CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187
https://doi.org/10.5281/zenodo.5875214

Available online at: http://www.iajps.com *Research Article*

CARDIOVASCULAR DISEASE PREVENTION, OVERVIEW OF RISK FACTORS

[1]HUSSAIN SAEED ALDHAMEN, [2]ABDULLAH SALMAN ALI BUEID, [3]ABDULLAH ALI ALSHAWAF, [4]ALI AHMED ALBAHRANI, [5]NAWI JABR ALSHAMMARI, [6]ANMAR ESSAM AMIN KOUDWARDIA, [7]NABIL HAMADY ALSHIKH, [8]MAJDI NAIF ALBESHRI, [9]AMIN SALEM ALKHATTABI, [10]BADRIA DAFFI ALBESHRI, [11]AMEEN MOUSA ALHAZMI, [12]MESHAL MOUSA ALHAZMI

Article Received: November 2021 **Accepted:** December 2021 **Published:** January 2022

Abstract:

Cardiovascular diseases (CVD) are a group of disorders of the heart and blood vessels which is the most significant cause of death globally. Despite the critical fatality rate 90% CVD can be prevented by taking necessary precautions. We conducted a search in literature to related topics concerning the prevention measures of CVD that are discussed in major electronic databases.

Health and wellbeing is one of the most primary and significant concerns for mankind. However this concern is constantly challenged by diseases and illnesses. While some of these diseases are fatal, some can be cured or their negative impacts could be minimized if diagnosed at early stages. The diseases that challenge the wellbeing of an organism can be categorized into two main categories based on the agent of the said disease. Early diagnosis plays a crucial role in secondary prevention. This requires intensive knowledge of risk factors contributing to CVD and different interactions among them. Some common risk factors of CVD include obesity, gender, age, blood lipids, and smoking. Since the invention of artificial intelligence CVD prediction has evolved into a new level, where machines are able to analyze millions of data sets and identify relations between different risk factors.

Corresponding author:
Hussain Saeed Aldhamen,

QR code

Please cite this article in press Hussain Saeed Aldhamen et al, ***Cardiovascular Disease Prevention, Overview Of Risk Factors .,*** *Indo Am. J. P. Sci, 2022; 09(01).*

INTRODUCTION:

Cardiovascular disease (CVD) is an umbrella term for a number of linked pathologies, frequently specified as coronary heart disease (CHD), cerebrovascular disease, peripheral arterial disease, rheumatic as well as congenital heart diseases as well as venous thromboembolism. Worldwide CVD accounts for 31% of death, most of this through CHD and also cerebrovascular accident [1].

Atherosclerosis is an older condition, which has been discovered in the mummies of Egyptian pharaohs. It is a pathological entity as well as its professional equivalent is cardiovascular disease (CVD). Only in recent times have its consequences got to epidemic percentages, mainly in Western affluent societies, although that epidemic is appearing to other non-Western populations today. The clinical symptoms of atherosclerosis are manifold, however still make up only the tip of the iceberg [2]. Somewhat atherosclerosis is global, the pathogenesis is complex, however it is most likely that more than 90% of the facts about the processes included are currently known, although how these truths fit into an overall understandable framework is less understood1.

In the industrialized world cardiovascular disease (CVD) accounts for virtually half of all fatalities. In a few of the places there has taken place a decline in death in current times, whereas in other countries it has boosted [2].When unrefined death rates drop below 15 per 1000 persons as well as life span at birth boosts to 55-60 years, the percentage of mortality as a result of CVD approaches 20-25%, and non-communicable conditions become a major public health problem [3].This has become the instance in the Eastern Mediterranean Region [3].Taking into account the proportionately large part of the total condition problem, deadly and also non-fatal, played by cardiovascular diseases it has actually come to be significantly essential to attempt to forestall that pattern.

CVD is a long-term status with a serious and often fatal results. In this review we discuss the main areas targeted for primary prevention of CVD

DISCUSSION:

Risk assessment of cardiovascular diseases (CVD):

Outright forecast of CVD risk of a person can be made using forecast graphes released or released by the WHO and also ACC/AHA. The referrals are created management of major cardiovascular threat elements with adjustments in lifestyle as well as prophylactic medicine therapies.

The ACC/AHA have created standards for the treatments of detection, management, or avoidance of CVD.In November 2013, The ACC and AHA launched upgraded risk-assessment standards for atherosclerotic CVD. Modifications as well as referrals consist of the adhering to [4-7].

