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Chapter

Role of Functional Food in Treating and Preventing Cardiovascular Diseases

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Abstract

Cardiovascular diseases (CVDs) are still a major cause of mortality worldwide and are a serious health problem. Various factors that contribute toward CVDs include hypertension, tobacco use, physical inactivity, diabetes mellitus, obesity and overweight, alcohol, dietary factors and psychosocial aspects such as stress, anxiety and depression. Nutraceuticals and diet are very important for prevention of CVDs. The potential of nutraceuticals or functional food in mitigating risk of CVDs is discussed. Functional food with health related properties include fruit and vegetable, fish, legumes, nuts, soya protein, coffee, tea, chocolate, animal based functional food. In addition, some diet plans have shown the potential of reducing the incidence of CVDs. This includes the Mediterranean, Dietary Approaches to Stop Hypertension (DASH), Okinawan and vegetarian diets. This chapter examines the risk factors of CVDs, including hypertension, tobacco usage, physical inactivity, diabetes mellitus, overweight and obesity. The chapter also brings to the fore, functional foods with properties related to health and effect of dietary patterns in the treatment and prevention of CVDs.

Keywords: non-communicable disease, cardiovascular diseases, nutraceutical, functional foods, dietary patterns

1. Introduction

Globally, non-communicable diseases (NCDs) account for 73% of all death with cardiovascular diseases (CVDs) and ischemic heart disease (IHD) as the main contributors of cardiovascular mortality in 2017 [1]. Cardiovascular, respiratory as well as associated disorders (CVRDs) are predominant subgroup of NCDs and are major causes of morbidity and mortality in developing and developed countries. In 2012, it was estimated that 55.9 million people died around the world and NCDs accounted for 37.9 million of those deaths. Specifically, CVRDs led to 23.9 million deaths [2]. **Table 1** shows the impact of CVRDs on the total deaths in different countries based on the income group. Cardiovascular diseases killed 17.5 million; respiratory diseases led to 4.0 million deaths; diabetes mellitus to 1.5 million and diseases related to kidney accounted for 864 000 deaths, respectively [4]. In addition, NCDs account for about 35% (around 2.6 million) of all deaths in sub-Saharan Africa and this makes NCDs the second most common cause of death after a combination of communicable, maternal, neonatal, and nutritional related diseases [5]. Researchers

Functional Foods - Phytochemicals and Health Promoting Potential

Indicator	Low income	Lower middle income	Upper middle income	High income	Total (world)
Population	850000	2510000	2430000	1290000	7060000
Total deaths	7450	19900	16900	11700	55900
CVRD deaths	1540	7780	9080	5530	23900
CVRD deaths	21%	39%	54%	47%	43%
CVRD death by caus	se	_			
CVDs	999	5220	6860	4440	17500
DM	135	549	559	254	1497
RD	309	1630	1460	645	4040
Kidney diseases	99	378	197	190	864

Note: CVRD = Cardiovascular, respiratory, and related disorder, CVDs = Cardiovascular diseases, DM = Diabetes mellitus, RD = Respiratory disorder. [2, 3].

Table 1.

Impact of CVRDs on the total deaths in different countries based on the income group in 2012. Thousands, unless otherwise noted.

estimate that more than three-quarters of deaths will be due to NCDs by the end of 2030 and more deaths in developing countries will be attributed to CVDs alone than contagious diseases such as malaria, tuberculosis and HIV/AIDS [6].

Cardiovascular diseases are complex and composite diseases that are characterised by high serum lipids and triglycerides, cholesterol, elevated plasma fibrinogen and agglomeration factors with increased production of platelet as well as disturbance in metabolism of glucose [7, 8]. They are a broad category of diseases involving the heart and blood vessels causing coronary artery diseases such as angina which can lead to heart attack, heart failure, hypertensive heart diseases, stroke, and many other problems. [9]. Cardiovascular diseases are still the leading cause of mortality globally leading to 12.3 million and 17.6 million deaths in 1990 and 2016, respectively [10–12]. Stroke and coronary artery disease result in of 80% and 75% CVD deaths in male and females, respectively [12].

Various epidemiological studies have demonstrated that diet habits and healthy life style might prevent chronic diseases such as CVDs but poor habits aggravate these diseases [8, 13]. Individuals that consume large amount of fruits, vegetables and sea food are less vulnerable to CVDs incidence [14]. The role of dietary factors such as sodium and saturated fats known to increase the risk of CVDs has been substantially explored [15]. The perception that food does not only furnish fundamental nutrition but can also play a role in preventing diseases and assure good health and life is now gaining attention. High intake of food that is calorie dense, poor nutrition, highly processed and easy to absorb food can contribute to inflammation of system, low insulin vulnerability as well as a group of metabolic diseases which include obesity, high blood pressure, dyslipidemia, and diabetes mellitus [16]. Food that furnish a health benefits apart from basic nutrition such as reducing high blood total cholesterol as well as low-density lipoprotein cholesterol are called functional foods.

2. Risk factors for cardiovascular diseases

The traditional risk factors for CVDs (**Table 2**) have been extensively researched and the dietary factor is important since it leads to high risk factors for CVDs such

Non-modifiable	Metabolic	Lifestyle	Novel	
Family history	Diabetes mellitus	Diet	Oxidative stress	
Advancing age	Obesity	Smoking	High homeostatic factors	
Family history	Hypertension	Physical activity	Small dense low density lipoprotein-C	
	Hyperlipidemia		High lipoprotein level	
	Metabolic syndrome		High homocysteine level	
			High inflammatory marker	
[17, 18].	$(\Delta)(\Delta)$			
Table 2. <i>Risk factors of cardiova</i>	scular diseases.			

as hypertension and dyslipidemia. However, the dietary factor phenomenon has not been fully investigated [3]. A 2011 global report indicated that hypertension contributed to 13% of CVDs deaths, tobacco 9%, physical inactivity 6%, diabetes mellitus 6% and obesity 5% of global deaths [19, 20].

2.1 Hypertension

Hypertension is systolic blood pressure values ≥140 mmHg and/or diastolic blood pressure values \geq 90 mmHg. The relationship between hypertension and CVDs has been investigated in different studies [21, 22]. Hypertension exhibits an independent interminable relationship with the incidence of various CVDs such as stroke, heart failure and peripheral arterial [23, 24]. Hypertension is commonly without symptoms which silently damage the arteries that furnish the heart, brain, kidneys and other vital organs with blood and produce various structural changes. Various epidemiological, animal and genetic studies have confirmed that excessive intake of sodium increases blood pressure. For example, excessive consumption of sodium (>5 g sodium per day as defined by World Health Organisation) [25] produces a significant rise in hypertension and is associated with the onset of hypertension and its cardiovascular complications [26, 27]. By contrast, low intake of sodium reduces hypertension prevalence and is associated with low cardiovascular morbidity and mortality rate [28]. As a result, a common nutritional plan to minimise the incidence of hypertension includes achieving and maintaining a healthy body weight; consumption of a diet rich in minerals such as calcium, phosphorus, and magnesium as well as moderate consumption of alcoholic beverages and sodium [29].

