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EDITOR'S NOTE

Dr Manoj Durairaj

Heart Transplant Surgeon, MS, MCh. (AIIMS, New Delhi), FACC.

Director, Marian Cardiac Centre and Research Foundation.

Program Director, Department of Heart and Lung Transplantation, Sahyadri Hospitals, Pune.

Dear Colleagues,

Greetings from the Editor's desk. At the outset I would like to record my thanks to Dr Mansi Patil, Director, Indian Association for Parenteral and Enteral Nutrition, for accepting to be the guest author for the September issue of "The Revival". Dr Mansi has written a detailed article on Nutrition and Heart Failure. She has beautifully illustrated the deleterious effects of increased sodium intake, high refined carbohydrate and saturated fats and their contribution in the increasing incidence CVD and heart failure. The role of iron replacement therapy (the Fifth Pillar in heart failure management) and the treatment protocol have also been propounded in this article. Therapeutic diets like DASH and Med Diet have been discussed. The exciting field of GUT HEART AXIS and its role in heart failure and various methods of dietary introduction of pro and prebiotics and their role in modulating heart failure have been alluded to by Dr Mansi.

I'm sure our dear Readers will be enthused over this article. Happy Reading!

Dr Manoj Durairaj
Editor "The Revival"

SUB EDITOR

Dr Talha Meeran

MBBS, MD, FACC, Consultant Cardiologist, Dept of Advanced Cardiac Sciences and Cardiac Transplant, Sir HN Reliance Foundation Hospital, Mumbai.

Dear Colleagues,

Nutrition and diet play a major role in our lives and more so for individuals with cardiovascular diseases like heart failure. Yet, we often do not emphasize enough on nutrition's role when counselling our patients. This exhaustive and in-depth review article from Dr Mansi Patil discusses all aspects of how several dietary factors can influence cardiovascular outcomes and help patients improve their disease burden and quality of life. In addition to the role of the much discussed salt, dietary fat and carbohydrate consumption, the paragraph on gut microbiota is particularly intriguing and thought provoking. Such articles serve as an additional reminder for developing a holistic approach when managing our heart failure patients and underline the importance of including qualified nutritionists or dietitians as part of our heart failure medical team.

Sincerely,
Dr Talha Meeran
Sub Editor "The Revival"

PRESIDENTIAL MESSAGE

Prof. (Dr) V. Nandakumar

Director & Chief, Division of Cardio Vascular/Thoracic Surgery & Cardiac Transplantation, Metromed International Cardiac Centre, Calicut, Kerala.

Dear Colleagues,

The September issue of ' The Revival ' has Dr.Mansi Patil's article on Nutrition and Heart Failure.In this article, she clearly depicts the importance of nutrition in the management of heart failure. She has covered this topic in detail stressing the relevance of salt and sugar consumption as well as the role of carbohydrate diet and treatment of anaemia. This will be a useful guide while managing patients with heart failure where attention to nutrition needs emphasis.

Best wishes,
Prof. (Dr) V. Nandakumar
President

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Special thanks to Dr Mansi Patil for authoring this month's article.

Designed by Maithili Kulkarni

NUTRITION AND HEART FAILURE

Dr Mansi Patil
Ph D, Nutrition Consultant

Dr. Mansi Patil is a practicing clinical nutritionist with a focus on Cardio-metabolic syndrome and holds a PhD in Environmental Sciences. Her work is focused on the Preventive aspect of Cardio-metabolic syndrome and its epidemiology especially related to hypertension and its dietary, lifestyle and behavioural risk factors.

She is the Director of IAPEN India Association for Parenteral and Enteral Nutrition and is actively involved in conducting educational programs for medical and para-medical professionals. She has initiated the Hypertension and Nutrition, a Core group of IAPEN India which is the first of its kind in India, and has developed a certificate course on lifestyle management in hypertension (endorsed by ISH). She is working with Victoria University, Australia for their MPH programs and for the Master's programs on Public Health Nutrition offered by the Maharashtra University of Medical Sciences (Pune Centre, India).

She is a visiting faculty with the Symbiosis Institute of Health Sciences. She is currently a Committee member of the Women in Hypertension Committee with the International Society of Hypertension and a Committee member of the American Society of Preventative Cardiology. She is also a committee member with the Regional Advisory Group for South and Central Asia for Hypertension. She is a trained UNICEF Nutrition Rehabilitation Centre expert and is a ToT (Trainer-of-Trainers) consultant for the Infant and Young Child Nutrition (IYCF) program. She has participated and presented her research at various national and international conferences. She has authored the book- Lifestyle management in Hypertension.

