and insulin resistance, and the risk increases with higher smoking exposure and persists over time in former smokers. The exposure of children and adolescents to secondhand smoke, especially among overweight children, is of concern as passive smoking causes metabolic derangements (weight gain and lipid abnormalities) that may lead to development of MetS and increased risk for CVD. Smokers with MetS had nearly twice the risk of cardiovascular events compared to those who did not smoke nor have MetS. Tobacco and the metabolic consequences of obesity are the two leading causes of preventable death; thus, it is crucial to recognize and address the adverse health effects of MetS, and include tobacco cessation in the lifestyle interventions for MetS, in addition to weight management and physical activity.

Keywords: Metabolic Syndrome; Smoking; Cardiovascular Risk; Smoking Exposure

Abbreviations: MetS: Metabolic Syndrome; LDL: Low-Density Lipoprotein; CVD: Cardiovascular Disease.

Introduction

Tobacco smoking and metabolic syndrome (MetS) are common in the United States (US); both increase the risk of cardiovascular disease (CVD) and death [1]. Nearly 20% of US adults use some type of tobacco product, with cigarettes being the most common (12.5%) [1]. Cigarette smoking causes nearly half a million deaths (one in every five deaths) each year [2] and 34,000 nonsmokers die from heart disease each year from exposure to secondhand tobacco smoke [3].

MetS is a constellation of physiological, hemodynamic, inflammatory and metabolic changes that increase the risk of

developing diabetes and CVD [4]. The condition is made up of five components including hypertension, insulin resistance, abdominal obesity, high levels of triglycerides (TG), and low levels of high density lipoprotein (HDL) [5]. Three of the five components must be present for diagnosis. MetS is a public health concern as its prevalence among US adults has been increasing in the past decades, such that 30 % of adults currently have MetS [6,7]. Its early detection is of highest importance as early lifestyle interventions and risk factor modification can decrease risk of CVD. The pathophysiology of how MetS develops is unclear [8]; however, it's linked to lifestyles, especially diet and physical activity.

Tobacco exposure may also contribute to development of MetS. Smokers are reported to have a higher risk of high TG, high low-density lipoprotein (LDL) levels, and low HDL levels. They are at greater risk of developing insulin resistance, which may increase the risk of developing MetS [9]. The combined adverse effect of smoking and MetS on cardiovascular health may be greater than either condition alone. This paper reviews the association between smoking and its exposure on development of MetS, as well as the combined effect of smoking and MetS on cardiovascular health.

Association between Smoking, Metabolic Syndrome and its Components

Several studies have reported an association between smoking and the development of metabolic abnormalities. In a large multiethnic study conducted across 6 different US states, almost 6,000 participants (n = 5913) were followed for 2 years [10]. The results indicate that smoking is associated with MetS (OR = 1.4, 95% CI [1.1-1.7]) as smokers had higher TG and C-reactive protein (CRP) and lower HDL levels than non-smokers. They reported that though African Americans generally have higher HDL levels than other race and ethnicities, smoking eliminates this advantage. African American smokers in the study had lower HDL levels than other racial and ethnic groups.

Two community-based surveys of Asian men in Korea [11] and Taiwan Ching-Chu C, et al. [12] reported a higher prevalence of MetS in current and former smokers that was nearly double that of nonsmokers (OR=1.83, 95% CI [1.26-2.65] and OR= 1.77, 95% CI [1.06–2.96], respectively). Smoking was associated with higher TGs and lower HDL levels, but not hypertension, abnormal fasting glucose, or increased waist circumference.

A cross-sectional study by Bermudez, et al. [13] from Venezuela reported that former smokers had a 13.8% higher prevalence of MetS than current smokers (47.9% in former smokers vs 42.1% in current smokers, $p<0.05$) [13]. This finding is supported by other longitudinal studies demonstrating that the effect is persistent over time [14-16]. The greater probability of getting metabolic syndrome among former smokers could be attributed to the weight gain that former smokers may experience after cessation. Further studies are needed to explore interventions to limit weight gain.

