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Association of cigarette smoking with metabolic syndrome (MetS): A review

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Abstract:
Metabolic syndrome combines central adiposity, dyslipidaemia, dysglycaemia and elevated blood pressure. Cigarette smoking may affect metabolic risk through inflammatory and autonomic pathways. Therefore, it is of interest to examine the epidemiological associations, mechanisms and cessation effects. Low HDL cholesterol and raised triglycerides are recurrent associations; other findings vary. Smoking cessation remains essential, while longitudinal metabolic research is still needed.

Keywords: Cigarette smoking, metabolic syndrome (MetS), insulin resistance, triglycerides, HDL cholesterol, abdominal adiposity, smoking cessation, narrative review

Background:
Metabolic syndrome (MetS) is a clinical cluster of abdominal adiposity, elevated triglycerides, low HDL cholesterol, dysglycemia and elevated blood pressure. The diagnostic construct is useful for recognizing joint cardiometabolic burden but combines measurements that may respond differently to tobacco exposure. Recent population-based studies continue to examine smoking patterns alongside MetS prevalence and treatment-related behaviours **[1].** The relationship between tobacco smoking and metabolic health is complicated by dose, duration, alcohol use, diet, activity, body composition and sociodemographic circumstances. Cigarette smoking is an established risk factor for cardiovascular disease regardless of whether an individual meets a particular MetS definition. Contemporary tobacco epidemiology also reflects changing smoking patterns, cessation practices and use of multiple products **[2].** Therefore, it is of interest to examine the component-specific associations and plausible mechanisms rather than presenting a new systematic search or pooled analysis.

Narrative review approach:
Selected epidemiological, clinical and mechanistic publications cited in the original manuscript were organized by metabolic phenotype and tobacco exposure. The discussion prioritizes distinctions among current, former and never smoking, dose-response patterns and the circumstances under which associations differ between populations. This qualitative review did not register a protocol, conduct formal PRISMA screening, perform standardized study-level bias scoring or calculate a pooled odds ratio. Reported findings are contextualized, not re-analysed at the participant level.

Metabolic syndrome definitions and interpretive challenges:
Diagnostic definitions differ in their handling of central adiposity and population-specific waist thresholds. The same smoking-related lipid changes may contribute to MetS classification in one population but not another if other metabolic components or cut-offs differ. A clinical overview of cardiometabolic consequences reinforces why MetS should be interpreted as a composite clinical risk marker rather than a uniform pathophysiological entity **[3].** Longitudinal investigations have considered changing metabolic profiles over time, providing a stronger basis for temporal inference than one-time classification **[4].** Even these studies face possible residual confounding from changing weight, physical activity, alcohol consumption and medication use. Any comparison of MetS incidence across studies should specify the exact definition and method of exposure ascertainment.

Nicotine, sympathetic activity and lipid metabolism:
Nicotine activates autonomic pathways and can affect catecholamine release, adipose-tissue lipolysis and free fatty acid flux. Experimental work has characterized molecular pathways connecting smoking-related exposures to altered metabolic regulation **[5].** These mechanisms are plausible but are not sufficient alone to estimate the prevalence of MetS in a real-world population. Atherogenic dyslipidaemia is among the more consistent clinical patterns associated with cigarette smoking. Adverse changes in triglycerides and HDL cholesterol may be more apparent than shifts in total body mass, underscoring that BMI and abdominal risk need not move in the same direction. Population analyses of metabolic risk and smoking have reported heterogeneity in effects by sex and background cardiometabolic status **[6].**

Central adiposity and insulin resistance:
Smoking can coexist with lower average body weight but a comparatively adverse distribution of central adiposity. Waist circumference, visceral fat and insulin resistance should not be treated as interchangeable outcomes. Epidemiological results suggest that smoking-related risk may depend on abdominal obesity and baseline glycaemic status **[7].** Population data also highlight possible differences between current and former smokers, partly because cessation is often followed by weight gain or changes in health behaviour **[8]**. Neurobiological evidence provides a plausible connection between nicotine exposure and appetite regulation, but individual weight trajectories after quitting vary substantially **[9].** Observed weight gain should not be used to argue against cessation given the much broader cardiovascular and oncological harms of continuing cigarette use.