Introduction:

Nutrition and heart failure

Heart failure (HF) is a significant health care burden that is becoming more prevalent over time. In order to increase patient longevity, symptom control, and quality of life, effective, evidence-based therapies for HF prevention and management are required. The main source of energy for myocardial contractility and maintaining heart efficiency is nutrition. Although numerous studies have shown the advantages of dietary intervention in individuals with chronic heart failure (CHF), these effects are still not fully understood. However, the ability of healthcare professionals to put clinical advice into practise is constrained due to the lack of agreement on complete nutritional recommendations and practical evidence. Precision nutrition techniques have the potential to improve clinical treatment, lower the burden of HF, and better serve the needs of a wide range of patients through the refinement of medical nutrition therapy.

At risk population

1. **Aging**- People aged 65 years and over are at a higher risk of developing heart failure.
2. **Family history of heart failure** increases the chance of developing heart failure. Initial research has proposed the role of genetics also as as risk factor, although further evidence is required to establish the role of genetics for the same.
3. **Unhealthy lifestyle behaviors**, such as a diet rich in saturated fats, low in fiber, high in simple carbohydrates, smoking, risky alcohol use, and a lack of physical activity, may also either trigger or contribute towards heart failure.
4. Heart or blood vessel diseases, severe lung disease, or viruses like **HIV or SARS-CoV-2** pose a higher risk.
5. Obesity, high blood pressure, diabetes, sleep apnea, chronic renal disease, anemia, thyroid illness, or iron excess may act as contributing factors as well.
6. Cancer therapies like radiation and chemotherapy can damage lead to damage of the heart tissue
7. **Black and African American persons** are more likely than other races to suffer heart failure at a younger age[8].

