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International Journal of Biochemistry & Physiology
ISSN: 2577-4360

Nonpharmacological Agents versus Cardiovascular Diseases

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Editorial
Volume 8 Issue 2
Received Date: June 27, 2023
Published Date: July 21, 2023
DOI: 10.23880/ijbp-16000224

Abbreviations: ALA: Alpha Linolenic Acid; CAD: Coronary Artery Disease; HDL: Lipo Protein Cholesterol; GLP-1: Glucagon Like Peptide-1; CMDs: Cardio Metabolic Diseases.

Editorial

Away from total cholesterol, oxidative stress and inflammation as well as antiplatelet effects are additional approaches for reducing cardiovascular diseases (CVDs) [1-4]. In spite of key advancements in the management of CVDs, these problems continue to escalation and may be a principal cause of mortality and morbidity [2]. Latest studies point out that these risk markers progress as a consequence of increased intake of western-type diets described by ultra-processed foods, processed meat and red meat, which are known to predispose to all the biomarkers of CVDs [5]. A number of studies have validated that Mediterranean-style diets characterized by vegetables, fruits, whole grains, nuts and certain agents such as fish oil, mustard oil, rape seed oil, olive oil and fish as well as fish peptides have advantageous effects on morbidity and mortality consequent to CVDs without declining serum cholesterol [4,6-8]. These advantages may be due to the presence of omega-3 and omega-6 fatty acids, alpha-linolenic acid (ALA) and poly- phenolics and fiber in the diet [9]. This putative communication targets to highlight that morbidity and mortality due to CVDs may be reduced through non-pharmacological interventions without the necessity of decreasing cholesterol under following salient heads:

Risk Factors and Biomarkers Associated with Cardiovascular Disorders

Though serum cholesterol, tobacco intake, diabetes mellitus and hypertension are major risk factors of coronary artery disease (CAD), the exact causes of atherosclerosis are still obscure [1]. Away from each other from these risk factors, there are several other minor risk factors such as obesity, sedentary behavior, depression, sleep disorders, low high-density lipo- protein cholesterol (HDL), and hypertriglyceridemia, which also incline to morbidity and mortality consequent to CVDs [1,2]. Apart from blood cholesterol, proliferation in oxidative stress and inflammation as well as platelet aggregation may be the underlying mechanisms in the pathogenesis of atherothrombosis [1-4]. Consequently, all those agents, which decrease inflammation and thrombosis may also cause a decline in morbidity and mortality due to CVDs [1-8]. Since western-style diets are also known to predispose to obesity, metabolic syndrome and diabetes mellitus, this may be an alternative pathway for development of CVDs [4,5,10]. It is characterized by insulin resistance, hyper-triglyceridemia, and low HDL as well as an increase in inflammation [4-6]. Besides, CVDs may increase due to deficiency of protective factors, such as Mediterranean foods, spices, proteins, fatty acids, physical activity and overall healthiness. Latest studies reveal that certain foods and nutrients can influence the secretion of glucagon- like peptide GLP- 1 receptors, which have been found to be vital in the pathogenesis and prevention of CVDs and metabolic diseases [11].

Nutritional Boosters Versus Glucagon Like Peptide-1 Levels and Cardio Metabolic Diseases

Glucagon-like peptide-1 (GLP-1) is an incretin hormone that possesses a wide range of effects on glucose metabolism and cardiovascular function, improving insulin sensitivity, decrease in appetite, heart rate and blood pressure modulation and myocardial contractility [12]. Fasting plasma level of biologically active GLP-1 ranges between 0 and 15 pmol/L in humans, probably increasing

2- to 3-fold after meals depending on meal size and nutrient composition [12]. GLP-1 hormone is mostly synthesized and secreted by enteroendocrine L-cells of the gastrointestinal tract in response to meals. Diets rich in micronutrients, such as Mediterranean-style diets, Indo-Mediterranean-style diets, the DASH diet and the Japanese diet, including high-fiber whole grain products, nuts, avocados and eggs, seem to affect the release of GLP-1 [12]. These foods might promote linked advantageous outcomes in healthy subjects and in patients with cardio-metabolic diseases (CMDs) [13]. Stimulation of endogenous GLP-1 secretion by manipulating food and nutrients of the diet may be a pertinent strategy for the prevention of morbidity and mortality consequent to CMDs [13].

Other Non-Cholesterol Lowering Agents and their Cardio-Protective Effects

Previous studies, including clinical trials reported that Mediterranean-style diets can cause a noteworthy decline in CVDs and its mortality without a considerable decrease in serum cholesterol [3,14,15]. These results appear to be rational because recent cohort studies found that ultra-processed foods, processed meat, unprocessed red meat, poultry, or fish intake can considerably influence incident cardiovascular disease and all-cause mortality [4,5]. Earlier studies using fish, fish oil and fish peptide also have demonstrated that these agents can cause a significant decline in CVDs and its mortality, although some of the results were controversial [6-10]. The Diet and Re-infarction trial showed that eating modest amounts of oily fish twice weekly can decrease all- cause mortality [6]. The beneficial effects of fish may be via antiplatelet effects, improvement in endothelial function and due to cardio-protective effects of fish peptides [10].

