Cracking the Cardio Code pt I

A Biogenetix Clinical Presentation

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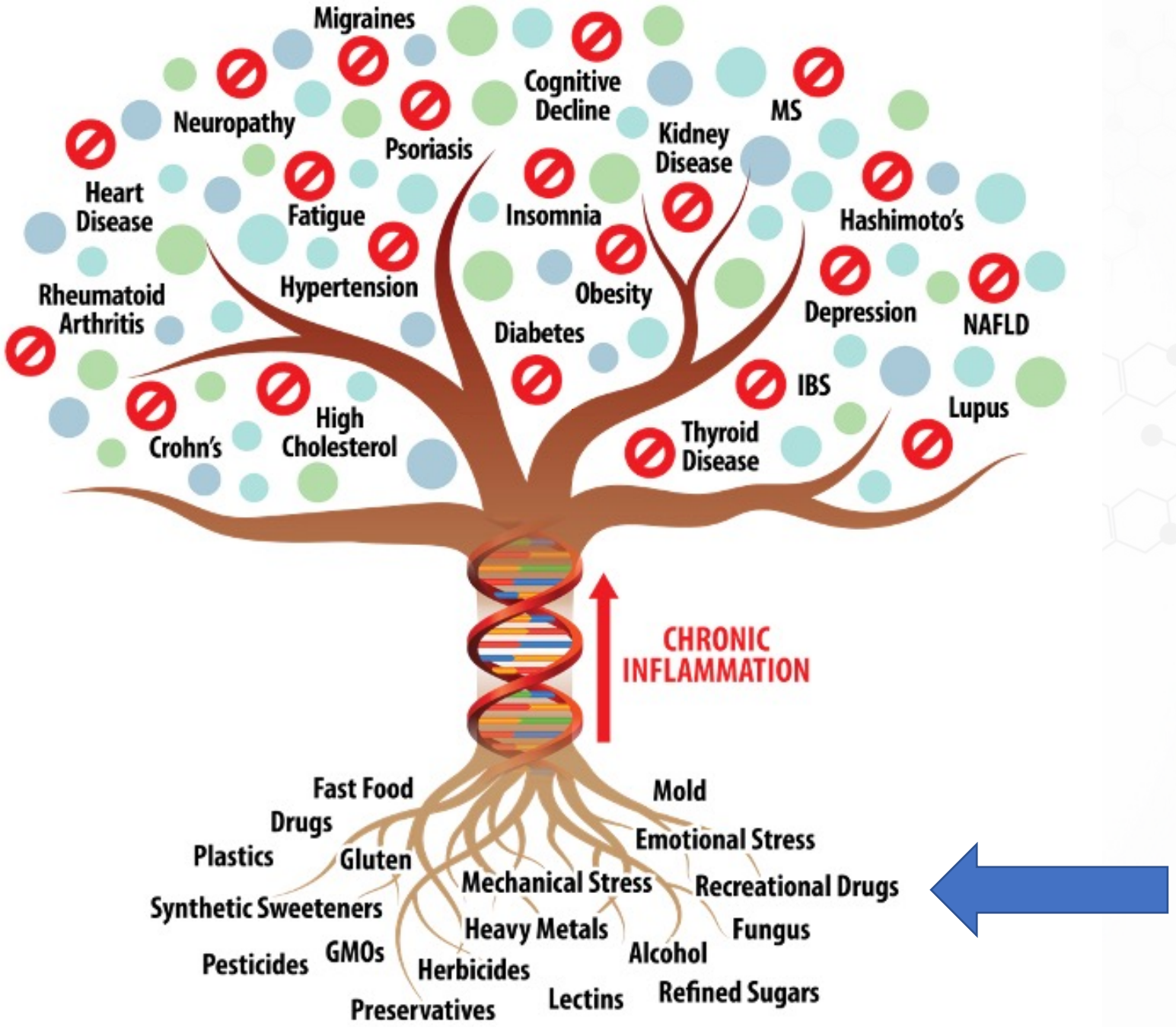
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*


Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
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GMOs
Alcohol
Herbicides
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National Heart, Lung, and Blood Institute

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HEART-HEALTHY LIVING

Understand Your Risk for Heart Disease

The first step toward heart health is understanding your risk of heart disease. Your risk depends on many factors, some of which are changeable and others that are not. Risk factors are conditions or habits that make a person more likely to develop a disease. These risk factors may be different for each person.

Preventing heart disease starts with knowing what your risks factors are and what you can do to lower them.

Risk factors for heart disease

- Have high blood pressure
- Have high blood cholesterol
- Have overweight or obesity
- Have prediabetes or diabetes
- Smoke
- Do not get regular physical activity
- Have a family history of early heart disease, for example if your father or brother was diagnosed before age 55, or your mother or sister was diagnosed before age 65
- Have a history of preeclampsia, which is a sudden rise in blood pressure and too much protein in the urine during pregnancy
- Have unhealthy eating behaviors
- Are age 55 or older for women or age 45 or older for men

Each risk factor increases your chance of developing heart disease. The more risks you have, the higher your overall risk.