2.2 Tobacco use

There are more than one billion smokers around the world and in 2013, tobacco usage accounted for more than 6.1 million deaths [18]. This estimation covers vulnerability to passive smoking (second hand smoke) which increases CVDs risk by 25 to 30% and public smoking bans substantially decrease the rate of heart attacks [30]. The most smoked form of tobacco is cigarette. More than 80% of tobacco users live in lower middle income countries and this number is expected to increase in the next decade [31]. Most smokers in lower middle income countries are male but this is not the case in high income countries. Early cessation of smoking contributes to substantial lower incidence of reinfarction within 1 year in patients who have had a heart attack and decreases the possibility of instant cardiac death in patients with CVDs [32]. There are two CVDs challenges associated with the use of tobacco products. Firstly, the rate of smoking is higher in the poorest populations of the world [33] and the second problem is smoking among girls [34]. The risk of IHD to tobacco smokers is 2–3 times higher than non-smokers, stroke is 1.5 times higher and lung cancer is 12 times higher. These risks are related to age gradient, with younger age group having higher relative risk (5–6 times) and these are similar for men and women [35].

2.3 Physical inactivity

The World Health Oranisation highlighted the fact that one in four adults is not physical active enough across the globe and the challenge of reduced physical activity increases as the income of a country increases [36]. Low physical activity eventually leads to obesity which has gradually increased throughout the past decades both in developing and developed countries. Low rate of atherosclerotic process, including improvement of endothelial dysfunction, low systematic inflammation and stroke that go along with physical activity, could explain the protective role of physical activity on CVDs risk [37]. Numerous epidemiologic studies conducted with non-identical and big populations have demonstrated that physical activity has protective effect on coronary arteries disease. For example, physical activity reduces blood pressure and the prevalence of hypertension by reducing vascular resistance and terminating the action of the sympathetic nervous and the renninangiotensin systems. Figure 1 shows the possible channels of physical activity that assist in decreasing the likelihood of CVDs. In 41837 women of age group between 55 and 69 years, Folsom et al. [38] study found that hypertension incidence decreased by 10% and 30% in participants with moderate and higher levels of physical activity than those with low levels of physical activity. The biological pathways support the usefulness of physical activity in decreasing the likelihood of stroke including ischemic and hemorrhagic. The possible ramifications on the likelihood of ischemic might be due to the mechanisms that reduce the development of atherosclerotic; while the possible ramifications on the likelihood of hemorrhagic stroke disease might be attributed to low blood pressure as well as additional associated risk factors. Nonetheless, there are conflicting findings from different studies about the relationship of physical activity and the incidence of stroke, with few studies showing relationships or no relationships [39, 40]. These disagreements are likely the result of the type of the study design, population, definition and evaluation of physical activity of the different studies.

2.4 Diabetes mellitus

Diabetes mellitus (DM) is caused by insufficient production of insulin by the pancreas or resistance by end-organ tissues and presents as a high blood glucose. There are three types of DM, namely, type 1, type 2, and gestational diabetes. Type 1 DM is an autoimmune disorder and usually takes place in early childhood and adolescents, gestational DM occurs during the second or third trimester of pregnancy, increases the future risk of those patients to type 2 DM [41–43]. Type 2 DM, the most common form accounts for 90–95% of diagnosed DM and continues to be rapidly growing worldwide and in the USA [44]. Globally, the prevalence of DM is escalating and its incidence in 1985 was 20 million compared to 382 million in 2014 [45]. The current estimation by the International Diabetes Federation expects that 592 million people will have DM by 2035 [46]. China and India have highest number of people with diabetes estimated at 69 million and 109 million, respectively and these numbers are expected to rise to 123 million and 150 million by 2040 [47]. A



Figure 1.

Possible mechanisms of physical activity that help in decreasing CVDs risk [37].

close association exists between DM and CVDs. The most familiar cause of mortality and morbidity in diabetic patients is CVDs since DM results in complications of both microvascular and macrovascular. Complications such as IHD, ischemic stroke, and amputations because of foot infections are examples of macrovascular. The comparative risk for CVDs morbidity and mortality in adults with DM varies from 1 to 3 in men and from 2 to 5 in women to those with no DM [48]. Many diabetic patients die due to IHD or stroke and both are often registered as the cause of death, not DM [2]. A number of evidences exist that demonstrate that the relationship of type 2 DM and associated cardiovascular risk promote the progressive nature of the vascular damage, leading to atherosclerosis [49]. Cardiovascular deaths account for 44% of death in those with type 1 DM and 52% of deaths in type 2 DM [50]. Debate still remains if the relationship between DM and CVD is associated with diabetes status itself or the risk factors diabetic patients are prone to.

2.5 Obesity and overweight

According to the World Health Organisation data, 39% of the global population above 18 years of age is overweight and of these, 13% are obese. Obesity is an abnormal or excessive fat accumulation in adipose tissue to the degree that health might be compromised [51]. Obesity increases the risk for CVDs since it increases the load of the atherosclerotic plaques, characterised by significant infiltration of macrophage and plaque fluctuation [52]. People who accumulate abnormal body fat, particularly at the waist, are at higher risk to have stroke and heart disease even if they do not have other risk factors. The incidence of overweight and obesity among adults in low-income countries varies from 4.7–21.0% and from 13.0–42.7% in upper-middle-income countries [53]. Different studies have demonstrated association between obesity and CVDs such as heart failure and cardiac death. Accumulation of abnormal body fat leads to various metabolic changes that increase the common risk factors of CVDs and affects systems modulating inflammation [54]. Recent studies have demonstrated that weight loss in individuals who are overweight and obese reduces the incidence of diabetes and CVDs. The benefit of weight reduction in overweight and obese individuals with or without hypertension is low blood pressure and serum triglycerides, and increased high-density lipoprotein [55, 56].

3. Nutraceuticals and functional foods

Nutraceutical foods, a borderline between food and drugs, are conventional or processed foods that have or added a useful food component which has a health beneficial effect [57, 58]. Nutraceuticals foods provide medicinal and health benefits and these include prevention, management and/or treatment of a disease. Examples of nutraceutical foods are isolated nutrients, dietary supplements, functional foods, medicinal products and processed foods such as cereals, beverages and soups [59, 60]. The interest of nutraceuticals in prevention of CVDs was invigorated after the examinations of a proximate relationship between their consumption, as shown by higher levels of plasma and low CVDs incidence [61, 62]. Japan is the first country that introduced functional foods in the 1980s and is the only country that has distinct regulatory system that approves functional foods [58]. Functional foods contain dietary fibres, polyphenolic compounds, herbs and botanicals and oligosaccharides with their correlating health benefits [63]. It is believed that functional foods use their cardio-protective effects mostly via antioxidant actions which lower blood lipid levels.

The following factors differentiate functional foods from dietary supplements: (1) Functional foods are expected not to only supplement the diet but should also play a role to prevent and/or treat disease(s) and (2) Functional foods are utilised as traditional foods or as exclusive items of a meal or diet [64]. Dietary components play useful roles apart from basic nutrition and this led to the development of nutraceuticals and functional food concept [65]. Functional foods have different mechanism of actions, such as decreasing low density lipoprotein and elevated blood total cholesterols [66].

3.1 Functional foods with health related properties

Different functional foods are beneficial in preventing and treating CVDs (**Table 3**). Dietary fibres of fruit (with pectin) and vegetable, fish oil and oily seeds such as walnut, almond and many others lower the lipid levels in humans and this is attributed to both prevention of fat absorption and termination of synthesis of hepatic cholesterol [81]. A higher consumption of whole grains, bioactive compounds, antioxidants vitamins and folic acid appears to reverse the harmful vascular effects of homocysteine in the heart [82, 83]. A substantial cardiovascular benefit of polyphenolic compounds, vitamins (ascorbic acid, vitamin E), and minerals such as selenium and magnesium in food is thought to be the ability of these components to scavenge free radicals generated during atherogenesis [84, 85].

