Magna Scientia Advanced Research and Reviews

eISSN: 2582-9394

Cross Ref DOI: 10.30574/msarr

Journal homepage: https://magnascientiapub.com/journals/msarr/

(REVIEW ARTICLE)

Check for updates

Literature Review: Smoking, obesity, and cardiovascular risk: evidence from recent studies

Sean Muhammad Rabbani [1], Dhihintia Jiwangga Suta Winarno [2] and Rosi Amrilla Fagi [3,*]

[1] *Medical Study Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.*
[2] *Department of Cardiothoracic Surgery, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital Surabaya, Indonesia.*
[3] *Department of Cardiovascular, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital Surabaya, Indonesia.*

Magna Scientia Advanced Research and Reviews, 2025, 15(02), 182-186

Publication history: Received 26 October 2025; revised on 08 December 2025; accepted on 10 December 2025

Article DOI: https://doi.org/10.30574/msarr.2025.15.2.0150

Abstract

This literature review examines the independent and combined effects of smoking and obesity on cardiovascular risk, with the aim of synthesizing recent evidence to clarify their roles in the development and progression of cardiovascular disease. The scope of this review covers observational studies, cohort analyses, and selected clinical investigations published in the past decade that evaluate associations between tobacco exposure, excess adiposity, and major cardiovascular outcomes, including coronary heart disease, stroke, and cardiovascular mortality. Across the reviewed studies, smoking consistently emerged as a strong and immediate risk factor through mechanisms involving endothelial dysfunction, inflammation, and thrombogenesis, while obesity contributed through metabolic disturbances such as insulin resistance, dyslipidemia, and chronic low-grade inflammation. Several studies further demonstrated a synergistic effect, in which the coexistence of smoking and obesity conferred a substantially higher cardiovascular risk than either factor alone. The evidence also indicates that smoking may attenuate some apparent protective effects of weight reduction, while obesity may amplify smoking-related vascular damage. In conclusion, recent literature underscores that smoking and obesity are not only independent but also interacting determinants of cardiovascular risk. Effective cardiovascular prevention strategies should therefore address both behaviors simultaneously, rather than in isolation, to achieve meaningful and sustained risk reduction.

Keyword: Smoking; Obesity; Cardiovascular Disease; Cardiovascular Risk; Metabolic Disorders

1. Introduction

Cardiovascular disease remains one of the leading causes of death worldwide and continues to pose a major public health challenge across both developed and developing countries. Despite advances in medical therapy and preventive strategies, the global burden of cardiovascular morbidity and mortality remains high, largely due to the persistence of modifiable lifestyle-related risk factors. Among these, smoking and obesity are two of the most prevalent and preventable contributors. Both conditions are widely distributed across age groups and socioeconomic strata, and their increasing coexistence reflects ongoing changes in lifestyle patterns, urbanization, and behavioral transitions in modern societies.

Smoking has long been established as a major cardiovascular risk factor due to its direct toxic effects on the vascular endothelium, promotion of inflammation, oxidative stress, and thrombus formation. These processes accelerate atherosclerosis and increase the likelihood of acute cardiovascular events such as myocardial infarction and stroke. On

* Corresponding author: Rosi Amrilla Fagi

the other hand, obesity contributes to cardiovascular risk primarily through metabolic disturbances, including insulin resistance, dyslipidemia, hypertension, and chronic low-grade inflammation. Excess adiposity also alters cardiac structure and function, placing an additional hemodynamic burden on the cardiovascular system. Although these two risk factors operate through different biological mechanisms, both ultimately converge on pathways that promote vascular damage and cardiac dysfunction.

In recent years, growing attention has been directed toward the combined impact of smoking and obesity on cardiovascular health. Epidemiological trends indicate that a substantial proportion of individuals who smoke are also overweight or obese, creating a dual-risk profile that may amplify cardiovascular harm. The interaction between smoking-related vascular injury and obesity-related metabolic stress suggests the possibility of synergistic effects that exceed the risks posed by either condition alone. However, cardiovascular prevention strategies in clinical practice and public health programs often continue to address smoking and obesity as separate entities, potentially underestimating the compounded risk when both are present.

Based on these considerations, the purpose of this literature review is to analyze recent evidence regarding the independent and combined effects of smoking and obesity on cardiovascular risk. This topic was selected in response to the growing need for integrated risk assessment and prevention strategies that reflect real-world patterns of risk factor clustering. The central hypothesis guiding this review is that smoking and obesity not only function as independent determinants of cardiovascular disease but also interact in a manner that significantly magnifies overall cardiovascular risk. A clearer understanding of this interaction is essential for optimizing preventive interventions, refining risk stratification, and ultimately reducing the long-term burden of cardiovascular disease.

