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A review about cardiovascular disease and risk factors

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Abstract

This overview discusses the causes and contributing factors of cardiovascular disease. Apart from the conventional risk factors for cardiovascular disease diabetes, hypertension, smoking, high cholesterol, and genetic predisposition a growing body of research suggests that environmental physical and chemical factors also contribute significantly to the rising incidence of non-communicable diseases. In the area, heart disease, or "CD," continues to be the leading cause of mortality. Premature deaths, or cardiovascular deaths in people under 72 years of age, are especially alarming since they represent approximately 60 million years of life that may be lost to the disease annually in Europe. To estimate the global health burden of cardiovascular disease, continuous surveillance and monitoring should form the foundation for the creation and execution of evidence-based prevention and treatment initiatives.

Keywords: Cardiovascular disease CVDs; High blood pressure HBP; Type 2 diabetes mellitus

1. Introduction

Globally, a significant cause of morbidity and mortality are cardiovascular diseases (CVDs). Numerous racial or ethnic groups in the US are impacted by CVDs, and this fact comes at a hefty cost. around 200 billion is anticipated to be spent annually on drugs, medical services, and lost productivity. In many adult populations, A sizable portion of this burden is caused by the underuse of preventative therapies and the improper management of risk factors for atherosclerotic cardiovascular disease, or ASCVD [1,2]. According to World Health Organization data, smoking is the primary cause of 10% of all CVDs [3]. Every year, tobacco use is thought to be the cause of about 6 million fatalities globally. In the USsmoking is directly associated with over 500,000 deaths, with 10% of these deaths being caused by secondhand smoke exposure. The notion that smoking raises the risk of myocardial infarction and deadly coronary artery disorders has been validated by epidemiologic research [4]. Furthermore, it has been demonstrated that smokeless and low-tar tobacco increases the risk of cardiovascular events. Less than half of the risk increase linked to active smoking is attributed to passive smoking, even though it has a 30% higher risk of ASCVD. [5,6]. In an effort to identify individuals who are most likely to have a cardiovascular event in the future and enable the implementation of preventative interventions, epidemiologic research has been conducted ever since the Framingham study. In their publication "Factors of risk in the development of coronary heart disease," Kannel et al. made "risk factors" well known [7]. During that time, the first clinical risk scores became well-known, and a logistic model was introduced that included seven risk factors, one of which was "cigarettes smoked" [8]. the coronary heart disease (CHD) risk profile that is most frequently employed. In order to evaluate the risk of CVD, gender-specific prediction equations were created, taking into consideration variables including age, diabetes, smoking, blood pressure categories, total cholesterol, and LDL cholesterol categories (9). The Adult Treatment Panel of the National Cholesterol Education Program (NCEP) in the US has accepted this score [10]. New causal evidence for both active and passive smoking was released fifty years after the original publication; however, it was removed in 2012 [5]. According to the 2014 study, "a policy banning smoking and

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a decrease in coronary events among individuals under 65 years of age" are causally associated, citing sufficient data to support a link between cardiovascular disorders and active smoking [6]. The 2018 study emphasized the need for prompt action to protect young adults' health and the potential risk associated with heavy e-cigarette use [11].

2. Risk Factors of Cardiovascular Disease

2.1. High Smoking Rate

Ten percent of deaths from cardiovascular disease (CVD) worldwide are caused by smoking, which is a significant preventable risk factor for the illness [12]. Despite the fact that tobacco control measures have decreased tobacco usage globally, the burden of smoking-related CVD risk still exists [14]. It is widely acknowledged that giving up smoking lowers the risk of CVD [14]. Those who quit smoking after 20 pack-years or more of smoking had a 40% decreased incidence of Compared to heavy smokers, CVD occurred within 6 years [15]. The data pointing to a dose-response connection between the risk of CVD and smoking intensity [16–17] .supported the rationale for quitting smoking as a CVD risk reduction approach. On the other hand, a systematic review [18] discovered that quitting smoking offered relatively little health benefits.

