



# DIET IN CARDIOVASCULAR MEDICINE (PART-1)

*( AN EVIDENCE BASED APPROACH )*

# WHY DIET ?

- AS WE KNOW THAT IN OUR DAILY PRACTICE CARDIAC PATIENTS ,THE SECOND QUESTION OF THE PATIENTS AFTER ASKING ABOUT MEDICATIONS IS “**SAHEB MARE JAMVA MA SU LEVU?**” , i.e WHAT ABOUT MY DIET ? WHAT TO EAT AND WHAT NOT?
- SO IT MAKES MANDATORY FOR EVERY TREATING PHYSICIAN AND CARDIOLOGISTS TO HAVE SOUND KNOWLEDGE REGARDING DIET THAT TOO IN ACCORDANCE WITH RECENT ADVANCES AND EVIDENCE BASED STUDIES.

# Association of Diet & CVD

- Cardiovascular diseases (CVD) are growing contributors to global disease burdens, with epidemics of CVD advancing across many regions of the world which are experiencing a rapid health transition. Diet and nutrition have been extensively investigated as risk factors for major cardiovascular diseases like coronary heart disease (CHD) and stroke and are also linked to other cardiovascular risk factors like diabetes, high blood pressure and obesity.
- Adequate evidence is available, from studies conducted within and across populations, to link several nutrients, minerals, food groups and dietary patterns with an increased or decreased risk of CVD.

# Contributors for Global NCD burden and its root causes

- The second half of the 20<sup>th</sup> century witnessed major health transitions in the world, propelled by socio-economic and technological changes that profoundly altered life expectancy and ways of living.
- . The most globally pervasive change among these health transitions has been the rising burden of noncommunicable diseases (NCDs). Epidemics of NCDs are presently emerging, or accelerating, in most developing countries. Cardiovascular diseases (CVD), cancers, diabetes, neuropsychiatric ailments and other chronic diseases are becoming major contributors to the burden of disease, even as infections and nutritional deficiencies are receding as leading contributors to death and disability

# Food & Facts

Adequate evidence is available, from studies conducted within and across populations, to link several nutrients, minerals, food groups and dietary patterns with an increased or decreased risk of CVD, like...

- > Dietary fats associated with an increased risk of CHD include trans-fats and saturated fats, while polyunsaturated fats are known to be protective.
- > Dietary sodium is associated with elevation of blood pressure, while dietary potassium lowers the risk of hypertension and stroke. Regular frequent intake of fruits and vegetables is protective against hypertension.
- > Composite diets (such as DASH diets, Mediterranean diet, 'prudent' diet) have been demonstrated to reduce the risk of hypertension and CHD.

# **Nutrients and their association with CVD (for PART-1):**

- Dietary Fats
- Carbohydrates
- Fibres
- Antioxidants
- Minerals

# Dietary fats: general outlook

- The relationship between dietary fats and CVD, especially CHD has been extensively investigated, with strong and consistent associations emerging from a wide body of evidence accrued from animal experiments, as well as observational studies, clinical trials and metabolic studies conducted in diverse human populations. This relationship was initially considered to be mediated mainly through the atherogenic effects of plasma lipids (total cholesterol, lipoprotein fractions and triglycerides).
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- Cholesterol in the blood and tissues is derived from two sources: diet and endogenous synthesis.
- Dietary cholesterol raises plasma cholesterol levels.
- Dairy fat and meat are major sources.
- Egg yolk is particularly rich in cholesterol but unlike dairy and meat does not provide saturated fatty acids (SFAs).
- Fatty acids are grouped into three classes—SFAs, monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs). While such a classification is useful in providing a structural grouping, it tends to oversimplify the effects of dietary fats. Individual fatty acids, within each group, are now known to have differing effects on lipids, lipoproteins and platelet-vascular homeostasis. SFA and MUFA can be synthesised in the body and hence are not dietary essentials. PUFA can be subdivided into n-6 and n-3 PUFA, derived from linoleic acid (LA) and  $\alpha$ -linolenic acid (ALNA), respectively. These are essential fatty acids, since they cannot be synthesised in the body.

# Saturated fatty acid (SFA)

- SFAs as a group raise total and LDL cholesterol, but individual SFAs have different effects. Myristic and lauric acids have greater effect than palmitic acid, but the latter is more abundant in food supply. The plasma cholesterol raising effects of these three SFAs is higher when combined with high cholesterol diets. Stearic acid has not been shown to elevate blood cholesterol and is rapidly converted to oleic acid (OA) in vivo. Metabolic (feeding) studies demonstrate a marked elevation of both HDL and LDL cholesterol induced by SFA diets.
- The relationship of dietary saturated fat to plasma cholesterol levels and to CHD was graphically demonstrated by the Seven Countries Study involving 16 cohorts, in which saturated fat intake explained 73% of the total variance in CHD across these cohorts<sup>14</sup>. In the Nurses Health Study<sup>19</sup>, the effect of saturated fatty acids was much more modest, especially if saturates were replaced by carbohydrates.

