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Food prescription Programs: A new Paradigm in Clinical Care Food as a Medicine The role of Phytochemicals in Human Health

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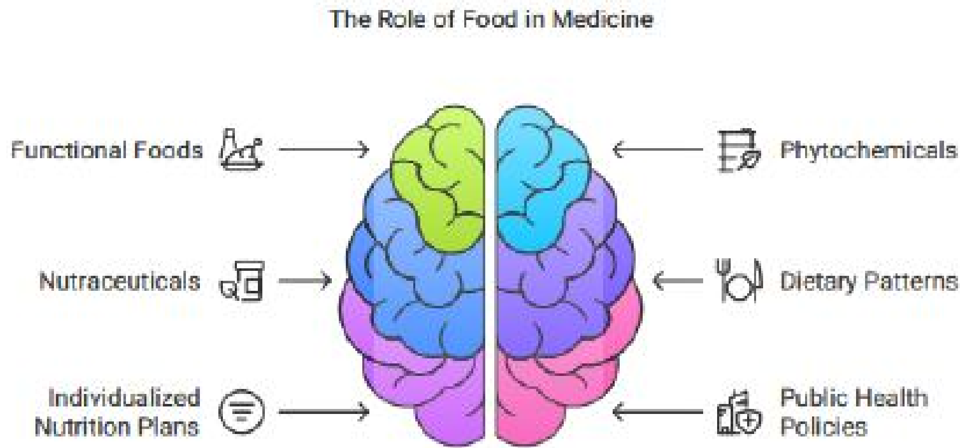
Abstract

The idea of "food as a medicine" has its origins in ancient healing systems and is currently receiving more attention in clinical settings and scientific inquiry. The therapeutic potential of food and its bioactive ingredients in the management and prevention of chronic illnesses, such as diabetes, obesity, cardiovascular disease, and neurodegenerative diseases, is examined in this article. Functional foods, phytochemicals, and nutraceuticals are highlighted for their ability to alter metabolic pathways, lower inflammation, and boost immune function. The study also emphasizes the increasing amount of data that shows better health outcomes are associated with dietary patterns like plant-based and Mediterranean diets. It also looks at how food-based treatments might be included into individualized nutrition plans and public health regulations. The "food as medicine" concept provides a sustainable and comprehensive strategy for illness prevention and health promotion in the twenty-first century by bridging the gap between nutrition research and medicine.

Key words : Functional Foods, Nutraceuticals, Chronic Disease Prevention, Dietary Interventions, Phytochemicals.

The primary cause of death in the US and throughout the world is cardiovascular disease (CVD), which is on the rise in low-, middle-, and high-income nations. Poor nutrition has become one of the leading contributors to modifiable metabolic and behavioral risks for CVD, accounting for an estimated 45% of cardiometabolic deaths in the United States, 36% of coronary deaths globally, 70% of new cases of diabetes worldwide, and significant loads of disability and early mortality. 3–5 Due to an increase in diet-related diabetes mellitus, obesity, and associated diseases such atherogenic dyslipidemia, hypertension, and inflammation, the United States' age-adjusted CVD death rate has increased since 2011, ending a 40-year drop[77,95,112,121,134].

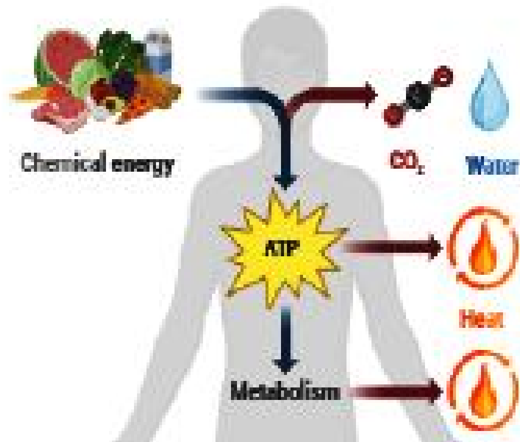
Low consumption of preventive dietary components (such as fruits, vegetables, whole grains, legumes, unsaturated fatty acids, and fish) and excessive consumption of



detrimental components (such as refined grains, processed meats, saturated fat, salt, and added sugars) are the causes of cardio metabolic risk. According to the verified Alternative Healthy Eating Index, Dietary Approaches to Stop Hypertension, and Mediterranean Diet scores, the average score for eating a healthy diet is low globally, ranging from 30 to 45 out of 100 (most healthy) across all areas. 8. Significant health inequalities by income, place of residence, race, and ethnicity are also brought on by cardiometabolic disorders and the lifestyle factors that contribute to them, such as poor nutrition quality and Food insecurity and other socioeconomic determinants of health are closely linked to these discrepancies. a social or economic situation at the home level where there is restricted access to enough food as well as reduced income, education, housing, and health care access, all of which are linked to historical ethnic and geographic inequality. Food poverty and inadequate nutrition are thus significant mediators of health inequities. Significant financial losses are also caused by cardiometabolic disorders. In the United States, the estimated direct and indirect economic expenses of diabetes were $327 billion in 2017, a 26% rise in only 5 years, and cardiovascular disease (CVD) were $405 billion in 20191; these costs are expected to double to $800 billion by 2030. [4,28,44,54,80,81,89,108,115,126].

Nutrition is also emphasized in clinical practice and population recommendations as the foundation of cardiovascular health throughout the lifetime and for the primary and secondary prevention of CVD. However, there have historically been few effective ways for clinicians to address the diets of their patients. This has been made worse by a lack of team-based care, electronic nutritional screening or

decision support, medical nutrition education (MNE), and health policies that provide incentives and reimbursements to support such an infrastructure. Similar to this, patients' problems of inadequate nutrition are rarely caused by their own knowledge or decisions alone, but rather by a variety of intricate social, environmental, cultural, agricultural, political, and economic factors. As a result, there haven't been many realistic, successful health sector initiatives to enhance nutrition, illnesses linked to food, and associated inequalities.