2.2. Obesity and type 2 diabetes mellitus

One of the main risk factors for type 2 diabetes mellitus development is "obesity"[19]. However, some studies have demonstrated that, although while obesity is typically associated with an increased risk of type 2 diabetes mellitus, there is a significant increase in risk associated with an enlarged waistline, a greater waist-to-hip ratio, or higher levels of VAT at any BMI level [20,21, 22]. Anthropometric indices of upper body/abdominal obesity or direct imaging measurements, like CT, have been used in several studies. Compared to BMI-matched people without type 2 diabetes, patients with the illness had higher blood pressure and bigger waistlines [23]. Type 2 diabetes mellitus and visceral or extreme obesity both raise the risk of cardiovascular problems and put a person at risk for irregularities in the circulatory system[24].High-risk variants of obesity and type 2 diabetes mellitus are co-occurring more commonly because of the increased risk of heart failure and other cardiovascular problems. This presents difficult therapeutic issues. Insulin resistance is associated with visceral and ectopic obesity, and this may mitigate the relationship between obesity, type 2 diabetes, and cardiovascular risk. It is commonly recognized that insulin resistance and the metabolic syndrome increase the risk of cardiovascular morbidity and death [25, 26]. Moreover, research has demonstrated that a person with the metabolic syndrome is more likely to experience cardiac dysfunction and heart failure [27]. The Uppsala Longitudinal Study of Adult Men found that having a metabolic syndrome (BMI >29.4 kg/m2), as opposed to having a larger waist circumference, increased the risk of heart failure by more than three times during the study's 20-year follow-up. This effect persisted even after taking established heart failure risk factors into account[28]. In a population free of cardiovascular illness, the MESA study also showed that heart failure is predicted by the metabolic syndrome [29].

2.3. Increases in Serum Total Cholesterol

Research has been done on the relationships between lipid profiles and CVD, which includes heart attacks and strokes. The elevated levels of conventional lipid indices, such as total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), were found to positively correlate with an increased risk of CVD incidence, as were the decreased levels of high-density lipoprotein cholesterol (HDL-C) in two large-scale prospective cohort studies[30, 31]. the connections between TC, LDL-C, HDL-C, and the incidence of CVD-related death risk[32, 33]. Because assessing the correlation between dyslipidemia and CVD events and all-cause mortality using a single lipid score has its drawbacks, some studies proposed that measuring ApoB should be the first priority in clinical practice, along with lipid ratios and lipoprotein, which had additional clinical value[34, 35]. Surprisingly, some research has indicated that derived lipid indices, like computed lipid ratio and apolipoproteins, may have a CVD predictive value similar to traditional lipid indices [36, 37].

2.4. High blood pressure

The structural alterations that hypertension brings about in several organs are the underlying reason for organ failure. To date, a large number of studies have established a link between high blood pressure HBP and various forms of hypertension-mediated organ damage HMOD [38, 39]. If target organ dysfunction is not treated, it eventually progresses from asymptomatic to symptomatic episodes of CVD[40, 41]. Current guidelines for hypertension state that, because it affects their management techniques, all patients with hypertension should have their HMOD evaluated during their clinical assessment. Asymptomatic HMOD detection may be a sign of uncontrolled hypertension [42, 43]. This problem with hypertension is [44].

2.5. Chronic kidney disease (CKD)

impacts 15-20% of adult individuals worldwide and is distinguished by either a poor glomerular filtration rate or significant albuminuria. CVD is particularly significant because it is the primary cause of death in this clinical population; however, CKD also raises the risk of many other adverse outcomes. Chronic kidney disease (CKD) is linked to several cardiovascular disease (CVD) events, such as peripheral artery disease, coronary heart disease, arrhythmias, heart failure, stroke, and venous thrombosis. Notably, severe CVD outcomes such heart failure, lower extremity amputations, and CVD death are particularly closely linked to chronic kidney disease (CKD). The wide-ranging effects of chronic kidney disease (CKD) on the cardiovascular system are likely caused by several pathophysiological mechanisms that connect the disease to cardiovascular disease. Common risk factors include volume overload, inflammation, anemia, altered bone mineral metabolism, diabetes, hypertension, and the presence of uraemic toxins are among these processes. Understanding the current state of CKD is necessary to accurately assess the risk of CVD in CKD cohorts. However, the majority of clinical guidelines employ CKD indicators in an inconsistent manner to predict CVD risk [45].

2.6. Alcohol intake and cardiovascular disease

There is debate concerning the link between alcohol use and globally, cardiovascular disease (CVD) is the primary cause of death [46]. When compared to either abstinence or excessive consumption, a number of observational studies have shown a J- or U-shaped epidemiologic relationship between light to moderate alcohol use and a lower risk of cardiovascular disease (CVD)[47]. However, it has been suggested that residual confounding explains the apparent heart benefits of alcohol because beneficial lifestyle, socioeconomic, and behavioral traits tend to correspond with moderate alcohol intake [48,49].