- Stroke is contributed to the list of coronary occasions generally covered by risk prediction equations.
- The guidelines focus mainly on the 10-year risk of atherosclerosis-related occasions; they focus secondarily on the evaluation of life time risk for grownups aged 59 or more youthful without high shorter-term danger.
- The strongest predictors of 10-year threat are identified as age, sex, race, overall cholesterol, HDL-C, blood pressure, blood-pressure therapy status, diabetes, as well as current cigarette smoking status.
- Adjunct formulas for refining danger quotes by sex and race are supplied.
- If risk forecast has to be additional developed after threat forecast equations have been performed, the guidelines indicate that coronary-artery calcium scores, family history, high-sensitivity C-reactive protein, and also the ankle-brachial index can be used.
- The guidelines recommend that statin treatment be thought about in people whose 10-year atherosclerotic cardiovascular disease (ASCVD) occasion danger is 7.5% or greater.

Guidelines from AHA/ACC recommend use of a revised calculator for approximating the 10-year danger of developing an initial ASCVD event, which is defined as a nonfatal MI, death from CHD, or stroke (deadly or nonfatal) in an individual who was originally devoid of ASCVD [6].The calculator integrates the following danger variables: sex, age, race, total cholesterol, HDL, systolic blood pressure, treatment for raised blood pressure, diabetes mellitus, as well as cigarette smoking.
For patients 20-79 years of age who do not have existing scientific ASCVD, the guidelines suggest examining medical danger variables every 4-6 years. For patients with reduced 10-year risk (< 7.5%), the guidelines advise evaluating 30-year or lifetime risk in patients 20-59 year-old.

The guidelines note that regardless of the patient's age, doctors need to connect risk data to the patient as well

as refer to the AHA/ACC way of living guidelines, which cover diet and physical activity. For patients with raised 10-year risk, medical professionals must interact risk information and refer to the AHA/ACC standards on blood cholesterol and obesity.

Table 1. Risk factors for CVD and recommended European target goals: key points[8],[12].

Non-modifiable	
• Age	Men: ⩾45 years
	Women: >55 years
• Personal history of CHD	
• Family history of CHD	
Modifiable	**Target goals**
• High TC	<5.0 mmol/l (<190 mg/dl)
• High LDL-C	<3.0 mmol/l (<115 mg/dl)
• Low HDL-C	>1.1 mmol/l (>40 mg/dl)
• High triglyceride	<1.2 mmol/l (<150 mg/dl)
• Hypertension	<140/85 mm Hg (140/80 or 130/80 mm Hg in diabetes)
• Diabetes mellitus	Normalise glucose concentrations (HBA_{1C} below 7 mmol/l)
• Current tobacco use	Smoking cessation
• Obesity	Body mass index <25 kg/m^2
• Sedentary lifestyle	Exercise for 30 minutes 3–5 times weekly

HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TC, total cholesterol.

Reduction of risk factors and Primary Prevention of CHD:

Exercise

Workout is generally identified as having a favorable influence on most of wellness end results as well as its result on CVD is no various. Death and morbidity directly because of workout stays very little equalize to very extreme degrees of workout as well as in the frustrating bulk the advantages outweigh the threats [5].

In 2002, the influential Institute of Medicine (IOM) concurred that moderately intense activity is beneficial, but it doubled the daily goal from 30 to 60 minutes, concluding that one half hour is not sufficient to maintain a healthy weight or to achieve maximal health benefits [8]. The IOM guideline was issued in a report focused primarily on providing diet and nutrition advice for the American public. Although the IOM is to be lauded for highlighting the importance of physical activity as part of a healthy life style, its recommendation fails to balance the issue of efficacy with that of feasibility, both of which are essential to achieve a public health goal. Seven out of 10 American adults currently fail to meet the 30-minute guideline; indeed, one in three engages in no leisure-time physical activity at all [9]. Thus, the IOM recommendation is likely to undercut any motivation that the American public, already largely unable to adhere to less lofty guidelines, might muster to boost their activity levels. Based both on this fact and our review of available scientific data, we believe that the public health message should continue to be that moderately intense exercise for 30 minutes per day confers significant and measurable cardiovascular health benefits. We recognize, however, that a dose-response relationship between physical activity and cardiovascular outcomes exists such that another 30 minutes of exercise would, on average, be expected to confer additional protection against the development of cardiovascular disease (CVD) in populations with low baseline activity levels [9].

Diet:

Diet is thought to play a substantial role in CVD danger yet the body of evidence regarding its usage is not clear, neither are the guidelines overwhelmingly consensual.