# Trans-fatty acids (t-FAs)

- t-FAs are geometrical isomers of unsaturated fatty acids that assume a saturated fatty acid-like configuration. Partial hydrogenation, the process used to create t-FA, also removes essential fatty acids such as LA and ALNA. Metabolic studies have demonstrated that t-FAs render the plasma lipid profile even more atherogenic than SFA, by not only elevating LDL cholesterol to similar levels but also decreasing HDL cholesterol. As a result, the ratio of LDL cholesterol to HDL cholesterol is significantly higher with a t-FA diet than with a SFA diet . **This greatly enhances the risk of CHD.**
- Hence eliminating t-FAs from the diet would be an important public health strategy to prevent CVD.

# Monounsaturated fatty acids

- The only nutritionally important MUFA is OA, which is abundant in olive and canola oils and also in nuts. The epidemiological evidence related to MUFA and CHD is derived from studies on the Mediterranean diet, as well as from the Nurses Health Study.
- MUFAs have been shown to lower blood glucose and triglycerides in type II diabetics and may decrease susceptibility of LDL to oxidative modification.

# Polyunsaturated fatty acids

- PUFAs are derived from Dietary LA (n-6 PUFAs) and dietary ALNA (n-3 PUFAs).
- The biological effects of n-3 PUFAs are wide ranging involving lipids and lipoproteins, blood pressure, cardiac function, arterial compliance, endothelial function, vascular reactivity and cardiac electrophysiology as well as potent anti-platelet and anti-inflammatory effects including reduced neutrophil and monocyte cytokine production.

## Various studies conducted on PUFA:

- Much of the epidemiological evidence related to n-3 PUFAs is derived from the study of fish consumption in populations or interventions involving fish diets in clinical trials. Fish oils were, however, used in the GISSI study of 11,300 survivors of myocardial infarction. In this factorial design, fish oil (1g/d) and vitamin E (300mg/d) were compared, alone and in combination, to placebo. After 3.5 years of follow-up, the fish oil group had a statistically significant 20% reduction in total mortality, 30% reduction in cardiovascular death and 45% decrease in sudden death. While most published studies do not indicate that dietary n-3 PUFA prevent restenosis after percutaneous coronary angioplasty or induce regression of coronary atherosclerosis, one study reported that occlusion of aortocoronary venous bypass grafts was reduced after 1 year by daily ingestion of 4g fish oil concentrate. The Lyon Heart Study incorporated an n-3 fatty acid (ALNA) into a diet altered to develop a 'Mediterranean diet' intervention. In the experimental group, plasma ALNA and EPA increased significantly and the trial reported a 70% reduction in cardiovascular mortality at 5 years in its initial report. Total and LDL cholesterol were identical in the experimental and control groups, suggesting that thrombotic and perhaps arrhythmic events may have been favourably influenced by n-3 PUFA. Since the diet altered many other variables, such as fibre and antioxidants (by increasing fruit and vegetable consumption), direct attribution of benefits to n-3 PUFA becomes difficult to establish.

# Effect of different fatty acids on cardiac arrhythmias:

- Diets rich in saturated fatty acids increase the risk of ventricular fibrillation and sudden cardiac death in primates. A recent population based case–control study, using biomarkers, revealed a modest association of t-FAs in general and a strong association of trans isomers of LA in particular, with primary cardiac arrest in humans. Several studies in different animal models, primate and rodent, have shown that n-3 PUFA are protective against cardiac arrhythmias, especially ventricular fibrillation

# Dietary fat considerations and recommendations

- Enhancing the nutritional quality of dietary fat consumption, to provide greater cardiovascular protection, may be attempted by decreasing the sources of saturated fats and eliminating t-FAs in the diet, increasing the consumption of foods containing unsaturated fatty acids (both MUFA and PUFA) and decreasing dietary cholesterol consumption.
- Modification of cooking oils either through appropriate admixture of different oils or through genetic modification of oilseed crops may provide methods for improving the quality of dietary fat consumed through edible oils.