3. Conclusion

In Conclusion there are many factors that cause cardiovascular diseases and others that are less influential and still need a lot of studies in the future, such as the effect of alcohol on heart diseases and genetic factors. As for risk factors such as high blood fat levels and diabetes they have a significant effect and are a major cause of blocked arteries and their hardening, as well as the effect of kidney diseases and infection with epidemic and infectious diseases on heart diseases.

Compliance with ethical standards

Disclosure of conflict of interest

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References

[1] Weir, H. K., Anderson, R. N., King, S. M. C., Soman, A., Thompson, T. D., Hong, Y., ... & Leadbetter, S. (2016). Peer reviewed: heart disease and cancer deaths—trends and projections in the United States, 1969–2020. *Preventing chronic disease*, *13*.

[2] Johnson, N. B., Hayes, L. D., Brown, K., Hoo, E. C., Ethier, K. A., & Centers for Disease Control and Prevention (CDC). (2014). CDC National Health Report: leading causes of morbidity and mortality and associated behavioral risk and protective factors--United States, 2005-2013. *MMWR suppl*, *63*(4), 3-27.

[3] Katanoda, K., & Yako-Suketomo, H. (2012). Mortality attributable to tobacco by selected countries based on the WHO Global Report. *Japanese Journal of Clinical Oncology*, *42*(6), 561-562.

[4] Barua, R. S., & Ambrose, J. A. (2013). Mechanisms of coronary thrombosis in cigarette smoke exposure. *Arteriosclerosis, thrombosis, and vascular biology*, *33*(7), 1460-1467.

[5] Lauder, L., Mahfoud, F., Azizi, M., Bhatt, D. L., Ewen, S., Kario, K., ... & Böhm, M. (2023). Hypertension management in patients with cardiovascular comorbidities. *European Heart Journal*, *44*(23), 2066-2077.

[6] US Department of Health and Human Services. (2014). The health consequences of smoking—50 years of progress: a report of the Surgeon General.

[7] Kannel, W. B., Dawber, T. R., Kagan, A., Revotskie, N., & STOKES III, J. O. S. E. P. H. (1961). Factors of risk in the development of coronary heart disease—six-year follow-up experience: the Framingham Study. *Annals of internal medicine*, *55*(1), 33-50.

[8] Truett, J., Cornfield, J., & Kannel, W. (1967). A multivariate analysis of the risk of coronary heart disease in Framingham. *Journal of chronic diseases*, *20*(7), 511-524.

[9] Wilson, P. W., D'Agostino, R. B., Levy, D., Belanger, A. M., Silbershatz, H., & Kannel, W. B. (1998). Prediction of coronary heart disease using risk factor categories. *Circulation*, *97*(18), 1837-1847.

[10] Lipsy, R. J. (2003). The National Cholesterol Education Program Adult Treatment Panel III guidelines. *Journal of managed care pharmacy: JMCP*, *9*(1 Suppl), 2-5.

[11] Centers for Disease Control and Prevention. (2010). National Center for Chronic Disease Prevention and Health Promotion (US); Office on Smoking and Health (US). *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease: A Report of the Surgeon General*, *9*.

[12] Katanoda, K., & Yako-Suketomo, H. (2012). Mortality attributable to tobacco by selected countries based on the WHO Global Report. *Japanese Journal of Clinical Oncology*, *42*(6), 561-562.

[13] Park, S. K., Oh, C. M., Kim, E., Ryoo, J. H., & Jung, J. Y. (2024). The longitudinal analysis for the association between smoking and the risk of depressive symptoms. *BMC psychiatry*, *24*(1), 364.

[14] US Department of Health and Human Services. (2014). The health consequences of smoking—50 years of progress: a report of the Surgeon General.

[15] Duncan, M. S., Freiberg, M. S., Greevy, R. A., Kundu, S., Vasan, R. S., & Tindle, H. A. (2019). Association of smoking cessation with subsequent risk of cardiovascular disease. *Jama*, *322*(7), 642-650.

[16] Bhat, V. M., Cole, J. W., Sorkin, J. D., Wozniak, M. A., Malarcher, A. M., Giles, W. H., ... & Kittner, S. J. (2008). Dose-response relationship between cigarette smoking and risk of ischemic stroke in young women. *Stroke*, *39*(9), 2439-2443.

[17] 17- McNeill, A. (2004). Harm reduction. *Bmj*, *328*(7444), 885-887.