Heart failure stages

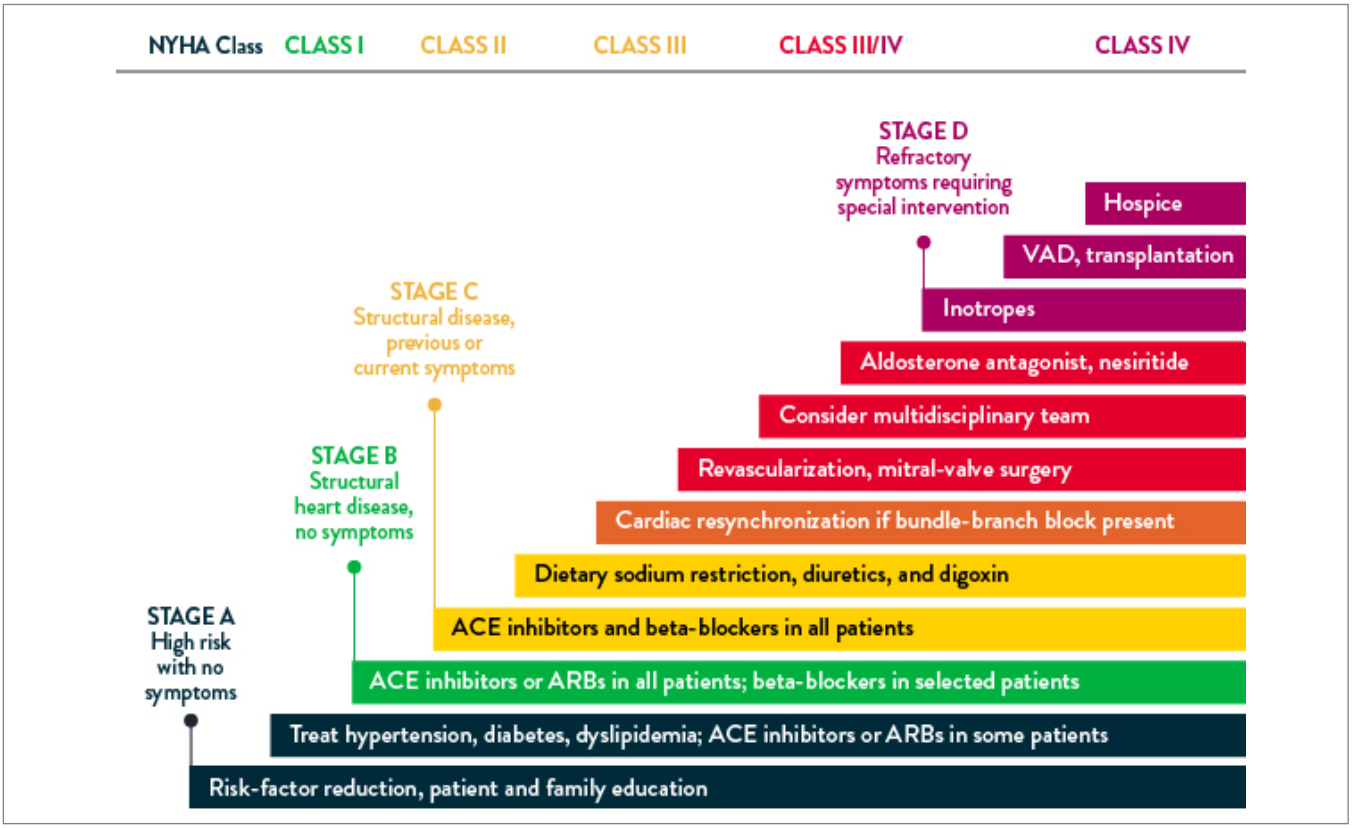

Fig 1: Stages of Heart Failure.Jessup M, Brozena S. Heart failure. N Engl J Med. 2003;348:2007-2018.

Dietary Recommendations:

1. Salt consumption

Chronic comorbidities such as hypertension, heart failure (HF), chronic renal disease, stroke, cardiovascular disorders, and an increase in mortality can be driven by a high sodium or salt intake. A dose-response effect between sodium intake and systemic hypertension has been established, thus strengthening th eevidence for sodium restriction in cases with underlying comorbidities. Previous research has demonstrated that HF patients have systemic inflammation, characterised by elevated levels of chemokines (monocyte chemoattractant protein-1 and IL-8), interleukin (IL)-1B and IL-6, tumour necrosis factor (TNF)-alpha, and adhesion molecule expression. When compared to low sodium restriction (up to 1.8 g/d)18, moderate sodium restriction (up to 2.8 g/d) was linked to decreased levels of neurohormones (B-type natriuretic peptide (BNP), aldosterone, plasma renin activity), and cytokines (TNF-alpha, IL-6) and increased