# Carbohydrates

- The relationship of dietary carbohydrates to CVD appears to be mediated through indirect mechanisms: contribution to total energy and its effect on overweight and obesity; influence on central obesity; effects on plasma lipids, especially triglycerides and effects on glycaemic control.
- High-carbohydrate diets appear to reduce HDL cholesterol levels and increase the fraction of small dense LDL, both of which may impact adversely on vascular disease. This dyslipidemic pattern is consistent with the elevation of plasma triglycerides
- The glycaemic index of foods might also be a determinant of the extent to which carbohydrates can influence the glycaemic status. Carbohydrate diets with high-glycaemic index might adversely impact on glucose control, with associated changes in plasma lipids

# Dietary fibre

- Dietary fibre is a heterogeneous mixture of polysaccharides and lignin that cannot be degraded by the endogenous enzymes of vertebrate animals.
- Watersoluble fibres include pectins, gums, mucilages and some hemicelluloses. Insoluble fibres include cellulose and other hemicelluloses. Most soluble fibres reduce plasma total and LDL cholesterol concentrations, as reported by several trials. Pectins, psyllium, gums, mucilages, algal polysaccharides and some hemicelluloses lower total and LDL cholesterol levels without affecting HDL cholesterol, the reductions in total cholesterol being usually in the range of 5–10%.
- Fibre consumption predicted insulin levels, weight gain and cardiovascular risk factors like blood pressure, plasma triglycerides, LDL and HDL cholesterol and fibrinogen more strongly than other dietary components in the CARDIA cohort study of young adults.

# Anti-oxidants

- The oxidation of LDL by oxygen free radicals results in the unregulated uptake of modified LDL by macrophages in arterial walls, accelerating the atherosclerotic process. Anti-oxidant nutrients, which can directly scavenge free radicals, include  $\alpha$ -tocopherol (vitamin E isomer) and ascorbic acid (vitamin C), which have shown anti-oxidant activity both in vitro and in vivo, as well as  $\beta$ -carotene (a provitamin A carotenoid) which has displayed antioxidant activity in vitro.
- These mechanisms suggested that increased dietary intake or supplementation of these nutrients would be protective against atherosclerotic vascular disorders.

# Folate

- The relationship of folate to CVD has been mostly explored through its effect on homocysteine, which has been incriminated as an independent risk factor for CHD and probably stroke.
- Folic acid is required for the methylation of homocysteine to methionine.
- Reduced plasma folate has been strongly associated with plasma elevated plasma homocysteine levels and folate supplementation has been demonstrated to decrease those levels
- There is also recent evidence that suggests that homocysteinaemia results in endothelial dysfunction, an effect that is reversed by oral folate supplementation.
- Data from the Nurses' Health Study showed that folate and vitamin B6, from diet and supplements, conferred protection against CHD (fatal and non-fatal events combined) and suggested a role for their increased intake as an intervention for primary prevention of CHD. Food grain fortification with folate and cyanocobalamin has also been recommended as a cost-effective measure for CHD prevention.
- Dietary intake of folate through natural food sources may be encouraged in the meanwhile, especially in individuals at a high risk of arterial or venous thrombosis and elevated plasma homocysteine levels.

# Flavonoids and other phytochemicals

- Flavonoids are polyphenolic anti-oxidants which occur in a variety of foods of vegetable origin, such as tea, onions and apples.
- Data from several prospective studies indicate an inverse association of dietary flavonoids with CHD.
- Fruits and vegetables also contain other phytochemicals that may have protective properties, including isothiocyanates and indoles (found in cruciferous vegetables), sulphides (found in onions and garlic), terpenes (found in citrus oils) and phytoestrogens. While their role in relation to CVD risk is not clearly established and trial evidence related to garlic supplements is generally not supportive, their consumption in the natural food form may have benefits, which need to be evaluated.

# Minerals: blood pressure and CVD

- Sodium: High blood pressure (HBP) is a major risk factor for CHD and both forms of stroke (ischaemic and haemorrhagic). The relative risk of CVD for both systolic and diastolic blood pressures, operates in a continuum of increasing risk for rising pressure but the absolute risk of CVD is considerably modified by co-existing risk factors.
- Salt or sodium intake has been directly correlated with mean blood pressure levels and prevalence of hypertension in many populations. Comprehensive epidemiological evidence was provided by the INTERSALT Study, which investigated the relationship of 24hr urinary electrolyte excretion to blood pressure in 52 population groups across 32 countries, using standardised methodology to provide comparable data.

- In adults aged 20–59 years, there was a significant positive relationship between urinary sodium excretion and blood pressure across the 52 population samples. Further, it was also observed that in four of these populations in whom the mean 24hr urinary sodium excretion was lower than 100mmol/d, systolic blood pressure did not rise with age.
- The results of low sodium—DASH diet trial<sup>80</sup> further strengthen the conclusion that reduction of daily sodium intake, through salt restricted diets, lowers blood pressure effectively and is additive to the benefits conferred by the DASH diet. This trial revealed that low sodium diets, with 24hr sodium excretion levels around 70mmol/d, are effective and safe. Sodium consumption has also been linked to the presence of left ventricular hypertrophy and restricted sodium intake has been demonstrated to result in regression of this important indicator of cardiovascular risk
- Based on various observational and trial data so far available, it would be justified to recommend a daily salt intake of less than 5g/dl.