Functional food	Active component	Mechanism of action	Reference	
Fruit and vegetable	Antioxidant vitamins, dietary fibre, carotenoid, polyphenolic compounds	Lower concentrations of the inflammatory mediator C-reactive protein Decrease low density lipoprotein Reduce markers of oxidative stress	[67, 68]	
Whole grains	Dietary fibre, minerals, B vitamins and polyphenols	Normal blood pressure and plasma lipids Decrease inflammation	[69, 70]	
Legumes and nuts	Mono and polyunsaturated fatty acids, arginine, soluble fibre, polyphenols, folic acid and B vitamins	Lower blood cholesterol Reduce post-prandial vascular reactivity Improved endothelial function Reduce concentrations of blood homocysteine Lower myocardial infarction Positive effect on blood pressure	[71–73]	
Fish	Omega 3 fatty acids	Improve endothelial function Lower blood pressure and heart rate Reduce aggregability of platelet Reduce fatal cardiac arrhythmias Anti-inflammatory	[74]	
Soy protein	Isoflavonoids, dietary fibre, polyunsaturated fatty acids, vitamins and minerals,	Reduce total cholesterol and low density lipoprotein-C levels Prevent the oxidation of low density lipoprotein Lower total serum	[75–77]	
Coffee and tea	Diterpenes (kahweol and cafestol)	Myocardial infarction	[78]	
Chocolate	Flavonoids	Improve NO-dependent Vasorelaxation Improve flow-mediated dilation in the brachial arteries Reduce ambulatory serum low lipoprotein-C levels and blood pressure Decrease blood cholesterol levels Increase high density lipoprotein-C Decrease oxidised low density	[79, 80]	

Table 3.Different functional foods beneficial in preventing and treating CVDs.

3.1.1 Fruits and vegetables

There is significant amount that low intake of fruit and vegetable is related to higher risk of CVDs while higher intake is related to low risk of CVDs [86, 87]. Fruits and vegetables are rich sources of polyphenolic compounds such as phenolic acid and flavonoids. Different studies associate the intake of polyphenols foods such as fruit and vegetable with low risk of CVDs [88, 89]. In addition, various studies have shown that the properties of flavonoids such as antioxidants and anti-inflammatory might also improve functions of vascular system [90, 91]. Fruits and vegetables reduce

the risk of CVDs by decreasing vulnerability of low density lipoprotein particles to oxidation [92]. Different types of bioactive compounds found in fruits and vegetables such as dietary fibre, carotenoids, ascorbic acid and minerals such as magnesium and potassium act collaboratively to nurture a comprehensive beneficial effect.

3.1.2 Whole grains, legumes and nuts

Whole grains are more important in terms of nutrition since they have phytochemicals that could work synergistically to decrease the risk of CVDs [93]. Moreover, whole grains are also a rich source of dietary fibre, vitamin B complex and minerals. The preventative effect of whole grains on the risk of CVDs is attributed to their influence on insulin vulnerability, blood pressure and inflammation which is associated with the excessive consumption of antioxidant nutrients available in the germ of whole grains [83, 94, 95]. Legumes are rich source of protein, soluble fibre, micronutrients such as folate and polyphenols [96]. Different bioactive components such as protein, dietary fibre and phytosterols are attributed to the cholesterol-lowering effect of legumes [97]. Nuts are rich sources of mono and polyunsaturated fatty acids, arginine, soluble fibre and various antioxidant polyphenols and these active components contribute to cholesterol lowering effects [71].

3.1.3 Fish

High consumption of fish and fish oil supplements contribute to low incidence of CVDs [98]. Fish is a rich source of omega 3 fatty acids such as docosahexaenoic acid (DHA; 22:6 n-3) and eicosapentaenoic acid (EPA; 20:5 n-3). They are available in oily fish such as salmon, tuna, herring and fish oil. Consumption of fish is associated with low risk for myocardial infarction, which is associated with useful influence of DHA and EPA on plaque fluctuation and modulation of endothelial function [99]. Fish oil supplements have beneficial influence on blood pressure and lipid profile [100, 101]. Moreover, DHA and EPA reduce low density lipoprotein oxidative sensitivity in postmenopause women which might assist in reducing the risk of CVDs [102].

3.1.4 Soy protein

Soy products are a rich source of polyunsaturated fatty acids, dietary fibre, micronutrients, low saturated fat content and isoflavones [103]. In addition, the protein content of soybean ranges from 35%–40% having all essential amino acids making soy protein comparable to protein of animal source and it is also cholesterol free [104]. Isoflavones prevent the oxidation of low density lipoprotein and reduces the risk of atherosclerosis [76]. Studies done in China among women and Japan demonstrated that a daily consumption of more than 6 g of soy reduces low density lipoprotein-C, total cholesterol, ischaemic and cerebrovascular incidence than consumption of less than 0.5 g [105, 106].

3.1.5 Coffee, tea and chocolate

Coffee and tea, the most popularly consumed beverages after water, are the chief source of caffeine. Diterpenes such as kahweol and cafestol are suspected to be behind the cardio-protective effect of coffee. Consumption of coffee might potentially decrease the incidence of myocardial infarction, but evidence in this regard is not conclusive [107, 108]. Although results are not consistent, consumption of green tea seems to protect against CVDs [109]. High intake of tea and flavonoids

contribute to the primary prevention of IHD and reduced risk of CVDs mortality [109, 110]. Cocoa is the main ingredient in chocolate manufacturing; it is a rich source of flavonoid and it has been lately evaluated for its plausible role in preventing CVDs [111]. The protective effect of chocolate is attributed to the decrease of blood cholesterols levels, substantial increase of high density lipoprotein including marked decrease of oxidised low density lipoprotein [112].

3.1.6 Animal based functional food

Meat and dairy products are major source of fat in the diet, particularly saturated fatty acids (SFA) which is the leading cause of total cholesterol and CVDs. Meat contains a lot of fat with more than 40% in saturated form, therefore, its quantity and quality has been changed to create new meat products of functional properties. Three meat reformulation methods were proposed in order to develop the functional meat products, namely, low total fat, low total cholesterol intake and modification of fatty acid profile [113]. Dairy products are related to numerous negative health effects because of earlier observations associated SFA content, which might result into increased low-density lipoprotein levels, which in turn increases the risk of CVDs [114]. Dairy products have high SFA and their consumption has long been implicated in contributing to the development of CVDs [115]. Therefore, the consumption of low-fat or non-fat dairy products has been recommended to reduce the risk of CVDs development. Nevertheless, studies tend to show that intake of whole-fat dairy has a favourable effect on health of cardiovascular system and might be more beneficial than intake of low-fat dairy, especially in connection with inflammatory markers. Recent several meta-analyses have demonstrated that low-fat dairy products and whole milk are associated with lower risk of hypertension [116–118]. The presence of calcium, vitamin D as well as other bioactive components such as peptides in dairy products is related to lower blood pressure irrespective of the fat content [119, 120]. High intake of fermented dairy products is associated with low density lipoprotein, low risk of hypertension and CVDs [121, 122]. Consumption of cheese is associated with a low risk of stroke and CVDs [123]. Moreover, consumption of yoghurt is also related to lower risk of CVDs [124]. The presence of bioactive lipids and peptides that have anti-inflammatory characteristics might have contributed to these effects as well as calcium in cheese which might reduce the intake of SFA, thereby decreasing the risk of high cholesterols levels [114]. Moulded cheeses such as Camembert and Roquefort have cardioprotective effects because of the presence of bioactive molecules such as andrastins A–D and roquefortine [125]. Additionally, the cardioprotective effects of fermented dairy products may also be induced by the intake of bacterial metabolites and probiotics. Probiotics reach the gastrointestinal tract while still alive and they can apply their effects directly. Intake of probiotic by supplementation or consumption of fermented dairy products is related to possible health benefits of cardiovascular which include positive effects on blood pressure and hyperlipidaemia [126].