The sad irony is that one of the main causes of poor health has until far gone unaddressed by health services. With the introduction of "Food Is Medicine" (FIM) programs, this is currently altering throughout U.S. health policy and medical treatment. FIM is described as nutritional treatments based on food that are incorporated into health systems to promote health equality and cure or prevent illness (Central Illustration). New clinical treatment pathways and payment models, electronic health record (EHR) screening for food and nutrition security and diet quality, and increased MNE all support these initiatives, which are frequently paired with nutrition and culinary instruction.

This covers risk assessment and referral criteria as well as the creation of workable frameworks for incorporating FIM into clinical care and health systems. The current understanding of FIM is evaluated and discussed in this JACC State-of-the-Art Review, which covers the following topics: 1) developments in the underlying science; 2) efficacy evidence; 3) connections to food security, nutrition security, and health equity; and 4) related new national, state, and private health policies and initiatives[23,33,59,64,83, 131].

Global Burden of Chronic disease :

Illness Chronic illness prevention and management are global priorities because of the significant effects that the epidemiologic burden of these conditions has on people, health care systems, and economies throughout the world. With chronic illnesses causing 74% of fatalities globally (or 41 million deaths annually) and expected to increase to 52 million by 2030, One in three persons globally suffers from more than one chronic illness, with diabetes mellitus (DM), cardiovascular diseases (CVDs), and cerebrovascular disease (stroke and vascular cognitive impairment, or VCI) being the most prevalent chronic diseases.

Type 2 diabetes (T2D) accounted for 96% of the 529 million people with DM in 2021. By 2050, it is predicted that the number of people with DM will have doubled to over 1.31 billion worldwide, with 1.27 billion of those people having T2D. From 271 million in 1990 to 523 million in 2019, the number of people with all CVD (such as rheumatic heart disease, peripheral arterial disease, and coronary artery disease [CAD]) about doubled. 8. The majority of CVD is caused by ischemic heart

disease (IHD), which affected 197 million people in 2019. Global death rates for IHD increased from 1990 to 2019, reaching.14 million. At the same time, stroke and VCI are becoming serious worldwide public health issues[36].

The prevalence of all the chronic illnesses listed above is sharply increasing, most likely as a result of modern lifestyle choices or limited access to healthcare in emerging nations. Modifiable risk factors (MRFs) are responsible for the global burden of diseases (GBD), particularly for diabetes mellitus (DM), cardiovascular disease (CVD), and cerebrovascular disease (stroke and VCI)[35,66,95].

Currently, 1 billion people worldwide suffer with hypertension (HTN), a major risk factor and a separate chronic illness. One in three middle-aged individuals have HTN, As demonstrated, MRFs share risks for DM, CVD, stroke, and VCI, all of which may be addressed by similar lifestyle medicine (LSM) strategies[47,107].

Lifestyle medicine :

Lifestyle medicine is a branch of medicine that uses clinical, behavioral, motivational, and environmental concepts to prevent, cure, or manage chronic illnesses. By using a primary, secondary, and tertiary preventative strategy, it tackles the underlying root causes of chronic illnesses, which has both direct and indirect multiscale implications on general health, quality of life, well-being, and health care spending. It is composed of six pillars: drug usage, social relationships, stress management, sleep health, diet, and PA. The significance and potential benefits of each LSM pillar in treating the underlying MRFs of diabetes mellitus, cardiovascular disease, and cerebrovascular disease (particularly stroke and VCI) will be demonstrated in the sections that follow[29].

Dairy products :

The main source of saturated fat, cholesterol, and salt diet is dairy products. Numerous studies have employed observational designs instead of intervention trials, and they differ greatly in terms of the demographics, methodology, and particular dairy products assessed. Such heterogeneity may result in null results in meta-analyses due to a lack of statistical power. Furthermore, certain research supported by commercial organizations may have benefited the sponsor or sponsors, according to a recent meta analysis of industry-sponsored studies; nevertheless, the results were not statistically significant[9,16,24,127].

According guideline on lifestyle management, a balanced diet may contain some fat-free and low-fat dairy products. However, there are differing views on the information regarding the health impacts of certain dairy products and dairy products in general. Mixed findings have come from observational research on the relationship between dairy consumption and the risk of CVD and stroke. A systematic review of meta-analyses of prospective population studies (n ¼ 21) (4) and three systematic reviews and meta-analyses published between 2015 and 2017 assessed the relationship between dairy product intake and CVD risk and found conflicting results[2,27,39,102].

Furthermore, when compared to the fruits and vegetables only diet group alone, the

DASH (Dietary Approach to Stop Hypertension) dietary pattern, which is high in fruits, vegetables, and low-fat dairy, further decreased systolic blood pressure (BP) by 2.7 mm Hg (p ¼ 0.001) and diastolic BP by 1.9 mm Hg (p ¼ 0.002), even though both significantly decreased BP[5].

According to Chen *et al.*,[17] substituting polyunsaturated fatty acid or vegetable fat for 5% of energy intake from dairy fat was linked to a 24% and 10% lower risk of CVD, respectively, while substituting 5% of energy intake from other animal fat for dairy fat was linked to a 6% increase in CVD risk. The same was demonstrated by Song *et al.*, who discovered correlations between dairy consumption, cardiovascular disease, and all-cause mortality in a larger analysis (n ¼ 131,342). Lastly, dairy consumption has been linked to an increased risk of bone fractures , breast cancer, ovarian cancer, prostate cancer, and all-cause mortality in a number of recent studies[13,17,69,75,78,101,119,139].

For hip fractures, breast and ovarian cancer, and all-cause mortality, there is data that contradicts these findings. The foods that were used in place of dairy products or the kind of dairy product that was assessed may be the cause of the observed differences. Researchers have tried to isolate the potential impacts of particular dairy products, such cheese or butter, on the risk of type 2 diabetes, cardiovascular disease, and other consequences[32,79].

Added Sugar :

The 1950s saw the first evidence of a link between metabolic disorders, excessive consumption of added sugars, and the risk of CVD. In the 1960s, scientific evaluations sponsored by the sugar industry came to the conclusion that there wasn't enough data to link sugar intake to negative health effects. However, a rising body of research has lately established a causal relationship between increased intake of dietary added sugars and the mortality rates of CVD, stroke, and coronary heart disease (CHD). The two main dietary added sugars are the liquid high fructose corn syrup (HFCS), which has a slightly higher fructose to glucose ratio than sucrose (55:45 vs. 50:50, respectively), and the granular sweetener sucrose[48,62,72,91].