Olive Oil

There is evidence that olive oil intake can cause a considerable decrease in the risk of CVDs, but its associations with total and cause-specific mortality are still obscure. The question has been cleared through outcome of two prospective cohorts; including, 60,584 women from the Nurses' Health Study, (1990-2018) and 31,801 men from the Health Professionals Follow-up Study (1990-2018) who were free of CVDs or cancer at baseline [15]. There were 36,856 deaths during 28 years of follow-up. The multivariable-adjusted pooled HR for all-cause mortality among subjects who had the highest intake of olive oil (>0.5 tablespoon/day or >7 g/d) was 0.81 (95% CI: 0.78-0.84) compared with those who never or infrequently took olive oil. Higher olive oil consumption was linked with a 19% lower risk of mortality due to CVDs (HR: 0.81; 95% CI: 0.75-0.87), 17% lower risk of cancer mortality (HR: 0.83; 95% CI: 0.78-0.89), 29% lower risk of mortality consequent to neurological diseases (HR: 0.71; 95% CI: 0.64-0.78), and 18% lower risk of mortality due to pulmonary diseases (HR: 0.82; 95% CI: 0.72-0.93). Replacing 10 g/d of margarine, butter, mayonnaise, and dairy fat with olive oil, in the analysis, was coupled with 8%-34% lower risk of total and cause- specific mortality. Though, when olive oil was compared with other vegetable oils combined, no significant links were registered. Increasing olive oil intake was linked with a lower risk of total and cause-specific mortality.

In the PREDIMED study, 7,450 subjects aged 55 to 80 years, 57% women, at high risk of CVDs, without CVD at enrolment, were assigned to one of three diets: a Mediterranean diet supplemented with extra-virgin olive oil, a Mediterranean diet supplemented with mixed nuts, or a low control diet [15]. After a median follow-up of 4.8 years, there were 96 events in the group assigned to a Mediterranean diet with extra-virgin olive oil (3.8%), 83 in the group assigned to a Mediterranean diet with nuts (3.4%), and 109 in the control group (4.4%). The hazard ratio, as compared to the control diet, was 0.69 for a Mediterranean diet with extra-virgin olive oil and 0.72 for a Mediterranean diet with nuts. The Lyon diet heart study reported that Mediterranean-style diet in conjunction with rape seed oil margarine can cause a significant decline in CVDs among patients with recent myocardial infarction [14]. The Indo- Mediterranean diet study included 1,000 high-risk subjects and administered 400g of fruits, vegetables and nuts in conjunction with another 400g of whole grains and 30-50 g per day of mustard oil [14]. After follow-up of two years, there was a significant decline in CVDs in the intervention group compared to low-fat diet group.

EPA Ethyl Ester

Hypertriglyceridemia is considered as a major risk factor of CAD. Since, icosapent ethyl, a highly purified eicosapentaenoic acid ethyl ester, lowers triglyceride levels, a multicenter, randomized, double- blind, placebo-controlled trial was conducted among 8,179 patients with established CVD or with diabetes, with a fasting triglyceride level of 135 to 499 mg per deciliter (1.52 to 5.63 mmol per liter) [16,17]. The patients were arbitrarily assigned to receive 2 g of icosapent ethyl twice daily (total daily dose, 4 g) or placebo. After follow-up for a median of 4.9 years, a primary end-point event occurred in 17.2% of the patients in the icosapent ethyl group, as compared with 22.0% of the patients in the placebo group (hazard ratio, 0.75; P <0.001). The corresponding rates of the principal secondary end-point were 11.2% and 14.8% (hazard ratio, 0.74; P < 0.001). The rate of cardiovascular death was also notably lower (4.3% vs. 5.2%; hazard ratio, 0.80; P=0.03), but a larger percentage of patients in the intervention group than in the placebo

group was hospitalized for atrial fibrillation or flutter (3.1% vs. 2.1%, P=0.004). Among patients with high triglyceride levels, despite the use of statins, the risk of ischemic events, including cardiovascular death, was considerably lower among those who received 2 g of icosapent ethyl twice daily compared with those who received placebo. In all the above studies, in which various interventions were given, there was either modest decline or no significant decrease in serum cholesterol [17].

Conclusion and Future Perspectives

Conclusively, morbidity and mortality due to CMDs may be reduced by certain interventions without the use of hypocholesterolemic agents and without a significant decrease in blood cholesterol. The results from various studies with nutrients and foods and Mediterranean-style diets as well as icosapent ethyl indicate that beyond serum cholesterol, oxidative stress and inflammation, cardiomyocyte and endothelial dysfunction and platelet aggregation are additional mechanisms, which could be approached for decreasing CVDs. There is an unmet need to find out diets and nutrients, likely to inhibit DPP-4 enzyme or have GLP-1 agonist effects.

Acknowledgement

This editorial is a joint venture between SRIMT, Lucknow and Halberg Hospital & Research Center, Moradabad (U.P., India). Authors are grateful to Mr. Pawan Singh Chauhan Chairman and members of Board of Directors SR Institute of Management & Technology, Bakshi Ka Talab, Lucknow-226201, U.P., and India for their generous support and throughout inspiration for exploring the R&D in the area of Biotechnology.

Conflicts of interest

The authors declare that they have no conflicts of interest with this work.

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