3.2 Dietary pattern and cardiovascular diseases

Various studies have associated the dietary components such as dietary fibre, saturated and trans fats at nutrient level; fruits, vegetables and high fat processed meat at food level [127–129] with changes in prevalence of CVDs. Some diet plans have shown the potential of reducing the incidence of CVDs (**Table 4**). In recent years, various dietary patterns have been recommended for modification in numerous health outcomes apart from the normal dietary guidelines. For example, the Mediterranean, Dietary Approaches to Stop Hypertension (DASH), *Okinawan*

Dietary pattern	High intake	Moderate intake	Low or no intake	Protective effect	References
Mediterranean	Fruits, vegetables, cereals, beans, nuts, seeds, and olive oil	Wine, poultry and fish	Red meat and dairy products	Decrease inflammatory markers	[130–132]
DASH	Fish, fruit, vegetables, whole grains, and nuts	N/A	Dairy products, red meat, sweets, and sugar- containing beverages.	Low blood pressure, anti- inflammatory	[133–135]
Okinawan	Sweet potatoes, and green leafy vegetables	Fish and alcohol	Meat and dairy products	Reduce oxidative stress	[81, 136]
Vegetarian	Fruits, vegetables, legumes and nuts,	N/A	Fish, meat, eggs and dairy products	Anti- inflammatory Low blood pressure and blood cholesterol	[81]

Table 4.

Protective effect of different dietary patterns on cardiovascular diseases.

and vegetarian diets are being promoted as healthy option. Mediterranean diet has gained traction and various reports have shown low incidence of CVDs among populations consuming this diet [137]. Plant foods and olive oil contain high antioxidant content which contributes to the health of the vascular system. The DASH diet restricts saturated fat, red meat, sweets, and beverages with sugar. Regular physical exercise, high intake of dietary potassium, moderate alcohol consumption and low salt intake represent corroborative-based approaches to decrease blood pressure by DASH diet [81]. Okinawan diet restricts calorie, there is high intake of vegetables and legumes, moderate consumption of sea foods and alcohol as well as low consumption of meat and dairy products [138]. This is useful for the health of cardiovascular system because Okinawan diet is nutrient-dense, antioxidant-rich and has a low-glycaemic-load [139]. Vegetarian diet excludes meat, poultry, or fish and may or may not include dairy and eggs. Vegetarian dietary pattern is recognised for its health promoting compounds because it is rich in dietary fibre, antioxidants, bioactive compounds, plant protein and lower saturated than non-vegetarian dietary patterns [140].

4. Conclusion

Overall, this book chapter highlighted the risk factors of CVDs, functional food with health properties and the influence of dietary in the treatment and prevention of CVDs. Generally there is an increasing trend of using functional food in treating and preventing CVDs. Therefore, there is no doubt that functional food can exert a significant effect on maintenance of human health. Continual consumption of different types of functional foods such as fruit and vegetable, whole grains, nuts, legumes, tea, coffee, chocolate, fish, functional meat and fermented dairy products

may help consumers and patients to lower the risk of CVDs. Consumption of full-fat dairy products contributes to higher intakes of important nutrients such as vitamin D and vitamin K. However, fermented dairy products are preferential for ideal intake of nutrients and possible CVDs health benefits. Dietary patterns such as Mediterranean, DASH, *Okinawan* and vegetarian should be promoted since they are associated with low risk of CVDs.

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References

[1] Global, regional, and national agesex-specific mortality for 282 causes of death in 195 countries and territories, 1980-2017. (2018). A systematic analysis for the global burden of disease study 2017. *Lancet*, 392(10159): 1736-1788.

[2] Ajay, V.S., Watkins, D.A. and Prabhakaran, D. (2017). Relationships among major risk factors and the burden of cardiovascular diseases, diabetes, and chronic lung disease PP 23-35. Cardiovascular, Respiratory, and Related Disorders. Disease Control Priorities, 3rd edition. pp. 23-35.

[3] World Health Organisation. (2014). "Global health estimates. WHO, Geneva. Switzerland.

[4] Yuyun, M.F., Sliwa, K., Kengne, P.A., Mocumbi, A.O. and Bukhman, G. (2020). Cardiovascular diseases in sub-Saharan Africa compared to highincome countries: An epidemiological perspective. *Global Heart*, 15(1): 15.

[5] Alwan, A., MacLean, D. R., Riley, L. M., d'Espaignet, E. T., Mathers, C. D., Stevens, G. A., and Bettcher, D. (2010). Monitoring and surveillance of chronic non-communicable diseases: progress and capacity in high-burden countries. *Lancet*, 376(9755): 1861-1868.

[6] Hui, X., Matsushita, K., Sang, Y., Ballew, S.H. and Fülöp, T. (2013). CKD and Cardiovascular Disease in the Atherosclerosis Risk in Communities (ARIC) Study: Interactions with age, sex, and race. *American Journal of Kidney Diseases*, 62(4): 691-702.

[7] Jousilahti, P., Vartiainen, E., Tuomilehto, J. and Puska, P. (1999). Sex, age, cardiovascular risk factors, and coronary heart disease: A prospective follow-up study of 14,786 middle-aged men and women in Finland. *Circulation*, 99(9): 1165-72. [8] Mustafa, K., Ajmal, I., Naz, T.,
Fazili, A.B.A., Bai, X. and Song.
Y. (2020). Bioactive functional foods for cardiovascular diseases. *American Journal of Biochemistry and Biotechnology*, 16 (3): 354.369.

[9] Abubakar, I. I., Tillmann, T. and Banerjee, A. (2015). Global, regional and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the global burden of disease study 2013. *Lancet*, 385(9963): 117-171.

[10] Lozano, R., Naghavi, M., Foreman, K., Lim, S., Shibuya, K., Aboyans, V. and AlMazroa, M. A. (2012). Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the global burden of disease Study 2010. *Lancet*, 380 (9859): 2095-2128.

[11] Naghavi, M., Abajobir, A. A., Abbafati, C., Abbas, K. M., Abd-Allah, F., Abera, S. F. and Ahmadi, A. (2017). Global, regional and national age-sex specific mortality for 264 causes of death, 1980-2016: a systematic analysis for the global burden of disease study 2016. *Lancet*, 390(10100): 1151-1210.

[12] Berrington de Gonzalez, A., Hartge, P., Cerhan, J.R., Flint, A.J., Hannan, L., MacInnis, R. J. and Beeson, W.L. (2010). Body-mass index and mortality among 1.46 million white adults. *New England Journal of Medicine*, 363(23): 2211-2219.

[13] Bitok, E. and Sabaté, J. (2018). Nuts and cardiovascular disease. *Progress in Cardiovascular Diseases*, 61(1): 33-37.

[14] Meier, T., Gräfe, K., Senn, F., Sur, P., Stangl, G.I., Dawczynski, C. and Lorkowski, S. (2019). Cardiovascular mortality attributable to dietary risk factors in 51 countries in the WHO European region from 1990 to 2016: a

systematic analysis of the global burden of disease study. *European Journal of Epidemiology*, 34(1): 37-55.