Added sugars are present in about 75% of packaged foods, but half of all added sugar consumption comes from the sugar-sweetened beverage (SSB) category, which includes soda, sweet teas, and fruit drinks. It is currently thought that the fructose and glucose moieties of sucrose and HFCS are not biologically comparable. The liver's uncontrolled absorption of fructose causes more hepatic lipogenesis than glucose. Pure fructose (and to a lesser extent HFCS) fed to young adults at up to 25% of energy requirements for just two weeks causes higher levels of atherogenic lipoproteins than pure glucose, according to a series of human feeding studies. These effects are independent of weight gain, but they are exacerbated by it (36). Triglycerides (TGs), LDL-C, and blood pressure all significantly increased with higher dietary free sugar intake compared to lower intake, according to a recent meta-analysis of 39 RCTs.

Regardless of weight increase, a pro-atherogenic risk profile is induced by excessive consumption of dietary added sugar.

According to the Health Professionals Follow-Up Study. An adjusted hazard ratio for CVD mortality at 25% of calories from added sugars was 1.0, according to an NHANES analysis of 11,733 healthy participants with a median follow-up of 14.6 years. In 2012, SSB intake (i.e., consuming one or more 8-ounce servings per day) was linked to 20.2% of the 197,981 diet-related deaths from CHD.

Last but not least, Li *et al*.[62] saw no decrease in CHD risk (and a tendency toward higher risk) when calories from saturated fat were replaced with similar calories from sugars and refined carbs in a cohort of 84,628 women and 42,908 men followed for 24 to 30 years. This result contrasted with a decrease in the risk of CHD when calories from saturated fat were substituted with those from whole-grain carbs or polyunsaturated or monounsaturated fats[12,22,93,100,120,124,136].

Legumes :

Legumes, which include a wide range of species like beans, peas, chickpeas, lentils, broad beans, soya beans, and lupins, are classified as the succulent seeds and pods of the botanical family Leguminosae or Fabaceae. They can be eaten as whole pods, their fresh shelled products, or as dried mature seeds, which are also referred to as pulses. The potential of legumes as ecologically friendly plant protein sources with several health advantages for people is becoming more widely acknowledged. Legumes are rich in protein, but they also include fiber, B vitamins, vital minerals like potassium, magnesium, and iron, and other bioactive substances[46,86].

Additionally, beans naturally contain minimal levels of saturated fat and carbs with a low glycaemic index. Consuming legumes has been repeatedly associated in the past with significant health advantages, including a lower risk of diabetes, cardiovascular disease (CVD), overweight/obesity, and several forms of cancer. As a result, an increasing amount of research is addressing the possible positive effects of legumes on cardio metabolic variables and cardiovascular health, emphasizing the benefit of include legumes in a regular diet. Legumes are included in a number of healthy eating indices and the dietary recommendations of Scientific Bodies and Medical Societies because of these factors. The primary cause of death worldwide and a significant factor in the years of healthy life lost as a result of disability is cardiovascular disease (CVD).

Consistent data suggests that nutrition can reduce the incidence of CVD by favorably modifying key risk variables, including obesity, diabetes mellitus, dyslipidemia, and hypertension. Through a number of etiological mechanisms, legumes, when included in a healthy, balanced diet, may help prevent the development of CVD [20,30,34,53,65,70,104,113,129,138].

Coffee :

One of the most popular drinks in the world is coffee. Its distinctively bitter flavor is caused by the abundance of bioactive polyphenols in it. The coffee infusion preserves the high potassium content of the original seeds in addition to polyphenols (mostly chlorogenic acid) and caffeine (an alkaloid with stimulatory qualities). Caffeine-naive people frequently have an abrupt rise in blood pressure when they consume coffee, whereas regular coffee

consumers typically do not. Long-term coffee drinking did not have any clinically significant impact on blood pressure or the risk of hypertension, according to a major meta-analysis. Coffee was not linked to the development of hypertension, according to the NHS [76,111,121,137].

Coffee's polyphenolic antioxidants have been shown to enhance insulin sensitivity and glucose metabolism. Consuming coffee, both caffeinated and decaffeinated, has been linked in a dose-dependent manner to a lower risk of type 2 diabetes in a number of major epidemiological studies. Compared to nondrinkers, approximately 186,000 participants in a recent research who drank more than four cups of coffee per day had an 18% lower chance of dying young over a 16-year follow-up[97,133,135].

Additionally, regular coffee users had a 7% to 12% reduced risk of dying young than nondrinkers, as well as lower incidence of stroke and digestive disorders, Additionally, studies have shown that people with established cardiovascular disease can safely maintain their regular coffee intake[38,106].

Drinking 3 to 4 cups per day was linked to risk reductions in all-cause mortality (RR: 0.83; 95% CI: 0.78 to 0.88), CV mortality (RR: 0.81; 95% CI: 0.72 to 0.90), and CV disease (RR: 0.85; 95% CI: 0.80 to 0.90), according to a thorough and statistically robust assessment of meta-analyses. Additionally, these scientists came to the conclusion that consuming more coffee was linked to an 18% decreased chance of developing cancer. Diterpenes found in coffee have the potential to increase cholesterol. Filtered coffee, however, mostly lacks these substances. Filtered coffee has no influence on blood lipid levels, according to several large prospective observational studies and meta-analyses[68,99,132].

Coffee consumption was substantially linked to a risk-adjusted decrease in the incidence of coronary artery calcification (CAC), according to a new observational research including 25,000 men and women. The subgroup that drank three to five cups of coffee per day had the lowest risk; their CAC scores were 40% lower than those of non-drinkers Coffee consumption and the incidence of heart failure were shown to have a U-shaped relationship (greater at lower and higher intakes, less at intermediate intakes) in a major meta-analysis. Furthermore, caffeine at doses as high as 500 mg/day (four or five cups of coffee) did not raise the frequency, inducibility, or severity of ventricular arrhythmias in five randomized placebo-controlled studies[18,87,92].