# Potassium

- Cardioprotective effects of dietary potassium have been hypothesised as the basis for low CVD rates in populations consuming 'primitive' diets and in vegetarians in industrialised cultures. The INTERSALT study provided evidence of an inverse association between urinary potassium excretion and blood pressure levels, across diverse populations. Migrant studies too revealed a rise in blood pressure when diets changed to a lower potassium and higher sodium intake.
- Whelton et al. concluded, from a meta-analysis of randomised controlled trials, that potassium supplements reduced mean blood pressures (systolic/diastolic) by 1.8/1.0mmHg in normotensive subjects and 4.4/2.5mmHg in hypertensive subjects<sup>90</sup>. An increase in dietary intake of potassium, from approximately 60– 80mmol/d was shown to be inversely and significantly related to the incidence of stroke mortality in women.

# Calcium and magnesium

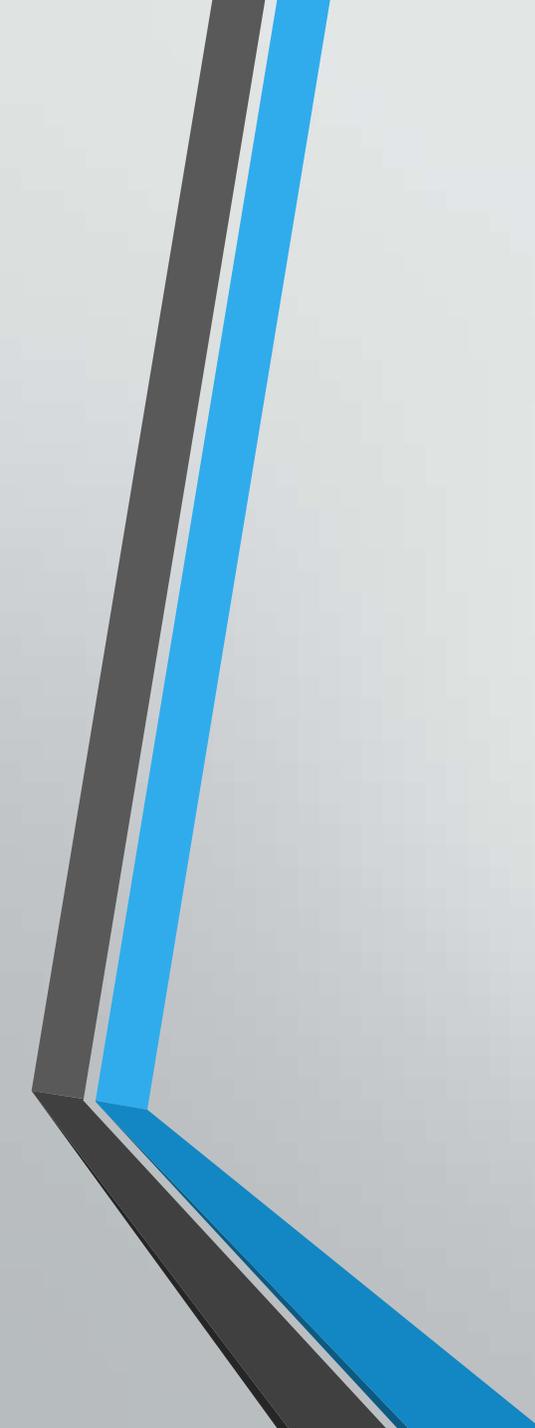
- A meta-analysis of studies involving calcium supplements reveal modest effects on blood pressure. The estimated blood pressure reduction was 2.1mmHg for systolic blood pressure and 1.1mmHg for diastolic blood pressure. A review of 29 studies of magnesium was inconclusive due to methodological problems but suggested that there was no negative association of blood pressure with magnesium.

# Dietary minerals recommendations.

- There is presently no evidence to recommend public health or clinical interventions involving the use of these minerals for cardiovascular protection in populations or individuals, other than in the form of a balanced diet providing an adequate daily intake.

# Topics to be discussed later, in Part-2 ...

- Food items and products (fruits, veggies, fish, soy, nuts, eggs, dairy products, alcohol).
- Dietary patterns and composite dietary interventions (Mediterranean, DASH, Japanese)
- Policy implications for balanced diet
- Diet and CVD: summary of evidence and recommendations
- Research/national/international recommendations.



THANKS FOR KIND ATTENTION

“GOOD DAY”