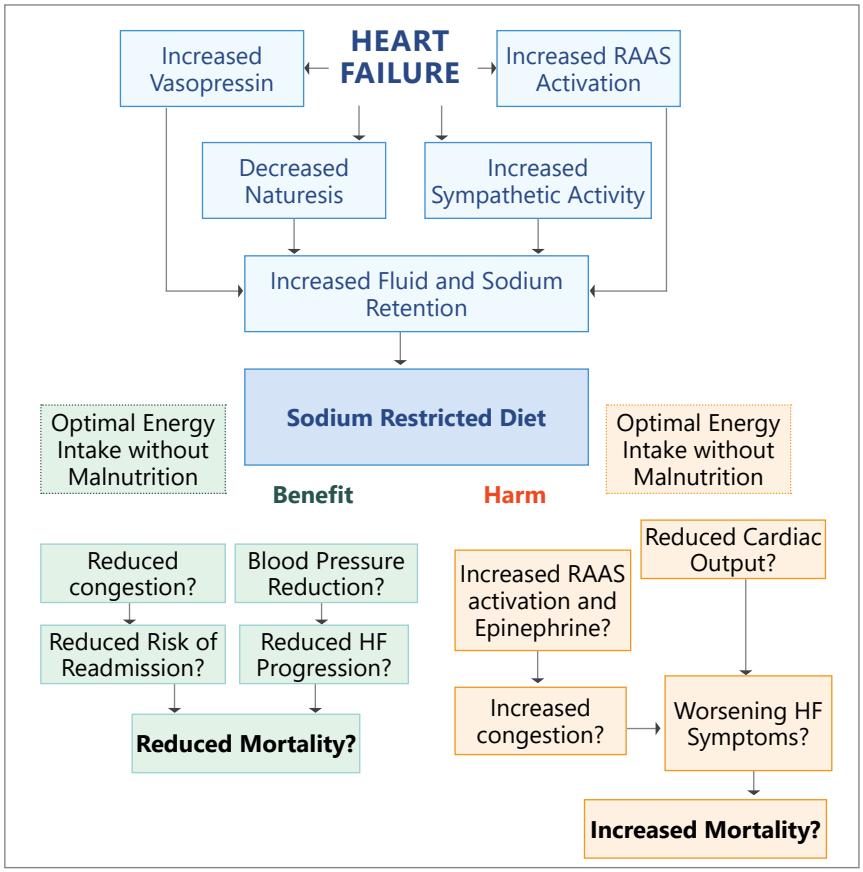

Fig 2: Effects of sodium restriction in heart failure[4]

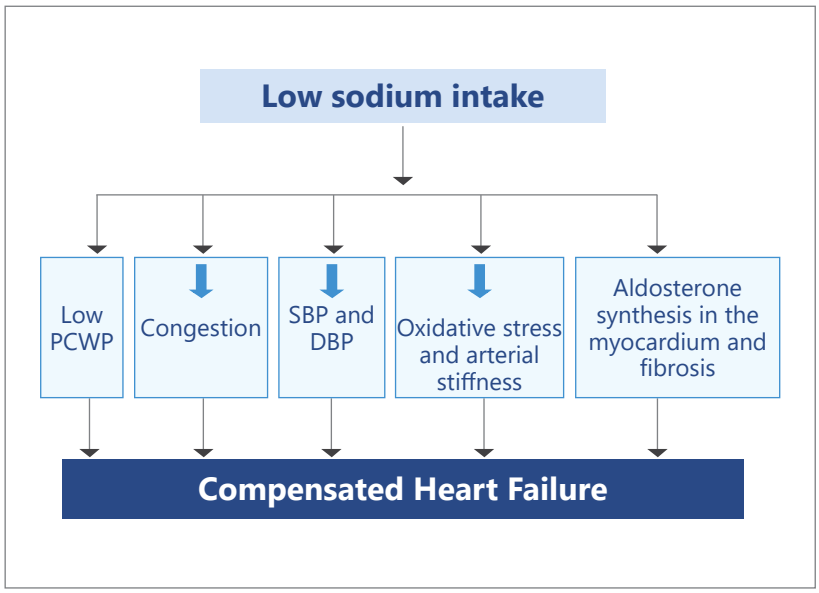

Fig 3: Heart failure and salt pathways

levels of anti-inflammatory cytokines (IL-10). Therefore, current recommendations advise limiting sodium intake to 2-3 g/day.

Multiple mechanisms have been proposed for the benefits of restricting sodium intake in the diet, although evidence supports the restriction of sodium in Stage A and Stage B (asymptomatic) patients satisfactorily.

In some studies on animal models a comparison between normal and excessive sodium consumption, sodium restriction in the early stages of HF was observed to be related with early aldosterone activation in animal models. These results imply that sodium restriction in the early stages of HF should be avoided to stop the advancement of neuroendocrine illness[19].

In a post-hoc study, patients who accomplished a sodium limitation of less than 1500 mg /day did not show a significant difference from the intervention group consuming 2300 mg/day although there was a reduction in BNP levels and KCCQ score from the baseline level in the former group. [4]

2. Refined Flours and Sugar Consumption

Carbohydrate consumption causes hypertriglyceridemia primarily via increasing hepatic synthesis of very low-density lipoprotein and potentially by decreasing lipoprotein lipase activity. A high refined-carbohydrate diet suppresses HDL levels in a healthy person, a lipoprotein that protects against CHD. Many metabolic investigations have demonstrated that high-carbohydrate diets raise fasting triglyceride levels and promote long-term increases in plasma residual lipoprotein cholesterol and triglycerides.

High-carbohydrate diets tend to enhance fat accumulation by converting excess carbohydrate to triacylglycerols and decreasing fat oxidation. Aside from lipid abnormalities, hyperglycemia and hyperinsulinemia caused by a high carbohydrate diet can cause hypertension, decreased fibrinolysis, and endothelial and inflammatory responses that are linked with an elevated risk of CHD.