Some risk factors cannot be changed. These include your age, sex, and a family history of early heart disease. Many others can be modified. For example, being more physically active and eating healthy are important steps for your heart health. You can make the changes gradually, one at a time. But making them is very important.

My doctor says it’s genetic…nothin’ you can do.

ESC Heart Fail. 2018 Jun; 5(3): 211–217.
Published online 2018 Feb 19. doi: 10.1002/ehf2.12267
PMCID: PMC5933969
PMID: 29457878

Genetic determinants of heart failure: facts and numbers

Frauke S. Czepluch, [1] Bernd Wollnik, [2] and Gerd Hasenfuß [1]

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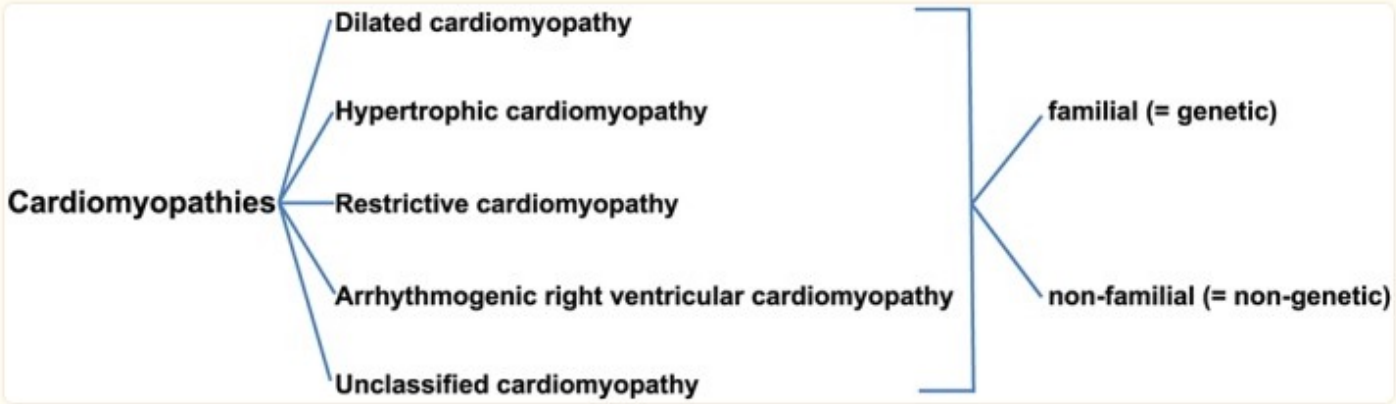

Figure 1

Classification of cardiomyopathies. Cardiomyopathies can be classified into five groups according to different morphological and functional criteria. Figure adapted from Elliot *et al.*[12]

Genetic cardiomyopathies and their associated genes

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In the last decades, nearly 100 genes whose mutations cause different forms of cardiomyopathies have been identified.[17] Most of these genes are associated with DCM and HCM, fewer with RCM and ARVC. Selected common genes whose mutations can cause cardiomyopathies are also shown in *Table* 1.

Table 1

Selected genes associated with cardiomyopathies

Cardiomyopathy form	Gene	Chromosome	Protein name
Dilated cardiomyopathy	*LMNA*	1	Lamin A/C
	MYH7	14	Beta myosin heavy chain
	TTN	2	Titin
	TNNT2	1	Troponin T
Hypertrophic cardiomyopathy	*MYBPC3*	11	Cardiac myosin binding protein C
	MYH7	14	Beta myosin heavy chain
	TNNI3	19	Troponin I
	TNNT2	1	Troponin T
	TPM1	15	Alpha-tropomyosin
	MYL3	3	Myosin light chain 3
Restrictive cardiomyopathy	*DES*	2	Desmin
	MYH7	14	Beta myosin heavy chain
	TNNI3	19	Troponin I
Arrhythmogenic right ventricular cardiomyopathy	*DSC2*	18	Desmocollin
	DSG2	18	Desmoglein 2
	DSP	6	Desmoplakin
	JUP	17	Plakoglobin
	PKP2	12	Plakophilin 2

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Prevention and treatment of heart failure by identifying its genetic (and environmental) determinants is of high importance. On the one hand, heart failure can be caused by mutations in different disease-associated genes. On the other hand, a complex interaction between genetic and environmental factors can also trigger heart failure. A clear genetic diagnosis can positively influence patient treatment and, thereby, improve prognosis. Furthermore, comprehensive genetic testing facilitates early identification of additional family members at risk for heart failure. Besides, understanding the pathogenesis of genetically induced heart failure at its molecular level may lead to the development of specific individual heart failure therapies in the future.