[18] Chang, J. T., Anic, G. M., Rostron, B. L., Tanwar, M., & Chang, C. M. (2021). Cigarette smoking reduction and health risks: a systematic review and meta-analysis. *Nicotine and Tobacco Research*, *23*(4), 635-642.

[19] Masters, R. K. (2023). Sources and severity of bias in estimates of the BMI–mortality association. *Population studies*, *77*(1), 35-53.

[20] Piché, M. E., Tchernof, A., & Després, J. P. (2020). Obesity phenotypes, diabetes, and cardiovascular diseases. *Circulation research*, *126*(11), 1477-1500.

[21] Neeland, I. J., Ross, R., Després, J. P., Matsuzawa, Y., Yamashita, S., Shai, I., ... & Eckel, R. H. (2019). Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *The lancet Diabetes & endocrinology*, *7*(9), 715-725.

[22] Tchernof, A., & Després, J. P. (2013). Pathophysiology of human visceral obesity: an update. *Physiological reviews*.

[23] Choi, H. N., Kim, Y. S., & Yim, J. E. (2024). Association of daily carbohydrate intake with intermuscular adipose tissue in Korean individuals with obesity: a cross-sectional study. *Nutrition Research and Practice*, *18*(1), 78.

[24] Haffner, S. M., Lehto, S., Rönnemaa, T., Pyörälä, K., & Laakso, M. (1998). Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *New England journal of medicine*, *339*(4), 229-234.

[25] Lakka, H. M., Laaksonen, D. E., Lakka, T. A., Niskanen, L. K., Kumpusalo, E., Tuomilehto, J., & Salonen, J. T. (2002). The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *Jama*, *288*(21), 2709-2716.

[26] Malik, S., Wong, N. D., Franklin, S. S., Kamath, T. V., L'Italien, G. J., Pio, J. R., & Williams, G. R. (2004). Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. *Circulation*, *110*(10), 1245-1250.

[27] Zhou, Y., Wang, C., Che, H., Cheng, L., Zhu, D., Rao, C., ... & He, K. (2023). Association between the triglyceride–glucose index and the risk of mortality among patients with chronic heart failure: results from a retrospective cohort study in China. *Cardiovascular Diabetology*, *22*(1), 171.

[28] Burger, P. M., Koudstaal, S., Dorresteijn, J. A., Savarese, G., van der Meer, M. G., de Borst, G. J., ... & UCC-SMART study group. (2023). Metabolic syndrome and risk of incident heart failure in non-diabetic patients with established cardiovascular disease. *International Journal of Cardiology*, *379*, 66-75.

[29] Liu, L., Lima, J. A., Post, W. S., & Szklo, M. (2021). Associations of time-varying obesity and metabolic syndrome with risk of incident heart failure and its subtypes: Findings from the Multi-Ethnic Study of Atherosclerosis. *International journal of cardiology*, *338*, 127-135.

[30] Sun, L., Clarke, R., Bennett, D., Guo, Y., Walters, R. G., Hill, M., ... & China Kadoorie Biobank Collaborative Group. (2019). Causal associations of blood lipids with risk of ischemic stroke and intracerebral hemorrhage in Chinese adults. *Nature medicine*, *25*(4), 569-574.

[31] Gu, X., Li, Y., Chen, S., Yang, X., Liu, F., Li, Y., ... & Gu, D. (2019). Association of lipids with ischemic and hemorrhagic stroke: a prospective cohort study among 267 500 Chinese. *Stroke*, *50*(12), 3376-3384.

[32] Curb, J. D., Abbott, R. D., Rodriguez, B. L., Masaki, K., Popper, J., Chen, R., ... & Yano, K. (2004). Prospective association between low and high total and low-density lipoprotein cholesterol and coronary heart disease in elderly men. *Journal of the American Geriatrics Society*, *52*(12), 1975-1980.

[33] Zhong, G. C., Huang, S. Q., Peng, Y., Wan, L., Wu, Y. Q. L., Hu, T. Y., ... & Hao, F. B. (2020). HDL-C is associated with mortality from all causes, cardiovascular disease and cancer in a J-shaped dose-response fashion: a pooled analysis of 37 prospective cohort studies. *European journal of preventive cardiology*, *27*(11), 1187-1203.

[34] Sierra-Johnson, J., Fisher, R. M., Romero-Corral, A., Somers, V. K., Lopez-Jimenez, F., Öhrvik, J., ... & Hamsten, A. (2009). Concentration of apolipoprotein B is comparable with the apolipoprotein B/apolipoprotein AI ratio and better than routine clinical lipid measurements in predicting coronary heart disease mortality: findings from a multi-ethnic US population. *European heart journal*, *30*(6), 710-717.