Inflammation, oxidative stress and metabolic signalling:
Chronic cigarette-smoke exposure promotes systemic inflammatory and oxidative pathways that may interfere with insulin signalling, endothelial function and adipokine balance. Studies involving metabolic, inflammatory and tobacco-related

endpoints indicate multiple overlapping routes rather than a single uniquely smoking-specific pathway **[10]**. Smoking can also interact with the metabolic consequences of alcohol intake, sedentary behaviour and unhealthy diet. Cross-sectional association is therefore vulnerable to correlated lifestyle exposures and reverse causation. Large community studies provide valuable signals but require adjustment for socioeconomic status, diet, activity and concurrent treatment **[11]**.

Epidemiological association: current and former smoking:
Cross-sectional research has linked cigarette smoking with elevated MetS prevalence in some populations, particularly when low HDL and hypertriglyceridaemia predominate **[12]**. Other work has demonstrated that ethnicity, inflammatory background and chosen diagnostic thresholds may modify the pattern **[13]**. A consistent direction in selected studies does not establish causality or uniform effect magnitude. More recent surveys have considered smoking intensity and metabolic status in diverse settings **[14].** Longitudinal cohort evidence is valuable for clarifying temporal ordering, although changes in smoking behaviour over follow-up complicate fixed-exposure analyses **[15].** Results may differ when comparisons use former smokers rather than strict never-smoker reference groups **[16].**

Dose-response and component-specific patterns:
Risk may be characterized by cigarettes per day, years of smoking and pack-years, each representing a different facet of exposure. These metrics are often self-reported and may not correspond precisely to nicotine absorption, puff volume or exposure from other products. Studies examining cumulative or categorical smoking exposure suggest that dose and sex can affect observed MetS associations **[17].** The triglyceride and low-HDL components may show stronger associations than blood pressure, for which acute nicotine effects and chronic measurement conditions can diverge. Smoking cessation and exposure changes should be considered when interpreting metabolic trajectories across follow-up intervals **[18].** Clinical findings among occupational groups further suggest that workplace, diet and stress exposures can affect observed associations **[19].**

Sex, age and population context:
Sex-specific patterns may reflect adiposity distribution, hormonal influences, smoking intensity and baseline disease burden. Risk estimates can shift meaningfully with age, employment status or regional smoking patterns. Several analyses from Asian populations' underscore that results should not be transferred without attention to local exposure prevalence and waist criteria **[20]**. Differences in socioeconomic conditions and healthcare access may also contribute to confounding and selection effects. The relationship may appear stronger in younger age strata for certain metabolic components while long-standing comorbidities dominate in older groups. Interpretation of population comparisons should therefore focus on component-level estimates and adjustment quality rather than treating a composite MetS label as sufficient **[21]**.

Smoking cessation and clinical prevention:
Smoking cessation has substantial well-established cardiovascular benefits, even though the short-term transition can involve increased appetite and weight. Guidance should integrate smoking cessation with supportive dietary, activity and weight management rather than trading one risk factor against another. Former-smoking analyses emphasize the need to document cessation duration and post-cessation change **[22]**. Replacement products and dual-use patterns further complicate assumptions about tobacco exposure and metabolic risk. Toxicant exposure may change without eliminating all cardiovascular risk, so comparative product studies should be interpreted in their clinical and regulatory context **[23]**. Studies of cardio metabolic outcomes after cessation should separate immediate changes in weight from later changes in vascular or metabolic function **[24]**. The literature shows the following studies related to it as in **Table 1**.

Table 1: Association between MetS and cigarette smoking observed across the included studies