[15] McGill, H.C. (1979). "The relationship of dietary cholesterol to serum cholesterol concentration and to atherosclerosis in man. *American Journal of Clinical Nutrition*, 32(12): 2664-2702.

[16] Reddy, K.S. and Katan M.B. (2004).
Diet, nutrition and the prevention of hypertension and cardiovascular diseases. *Public Health Nutrition*, 7(1): 167-186.

[17] Berry, J.D., Dyer, A., Cai, X., Garside, D.B., Ning, H., Thomas, A. and Lloyd-Jones, D.M. (2012). Lifetime risks of cardiovascular disease. *New England Journal of Medicine*, 366(4): 321-329.

[18] GBD (Global Burden of Disease)
2013 Risk Factors Collaborators.
(2015). Global, regional and national comparative risk assessment of 79
behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries,
1990-2013: A systematic analysis for the global burden of disease study 2013. *Lancet*, 386(10010): 2287-323.

[19] Danaei, G., Finucane, M.M., Lu, Y., Singh, G.M., Cowan, M.J., Paciorek, C.J. and Rao, M. (2011). National, regional and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants. *Lancet*, 378(9785): 31-40.

[20] Emerging Risk Factors Collaboration. (2011). Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. *Lancet*, 377(9771): 1085-1095. [21] Lewington, S., Clarke, R., Qizilbash, N., Peto, R. and Collins, R. (2002). Age-specific relevance of usual blood pressure to vascular mortality: a metaanalysis of individual data for one million adults in 61 prospective studies, *Lancet*, 360: 1903-1913.

[22] Britton, K.A., Gaziano, J.M. and Djousse, L. (2009). Normal systolic blood pressure and risk of heart failure in US male physicians. *European Journal of Heart Failure*, 11: 1129-1134.

[23] Kalaitzidis, R.G. and Bakris, G.L. (2010). Prehypertension: is it relevant for nephrologists. *Kidney International*, 77: 194-200.

[24] World Health Organization. (2012). Guideline: Sodium intake for adults and children; World Health Organization: Geneva, Switzerland.

[25] Weinberger, M.H. (1996). Salt sensitivity of blood pressure in humans. *Hypertension*, 27: 481-490.

[26] Strazzullo, P., D'Elia, L., Kandala, N.B. and Cappuccio, F.P. (2009). Salt intake, stroke, and cardiovascular disease: Meta-analysis of prospective studies. *British Medical Journal*, 339: b4567.

[27] Whelton, P.K. and He, J. (2014). Health effects of sodium and potassium in humans. *Current Opinion in Lipidology*, 25: 75-79.

[28] Austin, M.A. (1991). Plasma triglyceride and coronary heart disease. *Arteriosclerosis and Thrombosis*, 11(1): 2-14.

[29] Basharat, S. (2020). Therapeutic effect of *Glycine max* (Soybean) bioactive components in cardiovascular disease and obesity. *Journal of Food and Nutrition*, 6: 1-7.

[30] Law, M.R., Morris, J.K. and Wald. N.J. (1997). Environmental tobacco smoke exposure and Ischaemic heart disease: An evaluation of the evidence. *British Medical Journal*, 315(7114): 973-80.

[31] Bilano, V., Gilmour, S., Moffiet, T., D'Espaignet, E.T. and Stevens, G.A. (2015). Global trends and projections for tobacco use, 1990-2025: An analysis of smoking indicators from the WHO comprehensive information systems for tobacco control. *Lancet*, 385 (9972): 966-76.

[32] Gritz, E.R., Vidrine, D.J. and Cororve Fingeret, M. (2007). Smoking cessation. A critical component of medical management in chronic disease populations. *American Journal of Preventive Medicine*, 33(6): S414-S422.

[33] WHO. (2008). WHO report on the global tobacco epidemic. The MPOWER package. Geneva: World Health Organization, Geneva, Switzerland.

[34] IOM. (2010). Promoting cardiovascular health in the developing world: A critical challenge to achieve global health. Washington, DC: The National Academies Press.

[35] Parish, S., Collins, R., Peto, R., Youngman, L. and Barton, J. (1995). Cigarette-smoking, tar yields, and nonfatal Myocardial-Infarction: 14,000 Cases and 32,000 controls in the United Kingdom. *British Medical Journal*, 311(7003): 471-77.

[36] World Health Organisation. (2016). WHO: Physical Activity. Fact sheet 385, WHO, Geneva. Switzerland.

[37] Cheng, S.J., Yu, H.K., Chen, Y.C., Chen, C.Y., Lien, W.C., Yang, P.Y. and Hu, G.C. (2013). Physical activity and risk of cardiovascular disease among older adults. *International Journal of Gerontology*, 7: 133e136.

[38] Folsom, A.R., Jacobs, D.R. Junior and Wagenknecht, L.E. (1996). Increase in fasting insulin and glucose over seven years with increasing weight and inactivity of young adults. The CARDIA study. Coronary artery risk development in young adults. *American Journal of Epidemiology*, 144: 235-246.

[39] Lee, I.M. and Paffenbarger Jr, R.S. (1998). Physical activity and stroke incidence: the Harvard alumni health study. *Stroke*, 29: 2049e2054.

[40] Evenson, K.R., Rosamond, W.D. and Cai, J. (1999). Physical activity and ischemic stroke risk. The atherosclerosis risk in communities study. *Stroke*, 30: 1333e1339.

[41] Redberg, R.F., Greenland, P., Fuster, V., Pyörälä, K., Blair, S.N. and Folsom, A.R. (2002). Prevention Conference VI: Diabetes and cardiovascular disease: writing group III: risk assessment in persons with diabetes. *Circulation*, 105: e144-52.

[42] Mayer-Davis, E.J., Lawrence, J.M., Dabelea, D., Divers, J., Isom, S. and Dolan L. (2017). Incidence trends of type 1 and type 2 diabetes among youths, 2002-2012. *New England Journal of Medicine*, 376: 1419-29.

[43] Dokken, B. (2008). The pathophysiology of cardiovascular disease and diabetes: beyond blood pressure and lipids. *Diabetes Spectrum*, 21: 160-5.

[44] Centers for Disease Control and Prevention. (2014). National diabetes statistics report: estimates of diabetes and its burden in the United States, Atlanta: US Department of Health and Human Services.

[45] Rivellese, A.A., Riccardi, G. and Vaccaro, O. (2010). Cardiovascular risk in women with diabetes. *Nutrition, Metabolism and Cardiovascular Disease*, 20: 474-480.

[46] Wild, S., Roglic, G., Green, A., Sicree, R., and King, H. (2004). Global

prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*, 27: 1047-1053.

[47] Aguiree, F., Brown, A., Cho, N.H., Dahlquist, G., Dodd, S., Dunning, T., Hirst, M., Hwang C., Magliano, D. and Patterson C. (2013). IDF Diabetes Atlas.

[48] IDF (International Diabetes Federation). (2015). IDF Diabetes Atlas 2015. 7th ed. Brussels: IDF.

[49] Grundy, S.M. (2012). Pre-diabetes, metabolic syndrome and cardiovascular risk. *Journal of the American College of Cardiology*, 59: 7.

[50] Morrish, N.J., Wang, S.L., Stevens,
L.K., Fuller, J.H. and Keen, H. (2001).
WHO multinational study group.
Mortality and causes of death in the
WHO multinational study of vascular disease in diabetes. *Diabetologia*,
44: S14-21.