The AHA advise the Dietary Approaches to Stop Hypertension (DASH) diet which is reduced in sugars and saturated fats, high in veggies, fruits and also

whole grains. This has actually been shown to as a method to lower blood pressure (BP) and also low-density lipoprotein cholesterol (LDL-C) which are independent risk aspects for CVD, but they do not attempt to reveal a straight decrease in CVD danger [10].

NICE advise decreasing saturated fat consumption, enhancing monounsaturated fatty acids and 5 parts of fruit and vegetables each day. They also suggest a high fibre diet and two parts of fish each week. They do acknowledge that they do not have proof that these changes will affect directly on CVD risk, however rather that they have advantages on various other areas of health. Significantly, most of the research studies referenced came from pre-1990s when dietary patterns were significantly different, and also nearly all their information were underpowered concerning CVD threat [9].

The ESC recommends switching from saturated to polyunsaturated fatty acids, an increase in fibre, fruit, veggie as well as fish intake as well as abstaining from alcohol as well as adherence to a Mediterranean kind diet. These have all been revealed to offer considerable reductions in CVD risk [9].

There is likewise clear evidence that industrially created transfats are causally connected to CHD [13] and also these are especially proscribed in ESC and also NICE guidelines.

The difference in between the referrals is multi-factorial. For instance, NICE standards on fiber intake appearance only at randomised regulated trials (RCTs) from the 1980s cf. the ESC which describes meta-analyses of data approximately the 2010s.

Concerning the suggestions on saturated fats, the ESC standards utilize designing information to theorize a CVD threat decrease from decrease in LDL-C rather than epidemiological proof or RCTs, whilst AHA guidelines do not comment especially on CVD risk. This is an area where NICE guidelines would take advantage of an update of its proof base and greater use of possible or epidemiological data to justify its suggestions.

In conclusion, there does seem to be good evidence for recommending diet plans high in fibre, vegetables and fruit consumption and reduced in basic sugars and salt. Adherence to a Mediterranean style diet regimen additionally appears to be cardioprotective.

Smoking:

Cigarette smoking has actually long been referred to as the significant threat factor for CVD. European data indicate that cigarette smoking doubles the 10 year CVD mortality rate [12] whilst 30% people CVD death is attributable to cigarette smoking [10].Not just is it unhealthy but this impact is dose relevant without risk-free lower limit seen. Secondhand smoke is likewise damaging as workplace direct exposure increases CVD threat by 30% and UK public health efforts consisting of cigarette smoking bans are related to a considerable fall in CVD occasions [9].

Stopping smoking is the solitary most affordable treatment in CVD avoidance, and also some benefits are seen within months of cessation [9],[10].All guidelines advise cessation, with brief as well as long-lasting advantages seen irrespective of size or strength of cigarette smoking habit.

Pharmacologically, using nicotine replacement therapy (NRT), buproprion (a norepinephrine dopamine reuptake inhibitor) and particularly varenicline (a partial nicotine receptor agonist) are universally advised. The two previous both improve abstaining prices by 50-- 70%, whilst varenicline increases abstaining [14].

Medication selection ought to be patient led, with a specific note to side-effect profiles. NRT formerly held warnings regarding its usage in those with CVD yet proof recommends that the benefits of cigarette smoking cessation surpass the threats [15]. Likewise recommended is doctor intervention as an affordable method of decreasing smoking cigarettes, especially efficient in secondary prevention post myocardial infarction (MI) [15].

E-Cigarettes are still controversial when it come to CVD danger. Whilst the reduction in poisonous items within cigarette smoke is certainly useful, animal versions of nicotine direct exposure still display CVD results with raised atherosclerotic plaques found in mice versions [16].Lasting information are waited for to identify the impact after humans.

Weight:

Having a body mass index (BMI) > 25 is a danger element for CVD with lowest all-cause mortality seen at BMI 20-25 but, because of enhanced all-cause death with BMI< 20, [17] reductions listed below this degree are not routinely suggested. No guidelines recommend details intervention relating to weight, yet advise upkeep of a healthy and balanced weight for reduction of CVD threat. BMI is a great predictor of CVD danger, particularly at greater degrees, yet there is

good proof that, whatsoever levels of BMI, visceral adiposity as well as liver fat are considerable factors of threat [16], [17].This helps to explain the diversification in the CVD risk profile seen in the overweight as it varies depending on the place of adipose deposition. There are actions to recommend that, alongside reduction in BMI, decrease in waist area as a proxy for reductions in visceral fat ought to come to be a crucial target for amelioration of CVD danger.