Garcia-Palmieri and associates used a 24-hour recall to examine baseline diets of 8,218 men in the Puerto Rico Heart Health Program and discovered a statistically significant negative relationship between carbohydrate intake and CHD. Following adjustment for age, smoking status, total energy intake, and other dietary and non-dietary coronary risk factors, dietary GL was associated prospectively with risk of CHD in a recent analysis of NHS data that assessed diets multiple times with a detailed semi-quantitative food frequency questionnaire. Furthermore, categorizing carbs by GI, rather than the standard categories of simple and complex, was a stronger predictor of CHD risk. A substantial association between BMI and glycemic load was also discovered. A high glycemic load diet did not significantly increase the risk of CHD in lean women (BMI less than 23), but was associated with a twofold increase in risk in women with an average or above-average BMI (BMI greater than 23), implying that insulin resistance plays an important role in modifying the effect of dietary GL on CHD risk.[11]

Preclinical research, on the other hand, suggests a direct influence of dietary components, including saturated fat and carbohydrates, on diastolic function, which appears to be independent of weight growth. A group of rats fed a high-sugar, low-saturated fat diet (high-sugar diet) had significantly lower diastolic function than those on the usual diet.[12]

A study by Jianbo Na et.al., on Drosophila Melanogaster model compelling it to a high sugar diet. The study observed the induction of a high sugar diet induced heart failure. The flies fed on high sugar diet exhibited flagging heartbeat along with patterns of irregular heartbeats, and reduced life span compared to the flies fed on low sugar diet.[13]

3. Saturated fat Intake

Although the degree of the effect is likely to vary depending on specific dietary patterns and individual sensitivity, it is well acknowledged that saturated fats can elevate total cholesterol levels in the blood. Because the bulk of blood cholesterol is packed in low-density lipoproteins (LDL), increases in TC correspond to increases in LDL. LDL is supposed to increase the risk of CHD, and it is also known as "bad cholesterol[14].Without any sex-related changes, Western Diet-fed mice had a substantial drop in LV ejection fraction (LVEF), indicating compromised systolic function, and a significant increase in isovolumetric relaxation time (IRT), myocardial performance index (MPI), and LVEDP, indicating impaired diastolic function[15].

Saturated fat consumption has been related to an increased risk of cardiovascular disease (CVD), and this impact is assumed to be mediated mostly by higher LDL cholesterol concentrations. Full-fat dairy products and red meat are major dietary sources of saturated fatty acids in the United States. Clinical trial data show that substituting polyunsaturated fat for saturated fat reduces the incidence of CVD; however, as described below, there is little evidence from such trials or epidemiologic studies that reducing saturated fat intake below 9% of total energy intake is associated with a lower CVD risk. Recommendations for additional reductions in saturated fat intake (for example, to 7% of total energy) are essentially based on the prognosis of a gradual reduction in CVD risk linked with larger LDL cholesterol reductions. However, from an operational viewpoint, additional decreases in saturated fat intake generally entail dietary recommendations that contain a higher proportion of carbohydrate.[16]