[51] World Health Organisation (2000). Obesity: Preventing and Managing the Global Epidemic; Report of a WHO Consultation. Technical Report Series 894. Geneva: WHO, Switzerland.

[52] De Rosa, R., Vasa-Nicotera, M. and Leistner, D.M. (2017). Coronary atherosclerotic plaque characteristics and cardiovascular risk factors - insights from an optical coherence tomography study. *Circulation Journal*, (8): 1165-1173.

[53] Lovren, F., Teoh, H. and Verma, S. (2015). Obesity and atherosclerosis: mechanistic insights. *Canadian Journal of Cardiology*, 31(2): 177-183.

[54] Mathieu, P., Poirier, P., Pibarot, P., Lemieux, I. and Despres, J.P. (2009). Visceral obesity: the link among inflammation, hypertension, and cardiovascular disease. *Hypertension*, 53(4): 577-584.

[55] Appel, L.J., Brands, M.W., Daniels, S.R., Karanja, N. and Elmer, P.J. (2006).

Dietary approaches to prevent and treat hypertension: A scientific statement from the American Heart Association. *Hypertension*, 47(2): 296-308.

[56] Miller, M., Stone, N.J. Ballantyne,
C., Bittner, V. and Criqui, M.H. (2011).
Triglycerides and cardiovascular
disease: A scientific statement from
the American Heart Association. *Circulation*, 123(20): 2292-333.

[57] Schieber, A., Stintzing, F.C. and Carle, R. (2001). By-products of plant food processing as a source of functional compounds – recent developments. *Trends in Food Science and Technology*, 12: 401-13.

[58] Kaur, S. and Das, M. (2011).Functional foods: an overview.Food Science and Biotechnology, 20(4): 861-75.

[59] Rimm, E.B., Stampfer, M.J. and Ascherio, A. (1993). Vitamin E consumption and the risk of coronary heart disease in men. *New England Journal of Medicine*, 328:1450-1456.

[60] Stampfer, M.J., Hennekens, C.H. and Manson, J.E. (1993). Vitamin E consumption and the risk of coronary disease in women. *New England Journal of Medicine*, 328: 1444-1449.

[61] Andlauer, W. and Fürst, P. (2002). Nutraceuticals: A piece of history, present status and outlook. *Food Research International*, 35: 171-176.

[62] Kwak, N.S. and Jukes, D.J.(2001). Functional foods. Part 1: The development of a regulatory concept. *Food Control*, 12: 99-107.

[63] Arshad F. (2003). Functional foods from the dietetic perspective in Malaysia. *Nutrition and Diet*, 60(2): 119-21.

[64] Kalra, E.K. (2003). Nutraceutical– definition and introduction. *AAPS Pharmaceutical Science*, 5: 25. [65] Laparra, J.M. and Sanz, Y. (2010). Interactions of gut microbiota with functional food components and nutraceuticals. *Pharmacology Research*, 61: 219-225.

[66] Boussageon, R., Bejan-Angoulvant, T., Saadatian-Elahi, M., Lafont, S., Bergeonneau, C., Kassaï, B. and Cornu, C. (2011). Effect of intensive glucose lowering treatment on all cause mortality, cardiovascular death and microvascular events in type 2 diabetes: meta-analysis of randomised controlled trials. *British Medical Journal*, 26, 343: d4169.

[67] Korantzopoulos, P., Kolettis, T,M., Kountouris, E., Dimitroula, V., Karanikis, P., Pappa, E., Siogas, K. and Goudevenos, J.A. (2005). Oral vitamin C administration reduces early recurrence rates after electrical cardioversion of persistent atrial fibrillation and attenuates associated inflammation. *International Journal of Cardiology*, 102: 321-326.

[68] Aguirre, R. and May, J.M. (2008). Inflammation in the vascular bed: importance of vitamin C. *Pharmacology and Therapeutics*, 119: 96-103.

[69] Mellen, P.B., Walsh, T.F. and Herrington, D.M. (2008). Whole grain intake and cardiovascular disease: a meta-analysis. *Nutrition, Metabolism and Cardiovascular Diseases*, 18(4): 283-90.

[70] Lutsey, P.L., Jacobs, D.R., Kori, S., Mayer-Davis, E., Shea, S., Steffen, L.M., Szklo, M. and Tracy, R. (2007). Whole grain intake and its cross-sectional association with obesity, insulin resistance, inflammation, diabetes and subclinical CVD: the MESA Study. *British Journal of Nutrition*, 98(02): 397-405.

[71] Sabat'e, J. and Ang, Y. (2009). Nuts and health outcomes: new epidemiologic evidence. *American* *Journal of Clinical Nutrition*, 89(5): 1643S–1648S.

[72] Sabate, J., Oda, K. and Ros, E. (2010). Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. *Archives of internal medicine*, 170(9): 821-7.

[73] Casas-Agustench, P., Lopez-Uriarte, P., Ros, E., Bullo, M. and Salas-Salvado J. (2011). Nuts, hypertension and endothelial function. *Nutrition, Metabolism and Cardiovascular Diseases*, 21: S21–S33.

[74] Mozaffarian, D. (2008). Fish and n-3 fatty acids for the prevention of fatal coronary heart disease and sudden cardiac death. *American Journal of Clinical Nutrition*, 87(6): 1991S–1996S.

[75] Harland, J.I. and Haffner, T.A. (2008). Systematic review, metaanalysis and regression of randomised controlled trials reporting an association between an intake of circa 25 g soya protein per day and blood cholesterol. *Atherosclerosis*, 200(1): 13-27.

[76] Wiseman, H. (1999). The bioavailability of non-nutrient plant factors: dietary flavonoids and phytooestrogens. *Proceedings of the Nutrition Society*, 58(1): 139-146.

[77] Taku, K., Umegaki, K., Sato, Y. Taki, Y., Endoh, K. and Watanabe, S. (2007). Soy isoflavones lower serum total and LDL cholesterol in humans: a metaanalysis of 11 randomized controlled trials. *American Journal of Clinical Nutrition*, 85(4): 1148-1156.

[78] Christensen, B., Mosdol, A., Retterstol, L., Landaas, S., and Thelle, D.S. (2001). Abstention from filtered coffee reduces the concentrations of plasma homocysteine and serum cholesterol—a randomized controlled trial. *American Journal of Clinical Nutrition*, 74(3): 302-307.

[79] Grassi, D., Necozione, S. and Lippi, C. (2005). Cocoa reduces blood pressure and insulin resistance and improves endothelium dependent vasodilation in hypertensives. *Hypertension*, 46(2): 398-405.

[80] Baba, S., Natsume, M., Yasuda, A., Nakamura, Y., Tamura, T., Osakabe, N., Kanegae, M. and Kondo K. (2007). Plasma LDL and HDL cholesterol and oxidized LDL concentrations are altered in normo-and hypercholesterolemic humans after intake of different levels of cocoa powder. *Journal of Nutrition*, 137(6): 1436-41.

[81] Alissa, E.M. and Ferns, G.A.
(2012). Functional Foods and nutraceuticals in the primary prevention of cardiovascular diseases. *Journal of Nutrition and Metabolism*. Volume 2012, Article ID 569486, 16 pages. .

[82] Broekmans, W.M.R., Kl^{*}opping-Ketelaars, I.A.A. and Schuurman, C.R.W.C. (2000). Fruits and vegetables increase plasma carotenoids and vitamins and decrease homocysteine in humans. *Journal of Nutrition*, 130(6): 1578-1583.