Alcohol:

Alcohol consumption is a questionable subject provided the well-known sequelae of normal as well as excess alcohol use. The problem exists as historically the evidence recommended a J-shaped contour when it concerns take the chance of, where abstaining is related to a boost in CVD compared to light drinkers, with low degrees of alcohol usage related to a lower degree of CHD [9],[18-20].Besides the understood physical results of alcohol, interfering with platelet aggregation, evidence from the INTERHEART study would certainly show up to substantiate these cases, showing reductions in risk for those with modest and also light use alcohol [18] A recent large mendelian evaluation by Holmes et al. [19] has, however, revealed that within a genetic subset for alcohol dehydrogenase, reductions in alcohol intake are connected with decrease in CVD risk throughout the spectrum of alcohol intake. This would certainly recommend that reductions in alcohol intake, even for moderate drinkers, are related to a reduction in CVD risk. It gets on this basis that the ESC standards suggest no safe level of alcohol consumption [9].NICE standards [11] were produced before this data being launched and proceed with advice on modest intake, encouraging not more than 4 devices per day for males and 3 for ladies, regardless of these being arbitrary numbers. The ACC also recommend small amounts along the same lines, with one to two drinks daily for males, as well as one beverage each day for ladies [20].As yet there does not seem to be a consensus of opinion relating to secure amounts, however high levels are evidently unhealthy.

Table 2. Evidence base for benefit of risk factor modification

Risk factor	Primary prevention of CHD
Smoking	Smoking cigarettes cessation will reduce the danger of death by 50%. Male that stop cigarette smoking have a lowered danger of myocardial infarction and also within 2-3 years the threat resembles those who have actually never smoked.
Diet	Nutritional modifications (decrease in saturated fat, cholesterol and a rise in polyunsaturated fat) can lead to lowered death from CHD. The addition of stanol esters and also plant sterols (which reduce cholesterol absorption) to food, for instance margarine, has been shown to minimize plasma cholesterol focus by around 10%. The impact corresponds with over intake causes variable results.
Cholesterol	Total serum cholesterol of > 6 mmol/l is related to a raised occurrence of CHD risk as well as danger of CHD death.
Exercise	Lack of physical fitness or exercise are associatedwith a raised danger of death from all causes and from cardiovascular disease both in middle-aged and older men.
Alcohol	Mortality from CHD is lowest in those that reported alcohol consumption 8 to 14 units of alcohol a week. Consuming over 21 systems a week boosts total mortality. Distinctions in between types and patterns of alcohol consumption remain vague.
Diabetes mellitus	Mortality from CHD increases concerning 3-fold to 10-fold and 2-fold to 4-fold in patients with kind 1 as well as type 2 diabetes mellitus, respectively.The UKPDS study showed that for every increment of 1% boost in HbA1c there was a 1.11-fold boost in the danger of CHD.
Blood pressure	Chronic hypertension is very closely pertaining to the danger of creating CHD. A decrease of 5 mmHg in diastolic blood pressure is associated with a 21% decline in threat of developing CHD.
Obesity	Although raised body mass index is associated with increased threat of CHD, there are no professional trials of the result of weight reduction on CHD morbidity as well as death.

CHD, coronary heart disease; UKPDS, UK Prospective Diabetes Study; HbA1c, glycated hemoglobin.

Hemostasis:

The cardioprotective effect of physical activity may partly result from its favorable influence on hemostatic factors. In the British Regional Heart Study, a 20-year follow-up of 3810 men aged 40 to 59 years, habitual leisure-time physical activity showed significant and inverse dose-response relationships with fibrinogen, plasma and blood viscosity, platelet count, coagulation

factors VIII and IX, von Willebrand factor, fibrin D-dimer, and tissue plasminogen activator antigen, even after adjustment for multiple possible confounders [26]. Initially sedentary men who became active in later life had a similar hemostatic profile to men with a consistent history of high activity, whereas men who became inactive in later life had a profile similar to those who had been inactive for the duration of follow up; these findings suggest that exercise must be current in order to produce favorable changes in hemostasis. However, sparse and inconsistent data from randomized trials testing the effect of physical activity performed at varying intensities on platelet function, blood coagulation, and fibrinolysis preclude definitive conclusions regarding these pathways [27].

CONCLUSION:

CVD is a major cause of disability as well as premature death throughout the globe. The underlying pathology of atherosclerosis establishes over several years and is generally advanced by the time signs and symptoms happen, normally in midlife. The threat of creating CAD raises with age, as well as consists of age > 45 years in men as well as > 55 years in women. A family history of early heart disease is also a danger factor, such as cardiovascular disease in the father or a brother detected before age 55 years as well as in the mother or a sister detected prior to age 65 years. Lots of conventional risk variables for CAD are related to way of life, consequently preventative therapy can be tailored to customizing certain factors.