Setting / Design / Sample	Smoking exposure and comparison	MetS definition / outcome	Main findings	Components most affected / modifiers	References
USA; community-based; cross-sectional; n=5,913	Never, former, current; intensity and pack-years	ATP III; cross-sectional MetS	Current smoking was associated with higher odds of MetS; adjusted ORs were generally around 1.2–1.4	Low HDL, high TG, and abdominal obesity; some sex and ethnicity interactions	**[16]**
Venezuela; population-based; cross-sectional; n=2,212	Non-smoker, former, smoker; intensity bands	Harmonized 2009 definition; cross-sectional	Smoking was associated with higher MetS odds (OR 1.54)	High TG, abdominal obesity, low HDL; inverse relation with hypertension	**[17]**
Japan; health screening center; cross-sectional; n=27,972 men	Never, former, current	Modified MetS definition; cross-sectional	Current smoking was positively associated with MetS (OR 1.59)	Stronger association in higher inflammation strata	**[18]**
South Korea; KNHANES; cross-sectional; n=14,607	Dual exposure, single exposure, former, never	LAP used as MetS proxy	Dual and single smoking exposure were associated with higher LAP	Suggests effect through central adiposity and triglycerides	**[19]**
South Korea; prospective cohort; n=4,542 men	Non-smoker, new smoker, ex-smoker, sustained smoker	WHO-WPR; 3-year incident MetS	Sustained smoking increased MetS risk; quitting effect reduced after weight-change adjustment	Weight gain after cessation acted as an important confounder	**[20]**
South Korea; cross-sectional; n=808	Non-smoker, former, current	Korean MetS criteria; cross-sectional	Overall MetS association was weak after adjustment	High TG and low HDL remained strongly associated with smoking	**[21]**
Japan; male office	Never, former, current;	Modified ATP III;	Smoking showed dose-related	Low HDL and high TG were	**[22]**

workers; cohort; n=3,649 baseline	cigarettes/day and pack-years	baseline and follow-up	increases in MetS and its components	most affected	
South Korea; national survey; cross-sectional; n=3,452 men	Cigarettes/day and pack-year quartiles	ATP III / Asian cut-offs; cross-sectional	Heavier smoking was associated with significantly higher MetS odds	Strongest effects for high TG and low HDL; alcohol interaction noted	**[23]**
South Korea; national cohort; n=6,099,717	Never, ex-smoker, current light/moderate, current heavy	Harmonized 2009 criteria; repeated screening and follow-up	Smoking increased risk of MetS development and reduced recovery, especially in heavy smokers	Triglycerides showed the strongest association	**[24]**
China; two cross-sectional samples; n=15,222 and 8,565	Never, ever, CSI tertiles, intensity bands	Chinese Diabetes Society 2013; cross-sectional	Current smoking and higher cumulative exposure were associated with higher MetS odds, especially in discovery sample	Low HDL and TG most consistently affected; stronger effects in younger adults	**[25]**
China; cross-sectional; n=8,169 men	Never, former, current; daily smoking intensity	Harmonized 2009 criteria; cross-sectional	Heavy smoking (≥40/day) was associated with higher MetS odds	High TG and low HDL most consistently affected; alcohol interaction present	**[26]**

Methodological limitations of the literature:

The prevalence of self-reported smoking introduces nondifferential and differential misclassification. Across cross-sectional studies, reverse causation is possible when people with metabolic disease quit smoking; this can make former smokers appear metabolically worse than current smokers. Differences in MetS diagnostic definitions, medication treatment, age structure and baseline adiposity may distort comparisons, and observational studies seldom capture all aspects of alcohol use, diet and socioeconomic conditions **[25].** Analyses that measure smoking status only at baseline may miss relapse or cessation during follow-up. More useful future studies will include repeated biochemical measures of exposure, longitudinal weight and waist assessments, component-specific reporting, preregistered confounder adjustment and sensitivity analyses for time-varying smoking status **[26].** Emerging regional studies continue to illustrate how metabolic associations differ across populations and measurement approaches **[27].** In the 2020–2022 KNHANES (n=15,359), Kim and Kim observed increased MetS odds among dual tobacco users (OR 1.48; 95% CI 1.12–1.94) and cigarette-only users (OR 1.19; 95% CI 1.01–1.41), compared with non-smokers **[28]**. A Thai Army conference abstract (n=16,668) reported a MetS prevalence of 21.1% and an association with smoking; its limited methodological details warrant caution **[29]**. Using Iranian STEPS surveys (n=40,046), Mohseni *et al.* reported interactions of smoking and physical inactivity with MetS and selected components **[30]**. Farhoud *et al.* found higher adjusted MetS odds among cigarette-smoking Palestinian university students (n=392; OR 3.48), in a setting with limited generalizability **[31]**.

Conclusion:

Cigarette smoking is associated with adverse cardiometabolic profiles, especially dyslipidaemia, but the size and direction of each MetS component association vary. The MetS composite should be interpreted with attention to diagnostic cut-offs exposure intensity and lifestyle confounding. Smoking cessation remains central to prevention and should be paired with weight-management and cardiovascular follow-up.

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