[83] McKeown, N.M., Meigs, J.B., Liu, S., Wilson, P.W.F. and P.F. Jacques, P.F. (2002). Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham offspring study. *American Journal of Clinical Nutrition*, 76(2): 390-398.

[84] Block, G., Mangels, A.R., Norkus, E.P., Patterson, B.H., Levander, O.A. and Taylor, P.R. (2001). Ascorbic acid status and subsequent diastolic and systolic blood pressure," *Hypertension*, 37(2): 261-267, 2001.

[85] Ferrari, C.K.B. Functional foods, herbs and nutraceuticals: towards biochemical mechanisms of healthy aging. *Biogerontology*, 5(5), 275-289. [86] Yamada, T., Hayasaka, S., Shibata, Y., Ojima, T., Saegusa, T., Gotoh, T., Ishikawa, S., Nakamura, Y., Kayaba, K. (2011). Frequency of citrus fruit intake is associated with the incidence of cardiovascular disease: the Jichi Medical School cohort *study. Journal of Epidemiology*, 21(3):169-75.

[87] Jacques, P.F., Lyass, A., Massaro, J.M., Vasan, R.S. and D'Agostino, R.B. Sr. (2013). Relationship of lycopene intake and consumption of tomato products to incident CVD. *British Journal of Nutrition*, 110(3): 545-51.

[88] Miyagi, Y., Miwa, K. and Inoue, H. (1997). Inhibition of human low density lipoprotein oxidation by flavonoids in red wine and grape juice. *American Journal of Cardiology*, 80: 1627-1631.

[89] Rein, D., Paglieroni, T.G., Pearson, D.A., Wun, T. and Schmitz, H.H. (2000). Cocoa and wine polyphenols modulate platelet activation and function. *Journal of Nutrittion*, 130: 2120S–2126S.

[90] Duffy, S.J., Keaney, J.F. Jr, Holbrook, M., Gokce, N. and Swerdloff, P.L. (2001). Short-and long-term black tea consumption reverses endothelial dysfunction in patients with coronary artery disease. *Circulation*, 104: 151-156.

[91] Zhao, X., Gu, Z., Attele, A.S. and Yuan, C.S. (1999). Effects of quercetin on the release of endothelin, prostacyclin and tissue plasminogen activator from human endothelial cells in culture. *Journal of Ethnopharmacology*, 67: 279-285.

[92] Chopra, M., O'Neill, M.E., Keogh, N., Wortley, G., Southon, S. and Thurnham, D.I. (2000). Influence of increased fruit and vegetable intake on plasma and lipoprotein carotenoids and LDL oxidation in smokers and nonsmokers. *Clinical Chemistry*, 46(11): 1818-1829. [93] Morris, J., Marr, J.W. and Clayton, D. (1977). Diet and heart: a postscript. *British Medical Journal*, 2(6098): 1307-14.

[94] Flint, A.J., Hu, F.B. and Glynn, R.J. (2009). Whole grains and incident hypertension in men. *American Journal* of *Clinical Nutrition*, 90(3): 493-498.

[95] Lutsey, P.L., Jacobs, D.R. and Kori, S. (2007). Whole grain intake and its cross-sectional association with obesity, insulin resistance, inflammation, diabetes and subclinical CVD: the MESA Study. *British Journal of Nutrition*, 98(2): 397-405.

[96] Mozaffarian, D., Appel, L.J. and Van Horn, L. (2011). Components of a cardio-protective diet new insights. *Circulation*, 123(24): 2870-91.

[97] Martins, J.M., Riottot, M., de Abreu, M.C., Viegas-Crespo, A.M., Lan, ca, M.J., Almeida, J.A., Freire, J.B. and Bento, O.P. (2005). Cholesterollowering effects of dietary blue lupin (*Lupinus angustifolius* L.) in intact and ileorectal anastomosed pigs. *Journal of Lipid Research*, 46(7): 1539-47.

[98] He, K., Song, Y., Daviglus, M.L., Liu, K., Van Horn, L., Dyer, A.R. and Greenland, P. (2004). Accumulated evidence on fish consumption and coronary heart disease mortality a metaanalysis of cohort studies. *Circulation*, 109(22): 2705-11.

[99] Ueeda, M., Doumei, T., Takaya, Y., Shinohata, R., Katayama, Y., Ohnishi, N., Takaishi, A., Miyoshi, T., Hirohata, S. and Kusachi, S. (2008). Serum N-3 polyunsaturated fatty acid levels correlate with the extent of coronary plaques and calcifications in patients with acute myocardial infarction. *Circulation Journal*, 72(11): 1836-43.

[100] Durrington, P., Bhatnagar, D., Mackness, M., Morgan, J., Julier, K., Khan, M. and France M. (2001). An omega-3 polyunsaturated fatty acid concentrate administered for one year decreased triglycerides in simvastatin treated patients with coronary heart disease and persisting hypertriglyceridaemia. *Heart*, 85(5): 544-8.

[101] Dyerberg, J., Eskesen, D.C., Andersen, P.W., Astrup, A., Buemann, B. and Christensen, JH. (2004). Effects of trans-and n-3 unsaturated fatty acids on cardiovascular risk markers in healthy males. An 8 weeks dietary intervention study. *European Journal of Clinical Nutrition*, 58(7): 1062-70.

[102] Lee, Y-S. and Wander, R.C. (2005). Reduced effect on apoptosis of 4-hydroxyhexenal and oxidized LDL enriched with n-3 fatty acids from postmenopausal women. *The Journal of Nutritional Biochemistry*, 16 (4): 213-21.

[103] Ho, S.C., Chen, Y.M. Ho, S.S.S. and Woo, J.L.F. (2007). Soy isoflavone supplementation and fasting serum glucose and lipid profile among postmenopausal Chinese women: a double-blind, randomized, placebocontrolled trial. *Menopause*, 14(5): 905-912.

[104] Young V. (1991). Soy protein in relation to human protein and amino acid nutrition. *Journal of the American Dietetic Association*, 91(7): 828-35.

[105] Zhang, X., Shu, X.O. and Gao, Y.T. (2003). Soy food consumption is associated with lower risk of coronary heart disease in Chinese women. *Journal of Nutrition*, 133(9): 2874-2878.

[106] Nagata, C., Takatsuka, N., Kurisu, Y and Shimizu, H. (1998). Decreased serum total cholesterol concentration is associated with high intake of soy products in Japanese men and women. *Journal of Nutrition*, 128 (2): 209-213.

[107] Christensen, B., Mosdol, A., Retterstol, L., Landaas, S. and D.S.

Thelle, D.S. (2001). "Abstention from filtered coffee reduces the concentrations of plasma homocysteine and serum cholesterol— a randomized controlled trial. *American Journal of Clinical Nutrition*, 74(3): 302-307.

[108] Panagiotakos, D.B., Pitsavos,
C., Chrysohoou, C., Kokkinos, P.,
Toutouzas, P. and Stefanadis, C.
(2003). The J-shaped effect of coffee
consumption on the risk of developing
acute coronary syndromes: the
CARDIO2000 case-control study.
Journal of Nutrition, 133(10): 3228-3232.

[109] Sumpio, B.E., Cordova, A.C., Berke-Schlessel, D.W., Qin, F. and Chen, Q.H. (2006). Green tea, the "Asian paradox," and cardiovascular disease. *Journal of the American College of Surgeons*, 202(5): 813-25.

[110] Geleijnse, J.M., Launer, L.J., van der Kuip, D.A., Hofman, A. and Witteman, J. (2002) Inverse association of tea and flavonoid intakes with incident myocardial infarction: The Rotterdam study. *American Journal of Clinical Nutrition*, 75: 880-886.