Primary prevention programs focused on reduction of risk habits on a population-wide basis and also the identification, stratification, and also picked treatment of high-risk individuals prior to their advancement of condition should be cornerstones of any kind of approach to reduce the population's worry of CHD. Also, prevention strategies must start in childhood. These two methods need to be complementary.

REFERENCE:

1. WHO. Cardiovascular diseases (CVDs). 2016. Available at: http://www.who.int/mediacentre/factsheets/fs317/en/ (accessed 10 October 2016).
2. Lopez AD. Assessing the burden of mortality from cardiovascular diseases. World Health Star. Q. 1993;46:91–96.
3. Dodu SRA. Emergence of cardiovascular disease in developing countries. Cardiology. 1988;75:56–64.
4. Greenland P, Alpert JS, Beller GA, Benjamin EJ, Budoff MJ, Fayad ZA, et al. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2010;122:e584–636.
5. Cohen R, Budoff M, McClelland RL, Sillau S, Burke G, Blaha M, et al. Significance of a positive family history for coronary heart disease in patients with a zero coronary artery calcium score (from the multi-ethnic study of atherosclerosis) Am J Cardiol. 2014;114:1210–4.
6. Goff DC, Jr, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129:S49–73.
7. World Health Organization. Prevention of Recurrent Heart Attacks and Strokes in Low and Middle Income Populations. Evidence-Based Recommendations for Policy Makers and Health Professionals. Geneva: 2003. [Last accessed on 2017 Jul 25]. Available from: http://www.who.int/bookorders .
8. Institute of Medicine: Dietary reference intakes for energy, carbohydrates, fiber, fat, protein, and amino acids. Washington DC: The National Academies Press; 2002.
9. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J 2016; 37: 2315–2381.
10. Eckel RH, Jakicic JM, Ard JD, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013. 2014: 63(25_PA).
11. NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification. NICE Guideline CG181, 2016.
12. Perk J, De Backer G, Gohlke H, et al. European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). Eur Heart J 2012; 33: 1635–1701.
13. de Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and

risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. BMJ 2015; 351: h3978–h3978.
14. Kottke TE, Battista RN, DeFriese GH, et al. Attributes of successful smoking cessation interventions in medical practice: a meta-analysis of 39 controlled trials. JAMA 1988; 259: 2882–2889.
15. Tzivoni D, Keren A, Meyler S, et al. Cardiovascular safety of transdermal nicotine patches in patients with coronary artery disease who try to quit smoking. Cardiovasc Drugs Ther 1998; 12: 239–244.
16. Bhatnagar A. E-Cigarettes and cardiovascular disease risk: evaluation of evidence, policy implications, and recommendations. Curr Cardiovasc Risk Rep 2016; 10: 1–10.
17. Khosravi A, Akhavan Tabib A, Golshadi I, et al. The relationship between weight and CVD risk factors in a sample population from central Iran (based on IHHP). ARYA Atheroscler 2012; 8: 82–89.
18. Leong DP, Smyth A, Teo KK, et al. Patterns of alcohol consumption and myocardial infarction risk: observations from 52 countries in the INTERHEART case-control study. Circulation 2014; 130: 390–398.
19. Holmes MV, Dale CE, Zuccolo L, et al. Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. BMJ 2014; 349: g4164–g4164.
20. Garcia S. Alcohol Consumption and CVD: The case for moderation, 2015. Available at: http://www.acc.org/latest-in-cardiology/articles/2015/01/05/13/06/alcohol-consumption-and-cvd-the-case-for-moderation (accessed 25 October 2016).
21. Wilhelmsson C. Coronary heart disease: epidemiology of smoking and intervention studies of smoking. Am Heart J. 1988;115:242–249. Wilhelmsson C. Coronary heart disease: epidemiology of smoking and intervention studies of smoking. Am Heart J. 1988;115:242–249. Wilhelmsson C. Coronary heart disease: epidemiology of smoking and intervention studies of smoking. Am Heart J. 1988;115:242–249.
22. Rosenberg L, Kaufman D, Helmrich S, Shapiro S. The risk of myocardial infarction after quitting smoking in men under 55 years of age. N Eng J Med. 1985;313:1511–1514.
23. Law M. Plant sterol and sterol margarines and health. BMJ. 2000;329:861–864.
24. Turner RC, Millns H, Neil HA, Stratton IM, Manley SE, Matthews DR, Holman RR. Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus. UKPDS. BMJ. 1998;316:823–828.
25. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, Abbot R, Godwin J, Dyer A, Stamler J. Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. Lancet. 1990;335:765–774.