Tea :

Flavonoids and polyphenols are among the important antioxidants found in tea. Both short-term and long-term tea drinking enhanced endothelium-dependent flow-mediated dilatation of the brachial artery; water had no impact in a research that followed 66 patients with confirmed congenital heart disease who were randomized to drink either black tea or water in a crossover design. In CHD patients, both short-term and long-term black tea intake improved endothelial vasomotor dysfunction.

Green tea could possibly have positive cardiovascular benefits. Green tea drinking at 5 cups per day, which is more than most individuals may consume daily, was linked to

a 12% reduction in all-cause mortality in males and a 23% reduction in women.

THE BOTTOM LINE: TEA. According to several observational studies and meta-analyses, drinking tea (plain and other varieties) seems to be safe and may be linked to better blood lipids and cardiovascular disease health [8,25,55,25,61].

Mushrooms and Cardiovascular Health:

Consuming mushrooms may have cardioprotective effects through a variety of pathways, according to evidence from preclinical and clinical research. According to the majority of research, mushrooms synthesize vitamin D and have anti-inflammatory and antioxidant properties. These effects are caused by bioactive substances called beta-glucans (polysaccharides), ergothioneine (amino acid), and ergosterol (sterol), which have been shown in RCTs to have immunomodulatory, hypocholesterolemic, antiatherosclerotic, and antihypertensive effects. Additionally, eating mushrooms has been linked to a decrease in CVD-related comorbidities such obesity, type 2 diabetes, and metabolic syndrome. Furthermore, research on the connection between vitamin D insufficiency and the onset of chronic illness is gaining a lot of attention[14,37,45,74,98,103,122].

Ergosterol is a substance found in all mushrooms that, when exposed to UV light, transforms into vitamin D2. This kind of vitamin D is bioavailable to humans and will raise blood vitamin D levels after eating, according to RCTs. Three ounces of the well-known white button species, when fresh, may supply all 400 IU of vitamin D each day. Vitamin D levels can be affected by mushroom species, growing region, season, and UV light exposure length.

Eating mushrooms has also been linked to a decreased risk of breast cancer (109). Lastly, recent research has shown demonstrated that satiety is preserved when mushrooms are used in place of beef [49,51,52,60].

Fish and marine OM3 :

Fatty fish flesh is rich in marine-derived OM3, primarily docosahexaenoic acid (DHA) (22:6n-3) and eicosapentaenoic acid (EPA) (20:5n-3). Mechanistically, after consumption and absorption, OM3 are preferentially transferred into cellular phospholipids instead of TGs, which lowers serum TG levels and very low-density lipoprotein synthesis. They also have less significant effects on blood pressure, insulin signaling, and inflammatory pathways; they may also have an antiarrhythmic effect, particularly in cases of atrial fibrillation and sudden cardiac death[19].

Additionally, among the highest versus lowest quintiles of OM3 intake, higher levels of EPA/DHA in plasma and adipose tissue—both objective biomarkers of intake—were linked to a lower risk of incident MI and fatal CVD [21,41].

Concerns have been raised regarding the possible damage of consuming fish that contain putatively harmful substances, namely pollutants like methyl mercury. The comparatively high concentration of trimethylamine N-oxide in fish, a substance that may be harmful to cardiovascular disease and overall health, has also raised concerns. Although the study only included patients with CVD risk factors

and did not differentiate between the types of fish consumed, a recent study also suggested a negative outcome with fish protein consumption of all types when compared with plant protein in the realms of CVD and all-cause death. Instead of using OM3 derived from food, the majority of RCTs assessing its impact on CVD risk have used concentrated fish oil supplements[85,140].

According to a recent AHA research recommendation, people with prevalent CHD may benefit from treatment with marine OM3 supplements because of a slight decrease in mortality[7,118,123].

Vitamin B12:

Deficits in the vital micronutrient vitamin B12 (cobalamin) have been associated with serious neurological and hematological effects[140]. B12 supplementation is said to have a variety of health benefits, including enhanced mood, energy levels, memory, cardiovascular health, and skin, hair, and nail health. The findings of multiple major prospective trials have not demonstrated that folic acid and vitamin B12 supplements reduce the risk of incident or recurring cardiovascular disease, despite the fact that it is evident that these supplements lower homocysteine levels[1,56].

Despite decreased homocysteine levels, treatment did not appear to be related with a lower risk of major CV events when compared to a placebo. Daily treatment with folic acid, vitamin B6, and vitamin B12 for an average of five years decreased homocysteine levels and the risk of stroke, but not the risk of major cardiovascular events[67].

Men and women who had suffered an acute MI during the previous seven days were recruited for the NORVIT (Norwegian Vitamin Trial). Vitamin B12 reduced homocysteine levels by 27%, however it had no effect on the major endpoints of sudden death, stroke, and recurrent MI [10,26].

Three thousand six hundred eighty persons who had recently experienced a stroke were randomly assigned to either a low or high fixed-dose combination of vitamins B12, B6, and folic acid. Over a two-year follow-up period, a moderate decrease in homocysteine levels in the high-dose group did not affect vascular results. B12 malabsorption occurs when many individuals, especially older ones, do not have enough stomach acid to separate B12 from dietary protein[27].

As a result, supplementing is especially crucial for those over 50 and for vegans as well (146). Lastly, it is crucial to remember that excessive use of B12, B6, or folate might have negative consequences, including an increased risk of lung cancer in males, according to a recent study[11].

Vitamin B12 supplementation: The Bottom line :

The use of vitamin B12 supplements to prevent CVD is not supported by several big trials. Some groups are susceptible to deficiencies in Freeman *et al*. Trending Nutrition Controversies, Part II, JACC VOL. 72, NO. 5, 2018:553–6–8–564 Supplementing with B12 is advised (2.4 mg/day is the recommended daily amount)[43].

Berries :

By improving the elimination of

cholesterol in individuals with metabolic disorders, bilberry consumption in conjunction with whole grain and fatty fish reduced the incidence of CVD. Berries and berry anthocyanins have been shown to lower blood pressure in hypercholesteromic people in randomized controlled clinical trials. Wild blueberry drink, blueberry polyphenols, bilberry anthocyanins, and blackcurrant anthocyanins all enhanced endothelial functions as indicated by an increase in flow-mediated vasodilation (FMV) in both acute and long-term intervention trials. The effects showed a good correlation with the activity of gut microbiota and the plasma content of anthocyanins and their metabolites, 5-O-caffeoylquinic acid[110,125].