Anemia in Chronic Heart failure

Iron Therapy for Chronic Heart failure

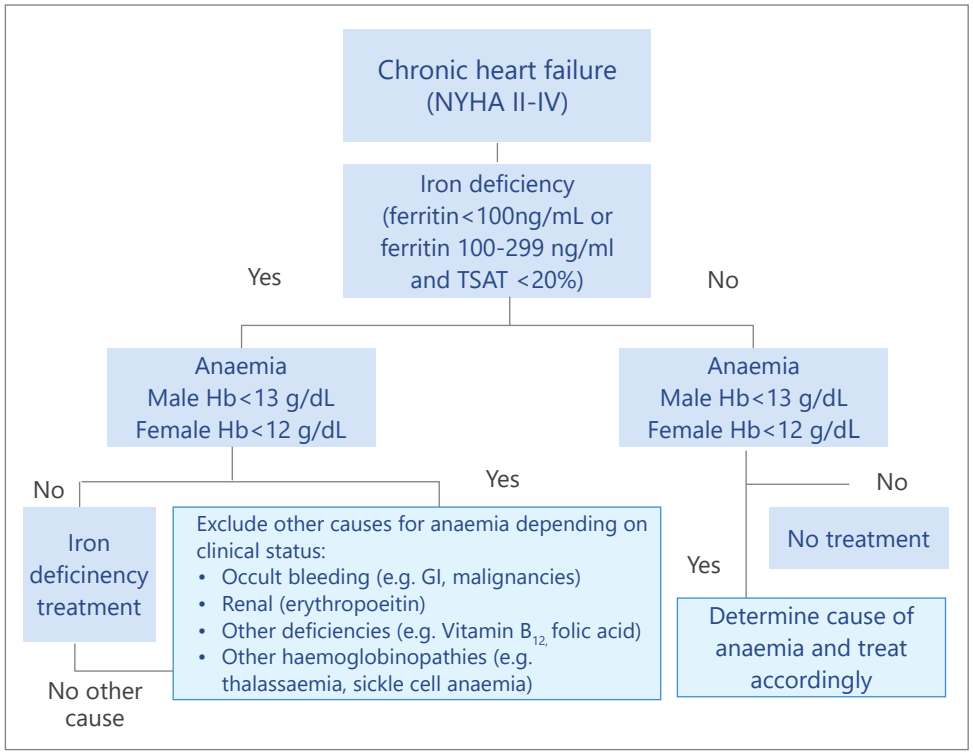

Fig 3: Suggested algorithm for diagnosis of iron deficiency in patients with heart failure.[2]

It should be noted that iron deficiency (ID) can develop without a fall in hemoglobin. Beyond the classic understanding of ID as the source of anemia, the spectrum of negative health and economic implications associated with ID is broad, including, to mention a few, poor pregnancy outcomes, poorer school performance, and lower productivity. Importantly, despite the fact that ID is associated with a variety of chronic illnesses and disorders, the bulk of randomized controlled trials (RCTs) focused on ID and iron repletion in patients with chronic kidney disease (CKD). Nonetheless, ID has been widely investigated in patients with other chronic conditions, including heart failure (HF), in recent years.

This microelement is especially crucial for tissues with high energy demands (e.g., myocardium, skeletal muscles) or strong mitogenic activity (e.g. haematopoietic cells). The presence of ID is also linked to abnormal haematopoiesis (erythroid, lymphoid and thrombocyte cell lines). Studies on HF patients have revealed a decline in total exercise capacity as well as more severe HF symptoms such as tiredness and exertional dyspnea. Clinical advantages of iron treatment in iron-deficient patients with HF are thus expected to arise not just from increased hemoglobin concentration, but also from improved non-haematopoietic tissue function, such as skeletal muscles.

Ferric sorbitol, iron dextrans (high- and low-molecular weight dextran), iron polymaltose, iron sucrose (ISC), ferric gluconate, ferric carboxymaltose (FCM), iron isomaltoside 1000, and ferumoxytol are among the parenteral (IV or intramuscular) iron formulations permitted for therapeutic use. Five of them were formally studied in HF patients. ISC was given in seven studies (to a total of 136 patients), while FCM was given in two multicenter, randomized, placebo-controlled, double-blind trials (a total of 454 patients). Only one short single-center research looked into iron dextran, iron isomaltoside 1000, and ferric gluconate. FCM, iron isomaltoside 1000, and ferumoxytol are considered more stable iron compounds that degrade more slowly. These formulations enable the administration of large single doses of iron. Iron dextran can also be given in large doses, but its safety profile is inferior to that of FCM, iron isomaltoside 1000, and ferumoxytol because it causes more severe immunological responses, including life-threatening anaphylaxis and delayed hypersensitivity-like incidents1. During the 1-year trial period, the mean and median total dosage in the FCM arm was 1500 mg of iron, with a dosing range of 500-3500 mg of iron. Over 75% of the patients required no more than two FCM injections to fix and maintain their iron levels[3].

Diets in HF :

While the DASH diet encourages a high potassium consumption while reducing sodium, SFA, and total fat, the MedDiet stresses a higher intake of unsaturated fatty acids (UFA), which are found in fatty fish, extra-virgin olive oil, canola oil, and mixed nuts.Large observational trials, on the other hand, have shown that adhering to a MedDiet reduces the risk of HF. Over a 10-year period, two large prospective Swedish cohorts of over 30,000 men and women discovered that strong adherence to the MedDiet led in a substantial decrease in the incidence of HF in both women (relative risk (RR): 0.79, 95% CI 0.68-0.93) and men (RR: 0.69, 95% CI 0.57-0.83). Higher adherence to the MedDiet did not result in a significant decrease in HF incidence in a multivariate adjusted model (HR: 0.82, 95% CI 0.64-1.05) in a German cohort of nearly 24,000 participants followed for 8 years, except when milk products were omitted from the MedDiet score (HR 0.75, 0.55–0.97). Both the MedDiet and the DASH diet are high in plant-based foods while being low in processed foods and red meat. In a large nationally representative American population, a plant-based dietary pattern was found to be linked with a considerably decreased risk of HF (HR: 0.59, 95% CI 0.41-0.86). Furthermore, in a large Swedish female cohort, increased intake of fruits and vegetables was substantially related with a decreased risk of HF (RR: 0.82, 95% CI 0.72 to 0.94), with the lowest probability of HF occurring among women consuming more than 5 servings of fruits and vegetables per day[4].