2. Pathophysiological mechanisms of smoking and obesity in cardiovascular disease

The development of cardiovascular disease as a result of smoking and obesity involves complex and overlapping pathophysiological pathways. Both risk factors independently disrupt vascular homeostasis, promote inflammation, and accelerate atherosclerotic processes. When present together, these mechanisms may interact and intensify cardiovascular injury, leading to earlier onset and greater severity of cardiovascular disease.

2.1. Pathophysiological Effects of Smoking on the Cardiovascular System

Smoking exerts direct and indirect harmful effects on the cardiovascular system through multiple biological mechanisms. The inhalation of cigarette smoke introduces thousands of toxic compounds, including nicotine, carbon monoxide, and reactive oxygen species, into the systemic circulation. These substances induce endothelial dysfunction, which represents an early and critical step in the development of atherosclerosis. Endothelial injury impairs nitric oxide production, leading to reduced vasodilation, increased vascular tone, and enhanced platelet adhesion.

In addition to endothelial damage, smoking promotes a pro-inflammatory state characterized by increased circulating inflammatory mediators and adhesion molecules. This inflammatory environment facilitates the migration of monocytes into the vascular wall, their transformation into macrophages, and the formation of lipid-laden foam cells. Over time, this process contributes to the formation and progression of atherosclerotic plaques. Smoking also enhances oxidative stress, which oxidizes low-density lipoproteins and further accelerates plaque development.

Furthermore, smoking increases blood coagulability by elevating fibrinogen levels, enhancing platelet aggregation, and impairing fibrinolysis. These changes elevate the risk of thrombosis, which plays a central role in acute cardiovascular events such as myocardial infarction and ischemic stroke. Nicotine also stimulates the sympathetic nervous system, leading to increased heart rate, blood pressure, and myocardial oxygen demand, thereby placing additional strain on the heart.

2.2. Pathophysiological Effects of Obesity on the Cardiovascular System

Obesity contributes to cardiovascular disease primarily through metabolic, inflammatory, and hemodynamic pathways. Excess adipose tissue, particularly visceral fat, functions as an active endocrine organ that releases a wide range of adipokines, cytokines, and pro-inflammatory mediators. This state of chronic low-grade inflammation is strongly associated with endothelial dysfunction and accelerated atherosclerosis.

One of the most important metabolic consequences of obesity is insulin resistance, which promotes hyperglycemia and dyslipidemia. These abnormalities contribute to the formation of atherogenic lipid profiles characterized by elevated triglycerides, increased small dense low-density lipoproteins, and reduced high-density lipoproteins. Obesity is also

closely linked to hypertension through mechanisms involving increased sympathetic activity, sodium retention, and activation of the renin–angiotensin–aldosterone system.

From a structural perspective, excess body weight increases cardiac workload and leads to adaptive changes such as left ventricular hypertrophy and myocardial remodeling. Over time, these changes impair cardiac function and increase susceptibility to heart failure. Obesity is further associated with a pro-thrombotic state, characterized by elevated coagulation factors and reduced fibrinolytic activity, which contributes to the risk of acute thrombotic events.

2.3. Interaction Between Smoking and Obesity in Cardiovascular Pathophysiology

When smoking and obesity coexist, their pathophysiological effects do not simply add together but may interact in a synergistic manner. Smoking amplifies oxidative stress and inflammation, which can worsen the metabolic disturbances associated with obesity. At the same time, obesity may enhance the vascular vulnerability to smoking-related toxins by sustaining a chronic inflammatory environment. This interaction accelerates endothelial dysfunction, intensifies atherosclerotic plaque formation, and increases plaque instability, thereby elevating the risk of acute cardiovascular events.

In addition, both smoking and obesity negatively influence lipid metabolism and insulin sensitivity, creating a compounded metabolic burden. The coexistence of sympathetic overactivity from nicotine exposure and hemodynamic overload from excess body weight further increases cardiac strain. Consequently, individuals who are both smokers and obese may experience earlier onset of cardiovascular disease, faster disease progression, and poorer clinical outcomes compared with those exposed to a single risk factor.