Studies on long-term dietary interventions and epidemiology suggest that berry juice and anthocyanins may help lower arterial stiffness. The effects of baked goods made with blueberries (Vaccinium angustifolium) on endothelial function (FMV) were comparable to those of blueberry drinks made with an identical quantity of freeze-dried blueberry powder[109].

Consuming berries and berry products over time may lower the risk factors for cardiovascular and metabolic illnesses and enhance the plasma lipid profile. The potential of a berry diet to reduce stress and promote healthy aging will be the focus of future study[57].

Dark chocolates :

Flavanols are the primary component responsible for the cardiovascular advantages of dark chocolate, which is also high in methylxanthines, caffeine, and other compounds. Research has demonstrated that flavanols can activate endothelial cells' NO synthase, which releases NO. This, in turn, activates smooth muscle cells' guanylate cyclase, raising cyclic guanosine monophosphate levels and lowering intracellular calcium ion concentration, which causes vasodilation44. Acute consumption of dark chocolate, as opposed to a placebo, can enhance endothelial function (as indicated by flow-mediated dilatation), according to a 45-person RCT [71].

Consuming dark chocolate considerably raised coronary artery diameter and endothelium-dependent coronary vasodilatation. flavanols can prevent platelet aggregation by encouraging NO44 release. Dark chocolate can dramatically lower shear stress-dependent platelet adhesion and platelet aggregation, according to two small-sample RCTs. Furthermore, pro-inflammatory cytokine production can be controlled by flavanols.[31,116].

This is especially crucial for postponing the onset of atherosclerosis. Inflammation is a significant risk factor for CVDs, particularly those associated with atherosclerosis, according to earlier research. Meta-analyses on dark chocolate's lipid-lowering properties have revealed that while eating dark chocolate and cocoa products can raise levels of high-density lipoprotein cholesterol, it can decrease levels of total cholesterol and low-density lipoprotein cholesterol8. Nevertheless, we found no indication in this MR investigation that the risk of 11 outcomes, excluding EH, was reduced, which is consistent with earlier findings[50,63,117].

Dark chocolate does have some cardiovascular advantages, according to the data from the intervention trials already

discussed. These studies only identified certain risk factors and parts of the pathophysiology of CVDs influenced by dark chocolate consumption, despite the fact that the pathophysiological processes underlying the onset and progression of CVDs are extremely complicated. Consuming dark chocolate does not appear to lower the risk of CVDs. Consuming dark chocolate may not have enough cardiovascular advantages to lower the risk of these conditions. Furthermore, the results of the majority of these small-sample controlled studies are not very reliable due to a number of constraints[3,15,58,128,141].

The increasing amount of scientific data highlights the significant impact that food has on controlling and preventing a variety of chronic illnesses in addition to preserving health. Common dietary ingredients, such as fiber-rich whole grains, anti-inflammatory spices, and antioxidant-rich fruits and vegetables, have shown therapeutic potential on par with or even better than pharmaceutical treatments. The "food as medicine" concept offers a sustainable, economical, and comprehensive approach to wellness as integrative and preventative methods become more and more prevalent in modern healthcare.

Future Scope :

The concept of "food as a medicine" is poised to play a pivotal role in the future of healthcare, nutrition, and public policy. Emerging fields such as nutrigenomics, metabolomics, and microbiome research are opening new frontiers in understanding how individual responses to food can influence health outcomes.

References :

1. Albert C.M., N.R. Cook and J.M. Gaziano (2008). *JAMA 299:* 2027–2036.
2. Alexander D.D., L.C. Bylsma, and A.J. Vargas, (2016). *Br J Nutr 115:* 737–50.
3. Almoosawi S., C. Tsang, L.M. Ostertag, L. Fyfe and E.A. Al-Dujaili (2012). *Food Funct. 3:* 1035–1043.
4. American Diabetes Association. (2018). *Diabetes Care. 41:* 917–928.
5. Appel L.J., T.J. Moore, and E. Obarzanek, (1997). *N Engl J Med 336:* 1117–24.
6. Arnett D.K., A. Khera and R.S. Blumenthal (2019). *JAMA Cardiol. 4:* 1043–1044.
7. Aung T., J. Halsey, and D. Kromhout, (2018). *JAMA Cardiol 3:* 225–34.
8. Bahorun T., A. Luximon-Ramma, and V.S. Neergheen Bhujun, (2012). *Prev Med 54* Suppl: S98–102.
9. Barnard N.D., W.C. Willett, and E.L. Ding, (2017). *JAMA 318:* 1435–6.
10. Bonaa K.H., I. Njolstad and P.M. Ueland, (2006). *N Engl J Med 354:* 1578–88.
11. Brasky T.M., E. White, and C.L. Chen, (2017). *J Clin Oncol 35:* 3440–8.
12. Bray G.A., and B.M. Popkin, (2014). *Diabetes Care 37:* 950–6.
13. Buehring G.C., H.M. Shen, H.M. Jensen, D.L. Jin, M. Hudes, and G. Block, (2015). *PLoS One 10:* e0134304.
14. Calvo M.S., A. Mehrotra, and R.B. Beelman, (2016). *Plant Foods Hum Nutr 71:* 245–51.
15. Chapman K., M. Holmes, and J. Seckl (2013). *Physiol. Rev. 93:* 1139–1206.
16. Chartres N., A. Fabbri, and L.A. Bero (2016). *JAMA Intern Med 176:* 1769–77.
17. Chen M., Y. Li, and Q. Sun, (2016). *Am J Clin Nutr 104:* 1209–17.