There are mixed reports on the effects of smoking on blood pressure and glucose levels. Results from the study conducted in Venezuela showed that smoking was inversely associated to high blood pressure but did not influence glucose levels [13]. Another study conducted in Iran supports these findings as it showed mean systolic and diastolic blood pressures were lower in smokers than in non-smokers [17]. However, former smokers were more likely to develop hyperglycemia and hypertension than people who currently smoke or never smoked [13].

All the referenced studies suggest that smoking increases insulin resistance and has consistent effects on lipids, namely increased TGs and lower HDL levels. These effects are persistent over time and may affect former smokers even more than current smokers.

Association between Secondhand Smoke Exposure and Metabolic Syndrome

The risk of active or former smoking on the development of MetS has been reported more than the risk of passive exposure to secondhand smoke (SHS), but SHS also poses a potential association with MetS. In a meta- analysis of 18 studies, Chen, et al. [18] reported that SHS is associated with the metabolic changes that can lead to the development of MetS; however, the association changes with age. Specifically, older people were more likely to have hyperglycemia, whereas younger people were more likely to have lipid abnormalities (high LDL levels and low HDL levels). Regardless of age, people who were exposed to SHS had a higher waist circumference, which is an indicator of abdominal obesity.

A cross sectional study conducted in Iran reported that children and adolescents exposed to SHS are 1.2 times more likely to be overweight and have a 1.6-fold increase risk of having MetS, compared to children and adolescents who were not exposed [19]. Kelishadi, et al. [20] reported that children and adolescents who were active smokers had twice the prevalence of low HDL and MetS compared to nonsmokers. In this study, both passive smoking and SHS exposure had 1.2 times higher prevalence of low HDL and MetS in comparison to nonsmokers. In another study by Moore et al, which was a population health study conducted in Colorado, children and adolescents who were overweight, or at risk of being overweight, and were exposed to SHS had a four-fold increased risk of MetS [21].

Gender might play a role in the risk of developing MetS following SHS exposure. A study conducted in South Korea reported that the association between SHS and MetS was only significant in females (OR = 1.02 CI [0.94 to 1.11] in males vs. OR = 1.17 CI [1.06 to 1.29] in females) [22]. Furthermore, the risk in females increased with increased time exposure to SHS.

In a longitudinal study that followed participants for approximately 3 years, Choi, et al. [23] reported that changes in SHS exposure status over time could modify the risk of developing MetS. In a study of over 70,000 verified non-smokers, those who had either continuous or new exposure to SHS were approximately 20-30% more likely to develop MetS. Former SHS was not associated with increased risk in this study. Two other studies support this hypothesis that SHS and environmental smoke exposure are independently associated with MetS and its components [21,24].

Dose-Dependent Relationship between Smoking Amount and Metabolic Syndrome

Although few studies investigated the dose-dependent relationship between smoking and MetS, the studies consistently report a stepwise increased risk for the MetS with increasing smoking duration and intensity (number of cigarettes smoked per day). A positive dose-response relationship was reported by Wang, et al. [25] in a study of over 15,000 adults over age 60 in the China Health and Nutrition Survey. Those in the highest quartile of cigarette exposure had twice the risk of MetS compared to non-smokers. However the cumulative smoking effect and the development of MetS components was more evident in adults <70 years old.

The dose-response relationship is supported by Kim, et al. [26] in a longitudinal study of over 3,000 Korean men followed for 12 years. They report a positive dose-response to both intensity (number of cigarettes per day) and cumulative dose (number of cigarettes over a lifetime) on risk for MetS. A study by Cena, et al. [27] found that three of the MetS components (systolic blood pressure, fasting glucose, and waist circumference) showed an increasing trend from light to heavy smokers. Another study by Hwang, et al. [28] did not find significant relationship of smoking to blood pressure and abdominal obesity, but they did report that HDL, triglycerides and glucose levels were affected by smoking intensity. A longitudinal study conducted in Chile that followed subjects for 22 years Cheng E, et al. [29] reported that even light smoking is associated with metabolic change. Adults who smoked <29 cigarettes a week still had lower HDL levels than the nonsmoker group.