[35] 35.Walldius, G., & Jungner, I. (2006). The apoB/apoA-I ratio: a strong, new risk factor for cardiovascular disease and a target for lipid-lowering therapy–a review of the evidence. *Journal of internal medicine*, *259*(5), 493-519.

[36] Yokokawa, H., Yasumura, S., Tanno, K., Ohsawa, M., Onoda, T., Itai, K., ... & Okayama, A. (2011). Serum low-density lipoprotein to high-density lipoprotein ratio as a predictor of future acute myocardial infarction among men in a 2.7-year cohort study of a Japanese northern rural population. *Journal of atherosclerosis and thrombosis*, *18*(2), 89-98.

[37] Taskinen, M. R., Barter, P. J., Ehnholm, C., Sullivan, D. R., Mann, K., Simes, J., ... & FIELD Study Investigators. (2010). Ability of traditional lipid ratios and apolipoprotein ratios to predict cardiovascular risk in people with type 2 diabetes. *Diabetologia*, *53*, 1846-1855.

[38] Cuspidi, C., Sala, C., Tadic, M., Gherbesi, E., Facchetti, R., Grassi, G., & Mancia, G. (2019). High-normal blood pressure and abnormal left ventricular geometric patterns: a meta-analysis. *Journal of Hypertension*, *37*(7), 1312-1319.

[39] Juhola, J., Magnussen, C. G., Berenson, G. S., Venn, A., Burns, T. L., Sabin, M. A., ... & Juonala, M. (2013). Combined effects of child and adult elevated blood pressure on subclinical atherosclerosis: the International Childhood Cardiovascular Cohort Consortium. *Circulation*, *128*(3), 217-224.

[40] Lorenz, M. W., Markus, H. S., Bots, M. L., Rosvall, M., & Sitzer, M. (2007). Prediction of clinical cardiovascular events with carotid intima-media thickness: a systematic review and meta-analysis. *Circulation*, *115*(4), 459-467.

[41] Sehestedt, T., Jeppesen, J., Hansen, T. W., Wachtell, K., Ibsen, H., Torp-Petersen, C., ... & Olsen, M. H. (2010). Risk prediction is improved by adding markers of subclinical organ damage to SCORE. *European heart journal*, *31*(7), 883-891.

[42] Williams, B., Mancia, G., Spiering, W., Agabiti Rosei, E., Azizi, M., Burnier, M., ... & Desormais, I. (2018). 2018 ESC/ESH Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH). *European heart journal*, *39*(33), 3021-3104.

[43] Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Dennison Himmelfarb, C., ... & Wright, J. T. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Journal of the American College of Cardiology*, *71*(19), e127-e248.

[44] Vasan, R. S., Song, R. J., Xanthakis, V., Beiser, A., DeCarli, C., Mitchell, G. F., & Seshadri, S. (2022). Hypertension-mediated organ damage: prevalence, correlates, and prognosis in the community. *Hypertension*, *79*(3), 505-515.

[45] Matsushita, K., Ballew, S. H., Wang, A. Y. M., Kalyesubula, R., Schaeffner, E., & Agarwal, R. (2022). Epidemiology and risk of cardiovascular disease in populations with chronic kidney disease. *Nature Reviews Nephrology*, *18*(11), 696-707.

[46] Stanaway, J. D., Afshin, A., Gakidou, E., Lim, S. S., Abate, D., Abate, K. H., ... & Borschmann, R. D. (2019). Erratum: Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*.

[47] Fisher, N. D. L., Orav, E. J., & Chang, G. (2018). Effects of alcohol consumption on blood pressure in hypertensive women. *The American journal of drug and alcohol abuse*, *44*(2), 200-205.

[48] Roerecke, M., Tobe, S. W., Kaczorowski, J., Bacon, S. L., Vafaei, A., Hasan, O. S., ... & Rehm, J. (2018). Sex-specific associations between alcohol consumption and incidence of hypertension: a systematic review and meta-analysis of cohort studies. *Journal of the American Heart Association*, *7*(13), e008202.

[49] Stockwell, T., Zhao, J., Panwar, S., Roemer, A., Naimi, T., & Chikritzhs, T. (2016). Do "moderate" drinkers have reduced mortality risk? A systematic review and meta-analysis of alcohol consumption and all-cause mortality. *Journal of studies on alcohol and drugs*, *77*(2), 185-198.