18. Choi Y., Y. Chang, and S. Ryu, (2015). *Heart 101:* 686–91.
19. Chowdhury R., S. Warnakula, and S. Kunutsor, (2014). *Ann Intern Med 160:* 398–406.
20. Collaborators G.B.D.D., (2017). *Lancet* (London, England) *393*(10184): 1958e72.
21. Del Gobbo L.C., F. Imamura, and S. Aslibekyan, (2016). *JAMA Intern Med 176:* 1155–66.
22. Di Nicolantonio J.J., S.C. Lucan, and J.H. O'Keefe, (2016). *Prog Cardiovasc Dis 58:* 464–72.
23. Downer S., S.A. Berkowitz, T.S. Harlan, D.L. Olstad, and D. Mozaffarian, (2020). *BMJ. 369:* m2482.
24. Drouin-Chartier J.P., D. Brassard, and M. Tessier Grenier, (2016). *Adv Nutr 7:* 1026–40.
25. Duffy S.J., J.F. Keaney, and M Holbrook, *et al*. (2001). *Circulation 104:* 151–6.
26. Ebbing M., O. Bleie, and P.M. Ueland, (2008). *JAMA 300:* 795–804.
27. Eckel R.H., J.M. Jakicic, and J.D. Ard, (2014). *J Am Coll Cardiol 63* 25 Pt B: 2960–84.
28. Economic Research Service, U.S.. Department of Agriculture. (2023). Food security in the U.S.: survey tools. Accessed July 22, 2023. https://www.ers.usda.gov/topics/food-nutrition-assistance/foodsecurity-in-the-u-s/survey-tools/
29. Edington D.W., *Am J Lifestyle Med. 14*(3): 274-277.
30. Ferreira H., M. Vasconcelos, A.M. Gil, and E. Pinto, (2021). *Crit Rev Food Sci Nutr 61*(1): 85e96.
31. Ferrucci L., and E. Fabbri. (2018). *Nat. Rev. Cardiol. 15:* 505–522.
32. Feskanich D., H.E. Meyer, T.T. Fung, H.A. BischoffFerrari, and WC. Willett (2018). *Osteoporos Int 29:* 385–96.
33. Food as medicine (2023). *Nat Med. 29:* 753–754.
34. Fung T.T., S.E. Chiuve, K.M. Rexrode, G. Logroscino, and F.B. Hu, (2008). *Arch Intern Med 168*(7): 713.
35. GBD (2021). *Lancet Neurol. 20*(10): 795-820.
36. GBD (2021). Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet*. 2023; *402*(10397): P203-P234.
37. Guillamon E., A. Garcia-Lafuente, and M. Lozano, (2010). *Fitoterapia 81:* 715–23.
38. Gunter M.J., N. Murphy, and A.J. Cross, (2017). *Ann Intern Med 167:* 236–47.
39. Guo J., A. Astrup, J.A. Lovegrove, L. Gijsbers, D.I. Givens and S.S. Soedamah-Muthu, (2017). *Eur J Epidemiol 32:* 269–87.
40. Hajat C., and E. Stein, (2018). *Prev Med Rep. 12:* 284-293.
41. He K., Y. Song and M.L. Daviglus (2004). *Circulation 109:* 2705–2711.
42. Holman H.R., (2020). *ACR Open Rheumatol. 2*(3): 167-173.
43. Institute of Medicine (US) (1998). Standing Committee on the Scientific Evaluation of Dietary Reference Intakes and its Panel on Folate, Other B Vitamins, and Choline. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: National Academies Press.
44. Jardim T.V., D. Mozaffarian, and S. Abrahams-Gessel, (2019). *PLoS Med.*

16.

45. Jo Feeney M., AM Miller, and P. Roupas (2014). Mushrooms—biologically distinct and nutritionally unique: exploring a "third food kingdom". Nutr Today 49: 301–307.
46. Joint FAO/WHO (1993). Food Standards Programme CAC. Codex classification of foods and animal feeds. Rome.
47. Kearney P.M., M. Whelton, K. Reynolds, P. Muntner, P.K. Whelton, and J. He, (2005). *Lancet. 365*(9455): 217-223.
48. Kearns C.E., L.A. Schmidt, and S.A. Glantz, (2016). *JAMA Intern Med 176:* 1680–5.
49. Keegan R.J.H., Z. Lu, J.M. Bogusz, J.E. Williams, and M.F. Holick, (2013). *Dermatoendocrinol 5:* 165–76.
50. Keller J., R. Gomez, G. Williams, A. Lembke, L. Lazzeroni, G.M. Murphy Jr, and A.F. Schatzberg (2017). *Mol. Psychiatry. 22:* 527.
51. Khatun K., H. Mahtab, P.A. Khanam, M.A. Sayeed, and K.A. Khan, (2007). *Mymensingh Med J 16:* 94–9.
52. Kohn J.B., (2016). *J Acad Nutr Diet 116:* 1520.
53. Krebs-Smith S.M., R.E. Pannucci, A.F. Subar, S.I. Kirkpatrick, J.L. Lerman, and J.A. Tooze, (2018). *J Acad Nutr Diet; 118*(9): 1591e602.
54. Kris-Etherton P.M., K.S. Petersen, and G. Velarde, (2020). *J Am Heart Assoc. 9:* e014433.
55. Kuriyama S., T. Shimazu, and K. Ohmori, (2006). *JAMA 296:* 1255–65.
56. Langan R.C., and A.J. Goodbred, (2017). *Am Fam Physician 96:* 384–9.
57. Lankinen M., M. Kolehmainen, T. Ja¨ a¨ skela¨ inen, J. Paananen, L. Joukamo, A.J. Kangas, P. Soininen, K. Poutanen, H. Mykka¨ nen, H. Gylling, M. Ores˘ ic˘ M. Jauhiainen, M. Ala-Korpela, M. Uusitupa, and U Schwab, (2014). *PLoS ONE, 9*(2): e90352 http:// dx.doi.org/ 10.1371/journal.pone.0090352.
58. Lee Y.S., B.J. Lorenzo, T. Koufis, and M.M. Reidenberg (1996). *Clin. Pharmacol. Ther. 59:* 62–71.(140).
59. Levi R., H. Seligman and D. Mozaffarian, (2023). *Accessed June 20:* 2023.
60. Li J., L. Zou, and W. Chen, (2014). *PLoS One 9:* e93437.
61. Li X., C. Yu, and Y. Guo, (2017). *Heart 103:* 783–9.
62. Li Y., A. Hruby, and A.M. Bernstein, (2015). *J Am Coll Cardiol 66:* 1538–48.
63. Libby P., (2006). *Am. J. Clin. Nutr. 83:* 456S–460.
64. Lichtenstein A.H., L.J. Appel, M. Vadiveloo, F.B. Hu, PM Kris-Etherton, and CM Rebholz, *et al*. (2021). *Circulation 144*(23): e472e87.
65. Lichtenstein AH, LJ Appel and M Vadiveloo, *et al*., (2021). *American Heart Association. Circulation. 144:* e472– e487.
66. Livingston G., J. Huntley and A. Sommerlad, (2020). *Lancet. 396*(10248): 413-446.
67. Lonn E., S. Yusuf and M.J. Arnold (2006). *N Engl J Med 354:* 1567–77.
68. Lopez-Garcia E., R.M. van Dam, and W.C. Willett, (2006). *Circulation 113:* 2045–53.
69. Lu W., H. Chen, Y. Niu, H. Wu, D Xia, and Y. Wu, (2016). *Nutr J 15:* 91.
70. Mach F., C. Baigent, A.L. Catapano, K.C. Koskinas, M. Casula and L. Badimon, (2020). *Eur Heart J 41*(1): 111e88.
71. Magrone T., M.A. Russo, and E. Jirillo, (2017). *Front. Immunol. 8:* 677.
72. Malik V.S., Hu F.B., Fructose and cardiometabolic health: what the evidence