Gut & prevention of HF :

In a study conducted by Marques et. al. in 2016 concluded that, independent of mineralocorticoid excess, fiber ingestion altered gut microbiota populations and increased the number of acetate-producing bacteria. Fiber and acetate both reduced gut dysbiosis as judged by the Firmicutes: Bacteroidetes ratio and increased the prevalence of Bacteroides acidifaciens. Both a high-fiber diet and acetate supplementation significantly lowered systolic and diastolic blood pressures, cardiac fibrosis, and left ventricular hypertrophy in mineralocorticoid-excess animals fed a control diet. Acetate exhibited comparable effects and significantly decreased kidney fibrosis. Transcriptome analysis revealed that increased fiber and acetate consumption was associated with a decrease in cardiac and renal Egr1, a master cardiovascular regulator implicated in ventricular hypertrophy, cardiorenal fibrosis, and inflammation5. Westernized red meat consumption encourages bacterial formation of Trimethylamine, TMA, which is then oxidized in the liver to the pro-atherogenic metabolite trimethylamine- N oxide,TMAO. By interfering with cholesterol transport, foam cell formation, and platelet aggregation, TMAO may contribute to atherosclerosis, with the latter potentially having a role in acute coronary syndromes. Reduced fiber consumption is connected with decreased bacterial synthesis of the short chain fatty acid butyrate, which has immunological modulatory effects in the gut mucosa and also serves as the primary energy source for colonocytes. Reduced butyrate levels in the stomach may promote local inflammation, worsen dysbiosis, and lead to decreased gut barrier function, which may result in leakage of bacterial toxins like LPS, fuelling local and systemic inflammation[6].

Probiotics in therapeutic application include microorganisms from the families Lactobacillus and Bifidobacterium, as well as the fungus Saccharomyces boulardii. Animal studies show that specific Lactobacilli strains may have cardioprotective properties. Before coronary artery ligation, rats were given a supplement containing Lactobacillus plantarum 299v, which decreased infarct size and enhanced left ventricular function. Another study found that Lactobacillus rhamnosus GR-1 supplementation had comparable cardioprotective effects in a rat model of myocardial ischemia.In humans, a pilot research found that the probiotic yeast Saccharomyces Boulardii not only decreased systemic inflammation but also improved left ventricular ejection fraction in patients with chronic heart failure[6].

Prebiotics, such as nondigestible dietary fibers and oligosaccharides, are substrates that are preferentially consumed by host bacteria and give a possible health benefit. Most recent deep sequencing investigations of individuals with cardiovascular disease show a decrease of microorganisms capable of generating SCFAs like butyrate. Prebiotics that promote microbial fermentation of dietary fibers to SCFAs may thus be beneficial in the gut, as well as in splanchnic and peripheral tissues, potentially leading to enhanced metabolic control. Some prebiotics, such as inulin, may be able to mitigate the negative effects of antibiotics by increasing the variety and functional capability of the gut microbiota[6].

Fecal Microbiota Transplant (FMT) is a well-established therapy for recurrent Clostridioides difficile infection and the most radical contemporary technique for addressing the gut flora. Previously, FMT from lean donors was reported to improve insulin sensitivity in obese patients with metabolic syndrome, however the benefit was only transient. In a later trial on 20 patients with the metabolic syndrome, FMT from vegan donors shifted the gut microbiota composition toward a vegan profile in some patients, but not TMAO production capability or vascular inflammation markers. FMT is presently being evaluated in numerous therapeutic settings, however its usage in acute and high-risk scenarios such as acute coronary syndrome and decompensated HF is limited due to the current technique of endoscopic administration.FMT is not without hazard, since it has recently been demonstrated to transfer drug-resistant E. Coli, leading to bacteremia in two patients, one of whom died[6].