3. Epidemiological evidence linking smoking, obesity, and cardiovascular risk

Epidemiological studies have consistently demonstrated that both smoking and obesity are major contributors to cardiovascular morbidity and mortality. Large population-based analyses, prospective cohort studies, and multinational investigations provide strong evidence that each risk factor independently increases the likelihood of developing coronary artery disease, cerebrovascular disease, and other cardiovascular complications. More recent studies have further highlighted the importance of evaluating the combined burden of these factors, as their coexistence is increasingly common in modern populations.

3.1. Cardiovascular Risk Associated with Smoking

Extensive epidemiological data indicate that smoking significantly increases the incidence of coronary heart disease, stroke, peripheral arterial disease, and cardiovascular mortality. The risk is dose-dependent and closely related to the number of cigarettes smoked per day as well as the duration of smoking exposure. Even low levels of tobacco consumption have been shown to increase cardiovascular risk, particularly in younger individuals.

Passive exposure to cigarette smoke also contributes to cardiovascular disease, suggesting that there is no completely safe level of tobacco exposure. Smoking cessation has been consistently associated with a progressive reduction in cardiovascular risk, with substantial benefits occurring within the first few years after quitting. However, former smokers may continue to carry a residual risk, particularly if cessation occurs later in life or in the presence of other cardiovascular risk factors.

3.2. Cardiovascular Risk Associated with Obesity

Obesity is a well-established predictor of cardiovascular disease across diverse populations. Epidemiological studies demonstrate a strong association between increased body mass index, central adiposity, and the risk of hypertension, type 2 diabetes mellitus, dyslipidemia, and cardiovascular events. Visceral obesity, in particular, has been shown to confer a higher cardiovascular risk than generalized obesity.

Long-term cohort studies reveal that obesity during early adulthood significantly increases the lifetime risk of cardiovascular disease, even in individuals who are otherwise metabolically healthy at baseline. Weight gain over time further amplifies this risk. Conversely, sustained weight reduction is associated with improvements in blood pressure, lipid profiles, and glycemic control, all of which contribute to a lower incidence of cardiovascular events.

3.3. Combined Impact of Smoking and Obesity on Cardiovascular Outcomes

An increasing number of studies have shown that the coexistence of smoking and obesity confers a substantially higher cardiovascular risk than either factor alone. Individuals who are both smokers and obese demonstrate higher rates of

myocardial infarction, stroke, and cardiovascular mortality compared with non-smokers of normal weight. This combined exposure is particularly concerning in younger populations, in whom early vascular damage may translate into premature cardiovascular disease later in life.

The interaction between smoking and obesity appears to adversely influence multiple intermediate risk factors, including blood pressure, lipid metabolism, glucose regulation, and systemic inflammation. Some epidemiological findings suggest that smoking may modify the relationship between body weight and cardiovascular risk, complicating traditional risk assessment based solely on body mass index. These observations underscore the importance of addressing both behaviors simultaneously in cardiovascular prevention programs.

4. Conclusion

Smoking and obesity represent two of the most significant and modifiable risk factors contributing to the global burden of cardiovascular disease. Evidence from recent pathophysiological and epidemiological studies clearly demonstrates that both factors independently promote vascular dysfunction, atherosclerosis, and adverse cardiovascular outcomes through distinct but overlapping mechanisms. Smoking primarily induces endothelial injury, oxidative stress, inflammation, and thrombogenesis, while obesity contributes through metabolic dysregulation, chronic low-grade inflammation, hemodynamic overload, and structural cardiac changes.

Importantly, growing evidence indicates that the coexistence of smoking and obesity does not merely result in an additive increase in cardiovascular risk, but rather exerts a synergistic effect that significantly amplifies disease progression and the likelihood of adverse cardiovascular events. This interaction accelerates vascular damage, worsens metabolic disturbances, and increases plaque instability, leading to earlier disease onset and poorer clinical outcomes, particularly in younger and economically productive populations.

From a clinical and public health perspective, these findings highlight the limitations of addressing smoking and obesity as isolated risk factors. Cardiovascular prevention strategies must adopt an integrated, multifactorial approach that simultaneously targets tobacco cessation, weight management, and associated metabolic abnormalities. Such comprehensive interventions are essential for effective risk reduction and for controlling the escalating global burden of cardiovascular disease.

In conclusion, understanding the combined impact of smoking and obesity is crucial for improving cardiovascular risk stratification, optimizing preventive strategies, and guiding future research. A dual-focus approach that addresses both behaviors concurrently offers the greatest potential for meaningful and sustained improvements in cardiovascular health outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] World Health Organization. (2023). Cardiovascular diseases (CVDs): Key facts. World Health Organization.