from sugar-sweetened beverages tells us. *J Am Coll Cardiol* 2015 *66:* 1615–24.

73. Marengoni A., S. Angleman and R. Melis (2011). *Ageing Res Rev. 10*(4): 430-439.
74. Martin K.R., (2010). *J Med Food 13 :* 1340–1346.
75. McCann S.E., J. Hays, C.W. Baumgart, E.H. Weiss, S. Yao, and C.B. Ambrosone, (2017). *Curr Dev Nutr 1:* e000422.
76. Mesas A.E., L.M. Leon-Munoz, F. Rodriguez Artalejo, and E. Lopez-Garcia (2011). *Am J Clin Nutr 94:* 1113–26.
77. Micha R., J.L. Penalvo, F. Cudhea, F. Imamura, C.D. Rehm and D. Mozaffarian (2017). *JAMA. 317:* 912–924.
78. Michaëlsson K., A. Wolk, and S. Langenskiöld, (2014). *BMJ 349:* g6015.
79. Michaelsson K., A. Wolk, H. Melhus, and L. Byberg, (2017). *Am J Epidemiol 185:* 345–61.
80. Miller V., R. Micha, E. Choi, D. Karageorgou, P. Webb and D. Mozaffarian, (2022). *JAMA Netw Open. 5:* e2146705.
81. Miller V., P. Webb, and F. Cudhea, (2022). *Nat Food. 3:* 694–702.
82. Mohebi R., C. Chen, and N.E. Ibrahim, (2022). *J Am Coll Cardiol. 80:* 565–578.
83. Mozaffarian D., H.M. Blanck, K.M. Garfield, A Wassung, and R. A Petersen (2022). *Nat Med. 28:* 2238–2240.
84. Mozaffarian D., (2022). *N Engl J Med. 387:* 2014–2017.
85. Mozaffarian D., and J.H. Wu, (2011). *J Am Coll Cardiol 58:* 2047–67.
86. Mullins A.P., and B.H. Arjmandi, (2021). *Nutrients 13*(2): 519.
87. Myers M.G., (1991). *Ann Intern Med 114:* 147–50.
88. National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP). About chronic diseases. Centers for Disease Control and Prevention. https://www.cdc.gov/chronicdisease/about/index.htm. Accessed February 14, 2024.
89. National Research Council Division of Behavioral and Social Sciences and Education, Institute of Medicine. Committee on National Statistics, Food and Nutrition Board; Nancy Kirkendall, Carol House, Constance F. Citro, rapporteurs. Research opportunities concerning the causes and consequences of child food insecurity and hunger. 2013. Accessed July 21, 2023.
90. Nchanji E.B., and O. C. Ageyo, (2021). *Nutrients;* 13.
91. Nestle M., (2016). *JAMA Intern Med 176:* 1685–6.
92. Newcombe P.F., K.W. Renton, P.M. Rautaharju, C.A. Spencer, and T.J. Montague, (1988). *Chest 94 :* 90–94. (95)
93. Nielsen S.J., and B.M. Popkin, (2004). *Am J Prev Med 27:* 205–210.
94. Noncommunicable diseases. World Health Organization. (2024). https://www.who.int/newsroom/factsheets/detail/noncommunicablediseases#:w:text¼ Noncommunicable%20diseases%20 (NCDs)%20kill%2041,%2 D%20and%20middle%2Dincome%20countries. Accessed February 14, 2024.
95. O'Donnell M.J., S.L. Chin, and S. Rangarajan, (2016). *Lancet. 388*(10046): 761-775.
96. O'Hearn M., L. Lara-Castor, and F. Cudhea, (2023). *Nat Med. 29:* 982–995.
97. Park S.Y., N.D. Freedman, C.A. Haiman, L. Le Marchand, L.R. Wilkens, and V.W. Setiawan, (2017). *Ann Intern Med 167:* 228–35.