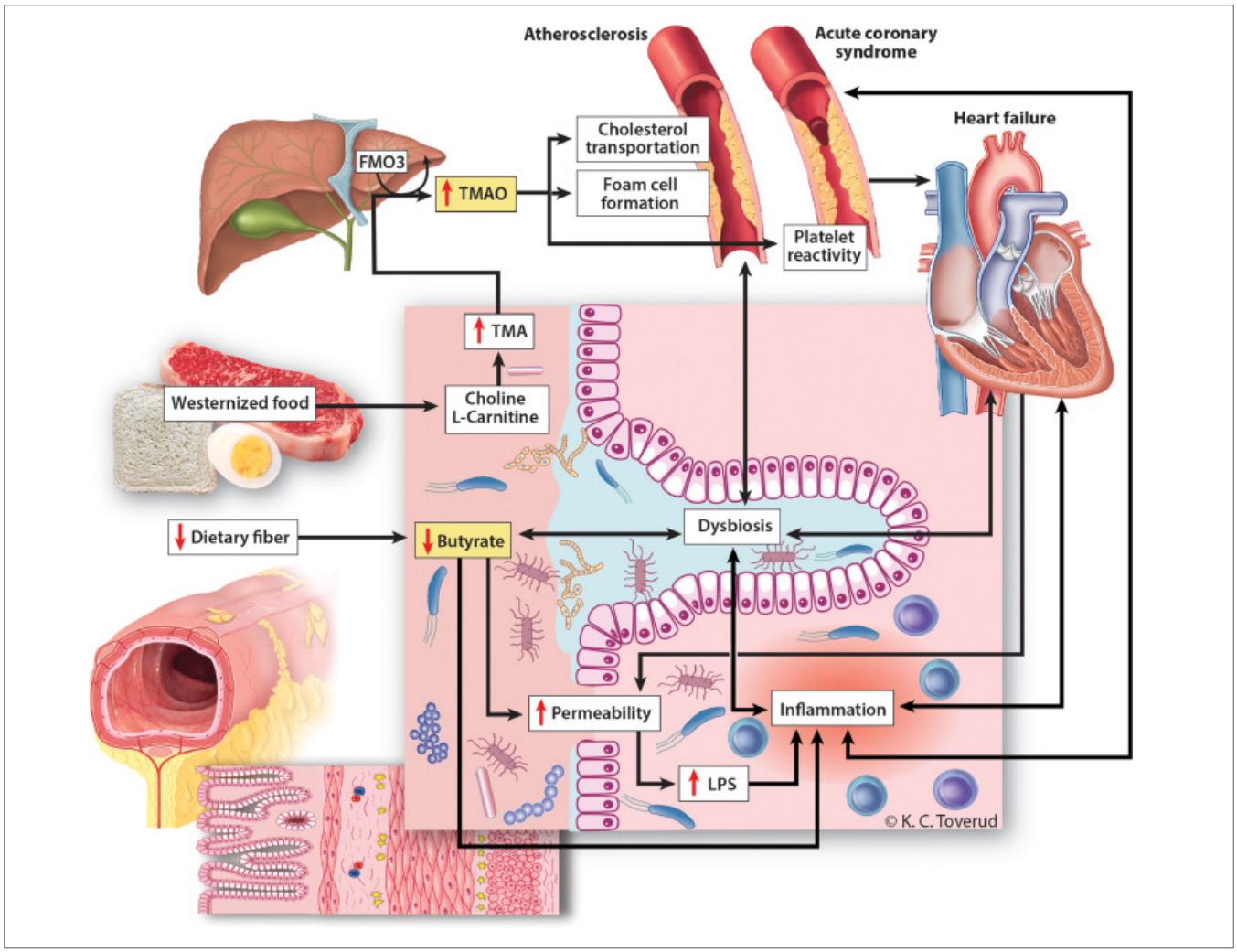

Fig 4: Diet-gut-heart interactions: proposed mechanisms[6].

CONCLUSION:

A holistic dietary approach to heart failure is the need of the day, with a focus on gut health and management of anemia. A diet restricted in sodium and free sugars aids in alleviating heart failure symptoms. Introduction of probiotics, fiber, iron supplementation and a Med-diet are protective and supportive in heart faiure.

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