Gossett, et al. [30] evaluated just one of MetS components and smoking intensity, namely lipoprotein abnormalities. The results are in alignment with findings from other studies, reporting that higher smoking burden is associated with increases in total cholesterol, triglycerides and LDL, and a decrease in HDL. However, this study reported that smoking intensity had only a minor effect and that the differences between cholesterol levels between all the study groups were small, although still statistically significant.

Combined Effects of Tobacco Smoke Exposure and Mets on Cardiovascular Risk

Smoking tobacco may increase risk of developing MetS or its components, but it may also have a synergistic negative impact on cardiovascular risk in people with MetS. Li, et al. [31] evaluated the combined effect on accelerated subclinical CVD by measuring the carotid intima-media thickness (CIMT) as an indicator of atherosclerosis. They concluded that smoking exacerbates the adverse effects of age and MetS in young adults. Using CIMT as an indicator of vascular age, people who smoked and had MetS were 6-7 years older than their counterparts (quitters or nonsmokers). Kohashi, et al. [32] followed 301 MetS patients for 12 months and found that persistent smokers had a progression of CIMT compared to nonsmokers and quitters, and concluded that this progression may put MetS patients at an increased risk of atherothrombotic events.

In a population-based prospective cohort study of over 3,500 Chinese participants without diabetes or CVD at baseline, Zhang, et al. [33] evaluated the combined effects of current smoking and MetS on cardiovascular events over 8 years of follow-up. They reported that 908 (25.9%) had MetS at baseline. Smokers had a dose-related increase in development of cardiovascular events, and those with MetS had over twice the risk as those who did not smoke or have MetS, respectively. Those who both smoked and had MetS had nearly twice the risk of cardiovascular events as those who had only one of the risk factors (OR = 1.81).

In a Korean population-based cohort study of 6 million people Park, et al. [34] investigated the combined effect of both risk factors on major adverse cardiovascular events (MACE). All participants were smokers, and 20% had MetS at baseline. They were followed for an average of 4.28 years for development of MetS, recovery from MetS, and MACE. Smoking played a more harmful role on cardiovascular health than the presence of MetS in this study. Light-to-heavy smokers had an approximately 50% and 90% higher risk of MACEs compared to nonsmokers, regardless of MetS status. During the study, approximately 7% of those without MetS at baseline developed MetS, and 29% of those with MetS at baseline recovered from MetS. Smokers were more likely to develop MetS (OR = 1.71 for heavy smokers), and were also up to 30% less likely to recover from MetS. Thus smokers

were more both more likely to develop MetS and less likely to recover. Elevated TGs was the MetS component most likely to be abnormal in smokers, and least likely to reverse.

Key Points

- ✓ Tobacco Use
- One-fifth of adults
- Increases risk of metabolic syndrome, especially abnormal lipids
- Current, former, and passive Smoke all have adverse metabolic effects
- ✓ Metabolic Syndrome
- One-third of adults
- Three of five components for diagnosis
- ✓ High triglycerides
- ✓ Low HDL
- ✓ Central adiposity
- ✓ Hypertension
- ✓ Abnormal glucose
- ✓ Tobacco and Metabolic Syndrome
- Preventable causes of heart disease and death
- Each increases cardiovascular risk alone
- Act synergistically to increase heart disease further when both present

Conclusion

Both current and former smoking increase the risk of MetS and its components, especially elevated TGs, lower HDL levels, and insulin resistance, and the risk increases with higher smoking exposure and persists over time in former smokers. The exposure of children and adolescents to secondhand smoke, especially among overweight children, is of concern as passive smoking causes metabolic derangements (weight gain and lipid abnormalities) that may lead to development of MetS and increased risk for CVD. Smokers with MetS had nearly twice the risk of cardiovascular events compared to those who did not smoke nor have MetS. Tobacco and the metabolic consequences of obesity are the two leading causes of preventable death; thus, it is crucial to recognize and address the adverse health effects of MetS, and include tobacco cessation in the lifestyle interventions for MetS, in addition to weight management and physical activity.

Conflict of Interest

The authors declare no conflict of interest.

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