[2] Ambrose, J.A., & Barua, R.S. (2004). The pathophysiology of cigarette smoking and cardiovascular disease: An update. Journal of the American College of Cardiology, 43(10), 1731–1737. https://doi.org/10.1016/j.jacc.2003.12.047

[3] Csordas, A., & Bernhard, D. (2013). The biology behind the atherothrombotic effects of cigarette smoke. Nature Reviews Cardiology, 10(4), 219–230. https://doi.org/10.1038/nrcardio.2013.8

[4] Lavie, C.J., Milani, R.V., Ventura, H.O. (2009). Obesity and cardiovascular disease: Risk factor, paradox, and impact of weight loss. Journal of the American College of Cardiology, 53(21), 1925–1932. https://doi.org/10.1016/j.jacc.2008.12.068

[5] Van Gaal, L.F., Mertens, I.L., & De Block, C.E. (2006). Mechanisms linking obesity with cardiovascular disease. Nature, 444(7121), 875–880. https://doi.org/10.1038/nature05487

[6] Hubert, H.B., Feinleib, M., McNamara, P.M., & Castelli, W.P. (1983). Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham Heart Study. Circulation, 67(5), 968–977. https://doi.org/10.1161/01.CIR.67.5.968

[7] Yusuf, S., Hawken, S., Ounpuu, S., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study). The Lancet, 364(9438), 937–952. https://doi.org/10.1016/S0140-6736(04)17018-9

[8] Ng, M., Fleming, T., Robinson, M., et al. (2014). Global, regional, and national prevalence of overweight and obesity in children and adults. The Lancet, 384(9945), 766–781. https://doi.org/10.1016/S0140-6736(14)60460-8

[9] Huxley, R.R., Woodward, M. (2011). Cigarette smoking as a risk factor for coronary heart disease in women compared with men. The Lancet, 378(9799), 1297–1305. https://doi.org/10.1016/S0140-6736(11)60781-2

[10] Hall, J.E., do Carmo, J.M., da Silva, A.A., Wang, Z., & Hall, M.E. (2015). Obesity-induced hypertension: Interaction of neurohumoral and renal mechanisms. Circulation Research, 116(6), 991–1006. https://doi.org/10.1161/CIRCRESAHA.116.305697

[11] Pischon, T., Boeing, H., Hoffmann, K., et al. (2008). General and abdominal adiposity and risk of death in Europe. The New England Journal of Medicine, 359(20), 2105–2120. https://doi.org/10.1056/NEJMoa0801891

[12] Thurlow, L.R., Joshi, M., Yan, G., et al. (2016). Global epidemiology of end-stage kidney disease and associations with obesity and smoking. The Lancet Global Health, 4(5), e335–e345. https://doi.org/10.1016/S2214-109X(16)30030-8

[13] Bazzano, L.A., He, J., Muntner, P., Vupputuri, S., & Whelton, P.K. (2003). Relationship between cigarette smoking and novel risk factors for cardiovascular disease in the United States. Annals of Internal Medicine, 138(11), 891–897. https://doi.org/10.7326/0003-4819-138-11-200306030-00009

[14] Canoy, D., Boekholdt, S.M., Wareham, N., et al. (2007). Body fat distribution and risk of coronary heart disease in men and women in the European Prospective Investigation into Cancer and Nutrition in Norfolk cohort. Circulation, 116(25), 2933–2943. https://doi.org/10.1161/CIRCULATIONAHA.106.673756

[15] Bowman, T.S., Gaziano, J.M., Buring, J.E., & Sesso, H.D. (2007). A prospective study of cigarette smoking and risk of coronary heart disease in women. Journal of the American College of Cardiology, 50(3), 269–275. https://doi.org/10.1016/j.jacc.2007.02.075

[16] Klein, S., Burke, L.E., Bray, G.A., et al. (2004). Clinical implications of obesity with specific focus on cardiovascular disease. Circulation, 110(18), 2952–2967. https://doi.org/10.1161/01.CIR.0000145546.97738.1E

[17] Chiolero, A., Faeh, D., Paccaud, F., & Cornuz, J. (2008). Consequences of smoking for body weight, body fat distribution, and insulin resistance. The American Journal of Clinical Nutrition, 87(4), 801–809. https://doi.org/10.1093/ajcn/87.4.801

[18] Clair, C., Rigotti, N.A., Porneala, B., et al. (2013). Association of smoking cessation and weight change with cardiovascular disease among adults with and without diabetes. JAMA, 309(10), 1014–1021. https://doi.org/10.1001/jama.2013.1644