[111] Huxley, R.R., Neil, H.A.W. (2003). The relation between dietary flavonol intake and coronary heart disease mortality: A meta-analysis of prospective cohort studies. *European Journal of Clinical Nutrition*, 57: 904.

[112] Galleano, M., Oteiza, P.I. and Fraga, C.G. (2009). Cocoa, chocolate, and cardiovascular disease. *Journal of Cardiovascular Pharmacology*, 54(6): 483-490.

[113] Pogorzelska-Nowicka, E., Atanasov, A.G., Horbanczuk, J. and Wierzbicka, A. (2018). Bioactive compounds in functional meat products. *Molecules*, 23: 307.

[114] Artaud-Wild, S.M., Connor, S.,Sexton, G. and Connor, W.E. (1993).Differences in coronary mortality can be

explained by differences in cholesterol and saturated fat intakes in 40 countries but not in France and Finland. A paradox. *Circulation*, 88: 2771-2779.

[115] Lordan, R. and Zabetakis, I. (2017). Invited review: The anti-inflammatory properties of dairy lipids. *Journal of Dairy Science*, 100: 4197-4212.

[116] Drouin-Chartier, J.P., Brassard, D., Tessier-Grenier, M., Côté, J.A., Labonté, M.È., Desroches, S., Couture, P. and Lamarche, B. (2016). Systematic review of the association between dairy product consumption and risk of cardiovascular-related clinical outcomes. *Advanced Nutrition*, 7: 1026-1040.

[117] Wang, H., Fox, C.S., Troy, L.M., Mckeown, N.M. and Jacques, P.F. (2015). Longitudinal association of dairy consumption with the changes in blood pressure and the risk of incident hypertension: The framingham heart study. British Journal of Nutrition, 114: 1887-1899.

[118] Ralston, R.A., Lee, J.H., Truby, H., Palermo, C.E. and Walker, K.Z. (2012). A systematic review and metaanalysis of elevated blood pressure and consumption of dairy foods. *Journal of Human Hypertension*, 26: 3-13.

[119] Da Silva, M.S. and Rudkowska, I. (2014). Dairy products on metabolic health: Current research and clinical implications. *Maturitas*, 77: 221-228.

[120] Alonso, A., Zozaya, C., Vázquez, Z., Alfredo Martínez, J. and Martínez-González, M.A. (2009). The effect of low-fat versus whole-fat dairy product intake on blood pressure and weight in young normotensive adults. *Journal of Human Nutrition and Dietetics*, 22: 336-342.

[121] Beltrán-Barrientos, L.M., Hernández-Mendoza, A., Torres-Llanez, M.J., González-Córdova, A.F. and Vallejo-Córdoba, B. (2016). Invited review: Fermented milk as antihypertensive functional food. *Journal of Dairy Science*, 99: 4099-4110.

[122] Qin, L.-Q., Xu, J.Y., Han, S.F., Zhang, Z.-L., Zhao, Y.Y. and Szeto, I.M. (2015). Dairy consumption and risk of cardiovascular disease: An updated meta-analysis of prospective cohort studies. *Asia Pacific Journal of Clinical Nutr*ition, 24: 90-100.

[123] Praagman, J., Dalmeijer, G.W., van der Schouw, Y.T., Soedamah-Muthu, S.S., Monique Verschuren,W.M., Bas Bueno-de-Mesquita, H., Geleijnse, J.M. and Beulens, J.W.J. (2015). The relationship between fermented food intake and mortality risk in the European prospective investigation into cancer and nutrition-Netherlands cohort. *British Journal of Nutr*ition, 113: 498-506.

[124] Wu, L. and Sun, D. (2017). Consumption of yogurt and the incident risk of cardiovascular disease: A meta-analysis of nine cohort studies. Nutrients, 9: 315.

[125] Petyaev, I.M. and Bashmakov, Y.K. (2012). Could cheese be the missing piece in the French paradox puzzle? *Medical Hypotheses*, 79: 746-749.

[126] Parvez, S., Malik, K.A., Ah Kang, S. and Kim, H.Y. (2006). Probiotics and their fermented food products are beneficial for health. *Journal of Applied Microbiology*, 100, 1171-1185.

[127] Baba, S., Natsume, M. and Yasuda, A. (2007). Plasma LDL and HDL cholesterol and oxidized LDL concentrations are altered in normoand hypercholesterolemic humans after intake of different levels of cocoa powder. *Journal of Nutrition*, 137(6): 1436-1441.

[128] Kim, Y. and Je, Y. (2016). Dietary fibre intake and mortality from

cardiovascular disease and all cancers: a meta-analysis of prospective cohort studies. *Archives of Cardiovascular Disease*, 109: 39-54.

[129] Cui, K., Liu, Y., L. Zhu, L., Mei, P. and Jin, Y.J.B.P.H. (2019). Luo Association between intake of red and processed meat and the risk of heart failure: a meta-analysis. *BMC Public Health*, 19: 354

[130] Satija, A. and Hu, F.B. (2018). Plant-based diets and cardiovascular health. *Trends in Cardiovascular Medicine*, 28: 437-41.

[131] Huang, C.L. and Sumpio, B.E. (2008). Olive oil, the Mediterranean diet, and cardiovascular health. *Journal of the American College of Surgeons*, 207(3): 407-416.

[132] De Lorgeril, M., Salen, P., Martin, J.L., Monjaud, I., Delaye, J. and Mamelle, N. (1999). Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon diet heart study. *Circulation*, 99(6): 779-785.

[133] Esposito, M. Ciotola, L. and Giugliano, D. (2006). Mediterranean diet, endothelial function and vascular inflammatory markers. *Public Health Nutrition*, 9(8): 1073-1076.

[134] Fung, T.T., Chiuve, S.E., McCullough, M.L., Rexrode, K.M., Logroscino, G. and Hu, F.B. (2008). Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Archives of Internal Medicine*, 168(7): 713-720.

[135] Obarzanek, E., Sacks, F.M. and Vollmer, W.M. (2001). Effects on blood lipids of a blood pressure-lowering diet. The Dietary Approaches to Stop Hypertension (DASH) Trial. *American Journal of Clinical Nutrition*, 74(1): 80-89.

[136] Appel, L.J., Miller, E.R. and Jee, S.H. (2000). Effect of dietary patterns on serum homocysteine: results of a randomized, controlled feeding study. *Circulation*, 102(8): 852-857.

[137] Aune, D., Giovannucci, E., Boffetta, P., Fadnes, L.T., Keum, N. and Norat, T. (2017). Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality—a systematic review and dose-response meta-analysis of prospective studies. *International Journal of Epidemiology*, 46: 1029-1056.

[138] Kushi, L.H., Lenart, E.B. and Willett, W.C. (1995). Health implications of Mediterranean diets in light of contemporary knowledge —1. Plant foods and dairy products. *American Journal of Clinical Nutrition*, 61(6): 1407S–1415S.

[139] Willcox, D.C. G. Scapagnini, G. and Willcox, B.J. (2014). Healthy aging diets other than the Mediterranean: a focus on the *Okinawan* diet. *Mechanisims* of Ageing and Development, 136: 148-162.

[140] Willcox, D.C., Willcox, B.J., Todoriki, H. and Suzuki. M. (2009). The *Okinawan* diet: health implications of a low-calorie, nutrient-dense, antioxidant-rich dietary pattern low in glycemic loads. *Journal of American College of Nutrition*, 28: 500s–516s.