98. Poddar K.H., M. Ames, C. Hsin-Jen, M.J. Feeney, Y. Wang, and L.J. Cheskin, (2013). *Appetite 71:* 379–87.
99. Poole R., O.J. Kennedy, P. Roderick, J.A. Fallowfield, P.C. Hayes, and J. Parkes, (2017). *BMJ 359:* j5024.
100. Powell E.S., L.P. Smith-Taillie, and B.M. Popkin, (2016). *J Acad Nutr Diet 116:* 1543–50.
101. Qin B., P.G. Moorman, and A.J. Alberg, (2016). *Br J Cancer 115:* 1122–30.
102. Qin L.Q., J.Y. Xu, S.F. Han, Z.L. Zhang, Y.Y. Zhao, and I.M. Szeto, (2015). *Asia Pac J Clin Nutr 24:* 90–100.
103. Rahman M.A., N. Abdullah, N. Aminudin, (2016). *Crit Rev Biotechnol 36:* 1131–42.
104. Rebello C.J., F.L. Greenway, and J.W. Finley, (2014). *Obes Rev 15*(5): 392e407. https://doi.org/10.1111/obr.12144.
105. Ribeiro D., (2015). *Inflammation. 38:* 858–870.
106. Richardson T., J. Baker, P.W. Thomas, C. Meckes, A. Rozkovec, D. Kerr, (2009). *QJM 102:* 555–61.
107. Rippe J.M., (2018). *Am J Lifestyle Med. 12*(6): 499-512.
108. Rockefeller Foundation. (2021). True cost of food: measuring what matters to transform the U.S. food system. July 2021. Accessed June 17, 2022. https://www.rockefellerfoundation.org/report/true-cost-of-food-measuring-what-matters-totransform-the-u-s-food-system/
109. Rodriguez-Mateos A., R. Del Pino-Garcı´a, T.W. George, A. Vidal-Diez, C. Heiss and J.P. Spencer (2015). *Functional foods and nutrition Current Opinion in Food Science, 2:* 36–42.
110. Rodriquez-Mateos A., C. Rendeiro, T. Bergillos-Meca, S. Tabatabaee, T.W. George, C. Heiss, and J.P. Spencer (2013). *Am J Clin Nutr 98:* 1179-1191.
111. Ros E., and F.B. Hu, (2013). *Circulation 128:* 553–65.
112. Roth G.A., G.A. Mensah, C.O. Johnson, G. Addolorato, E. Ammirati, and L.M. Baddour, (2020). *J Am Coll Cardiol 76*(25): 2982e3021.
113. Roth G.A., G.A. Mensah and C.O. Johnson, *et al*. (2020). *J Am Coll Cardiol. 76*(25): 2982-3021.
114. Roth G.A., G.A. Mensah and CO Johnson, *et al*. (2020). *J Am Coll Cardiol. 76:* 2982–3021.
115. Scrafford C.G., X. Bi, J.K. Multani, M.M. Murphy, J.K. Schmier, and L.M. Barraj, (2019). *J Acad Nutr Diet. 119:* 599–616.
116. Selmi C., C.A. Cocchi, M. Lanfredini, C.L. Keen, and M.E. Gershwin, (2008). *Mol. Nutr. Food Res. 52:* 1340–1348.
117. Shrime M.G., (2011). *J. Nutr. 141:* 1982–1988.
118. Siscovick D.S., T.A. Barringer, and A.M. Fretts, (2018). *Circulation 137:* e1–18.
119. Song M., T.T. Fung, and F.B. Hu, (2016). *JAMA Intern Med 176:* 1453–63.
120. Stanhope K.L., A.A. Bremer, and V. Medici, (2011). *J Clin Endocrinol Metab 96:* E1596–605.
121. Steffen M., C. Kuhle, D. Hensrud, P.J. Erwin, and M.H. Murad, (2012). *J Hypertens 30:* 2245–54.
122. Stepien M., L. O'Mahony and A. O'Sullivan (2013). *J Nutr Sci 2:* e29.
123. Tang W.H.W., Z. Wang, and B.S. Levison, (2013). *N Engl J Med 368:* 1575–84.
124. Te Morenga L.A., A.J. Howatson, R.M. Jones, and J. Mann, (2014). *Am J Clin Nutr 100:* 65–79.
125. The Financial Burden of NCDs. (2024). NCD Alliance. https:// ncdalliance.org/

why-ncds/the-financial-burden-of-ncds. Accessed February 14, 2024.

126. The White House. Biden-Harris Administration national strategy on hunger, nutrition, and health. September 2022. Accessed September 30, 2022. https://www.whitehouse.gov/wp-content/uploads/2022/09/White-House-National-Strategy-onHunger-Nutrition-and-Health-FINAL.pdf.
127. Thorning T.K., A. Raben, T. Tholstrup, S.S. SoedamahMuthu, I. Givens, and A. Astrup, (2016). *Food Nutr Res 60:* 32527.
128. Toole J.F., M.R. Malinow, and L.E. Chambless, (2004). *JAMA 291:* 565–75.
129. Trichopoulou A., T. Costacou, C. Bamia, and D. Trichopoulos, (2003). *N Engl J Med 348*(26): 2599e608.
130. Tsao C.W., A.W. Aday and ZI Almarzooq, *et al.* (2023). *Circulation., 147:* e93–e621.
131. U.S. Department of Agriculture. Dietary guidelines for Americans, 2020-2025. 9th ed, December (2020). https://www.dietaryguidelines. gov/resources/2020-2025-dietary-guidelines-online-materials
132. Urgert R., and M.B. Katan, (1997). *Annu Rev Nutr 17:* 305–24.
133. Van Dam R.M., and F.B. Hu, (2005). *JAMA 294:* 97–104.
134. Wang D.D., Y. Li, A. Afshin, (2019). *J Nutr., 149:* 1065–1074.
135. Wedick N.M., A.M. Brennan, Q Sun, F.B. Hu, C.S. Mantzoros, and R.M. van Dam, (2011). *Nutr J 10:* 93.
136. Welsh J.A., A.J. Sharma, L. Grellinger, and M.B. Vos, (2011). *Am J Clin Nutr 94:* 726–34.
137. Winkelmayer W.C., M.J. Stampfer, W.C. Willett, and G.C. Curhan, (2005). *JAMA 294:* 2330–5.
138. World Cancer Research Fund/American Institute for Cancer Research. Continuous update Project expert report (2018). In: Recommendations and public health and policy implications; 2018.
139. Yang M., S.A. Kenfield, and E.L. Van Blarigan, (2015). *Int J Cancer 137:* 2462–9. (54)
140. Zeisel S.H. and M. Warrier, (2017). Annu *Rev Nutr 37:* 157–81.
141. Zhu J.T., R.C. Choi, G.K. Chu, A.W. Cheung, Q.T. Gao, J. Li, Z.Y. Jiang, T.T. Dong,and K.W. Tsim (2007). *J. Agric. Food Chem. 55:* 2438–2445.
142. Zhu Y., M. Xia, Y. Yang, F. Liu, Z. Li, Y. Hao, M. Mi, T. Jin, and W Ling, (2011). *Clin Chem, 57:* 1524-1533.