Am J Clin Nutr. 2010 Mar; 91(3): 502–509.
Published online 2010 Jan 20. doi: 10.3945/ajcn.2008.26285
PMCID: PMC2824150
PMID: 20089734

Saturated fat, carbohydrate, and cardiovascular disease[1,2,3,4]

Patty W Siri-Tarino, Qi Sun, Frank B Hu, and Ronald M Krauss

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A focus of dietary recommendations for cardiovascular disease (CVD) prevention and treatment has been a reduction in saturated fat intake, primarily as a means of lowering LDL-cholesterol concentrations. However, the evidence that supports a reduction in saturated fat intake must be evaluated in the context of replacement by other macronutrients. Clinical trials that replaced saturated fat with polyunsaturated fat have generally shown a reduction in CVD events, although several studies showed no effects. An independent association of saturated fat intake with CVD risk has not been consistently shown in prospective epidemiologic studies, although some have provided evidence of an increased risk in young individuals and in women. Replacement of saturated fat by polyunsaturated or monounsaturated fat lowers both LDL and HDL cholesterol. However, replacement with a higher carbohydrate intake, particularly refined carbohydrate, can exacerbate the atherogenic dyslipidemia associated with insulin resistance and obesity that includes increased triglycerides, small LDL particles, and reduced HDL cholesterol. In summary, although substitution of dietary polyunsaturated fat for saturated fat has been shown to lower CVD risk, there are few epidemiologic or clinical trial data to support a benefit of replacing saturated fat with carbohydrate. Furthermore, particularly given the differential effects of dietary saturated fats and carbohydrates on concentrations of larger and smaller LDL particles, respectively, dietary efforts to improve the increasing burden of CVD risk associated with atherogenic dyslipidemia should primarily emphasize the limitation of refined carbohydrate intakes and a reduction in excess adiposity.

Editorial > Br J Sports Med. 2017 Aug;51(15):1111-1112. doi: 10.1136/bjsports-2016-097285. Epub 2017 Apr 25.

Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced from healthy lifestyle interventions

Aseem Malhotra [1], Rita F Redberg [2 3], Pascal Meier [4 5]

Affiliations + expand

PMID: 28442474 DOI: 10.1136/bjsports-2016-097285

Curr Opin Endocrinol Diabetes Obes. 2023 Feb; 30(1): 65–71.
Published online 2022 Dec 8. doi: 10.1097/MED.0000000000000791
PMCID: PMC9794145
PMID: 36477384

A short history of saturated fat: the making and unmaking of a scientific consensus

Nina Teicholz

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The idea that saturated fats cause heart disease, called the diet-heart hypothesis, was introduced in the 1950s, based on weak, associational evidence. Subsequent clinical trials attempting to substantiate this hypothesis could never establish a causal link. However, these clinical-trial data were largely ignored for decades, until journalists brought them to light about a decade ago. Subsequent reexaminations of this evidence by nutrition experts have now been published in >20 review papers, which have largely concluded that saturated fats have no effect on cardiovascular disease, cardiovascular mortality or total mortality. The current challenge is for this new consensus on saturated fats to be recognized by policy makers, who, in the United States, have shown marked resistance to the introduction of the new evidence. In the case of the 2020 Dietary Guidelines, experts have been found even to deny their own evidence. The global re-evaluation of saturated fats that has occurred over the past decade implies that caps on these fats are not warranted and should no longer be part of national dietary guidelines. Conflicts of interest and longstanding biases stand in the way of updating dietary policy to reflect the current evidence.

As a clinician…

- What are we trying to accomplish?
- What variables is the patient willing to control?
- What's already been wrecked or compromised?
- What's the literature say?
    1. Ancestry
    2. Comorbidities
    3. Regional concerns
- Then put together a way forward...

Adv Nutr. 2019 Jul; 10(4): 634–646.
Published online 2019 Apr 30. doi: 10.1093/advances/nmz007
PMCID: PMC6628854
PMID: 31041449

Effects of a Paleolithic Diet on Cardiovascular Disease Risk Factors: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

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Conclusion

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Based on our analyses, a PD decreased the anthropometric indexes (weight, BMI, WC, and body fat percentage), blood pressure (SBP and DBP), and circulating CRP concentrations, and improved the lipid profile (LDL cholesterol, TGs, and TC; HDL cholesterol increased). However, we have insufficient evidence to make solid conclusions regarding the efficacy of a PD on improving CVD risk factors, mostly owing to a lack of qualified RCTs. Thus, putative long-term useful effects of different components of a PD on CVD risk factors need to be explored in additional well-designed large trials.

Eur J Nutr. 2022; 61(2): 957–971.
Published online 2021 Oct 21. doi: 10.1007/s00394-021-02696-9
PMCID: PMC8854325
PMID: 34671828

A score appraising Paleolithic diet and the risk of cardiovascular disease in a Mediterranean prospective cohort

Víctor de la O,[1,4] Itziar Zazpe,[1,2,3,4] Leticia Goni,[1,3,4] Susana Santiago,[2,4] Nerea Martín-Calvo,[1,3,4] Maira Bes-Rastrollo,[1,3,4] J. Alfredo Martínez,[2,3,4,5] Miguel Á. Martínez-González,[1,3,4,6] and Miguel Ruiz-Canela[✉1,3,4]

Cardiovascular disease (CVD) remains the most common cause of death, accounting for 2.2 million deaths in females and 1.9 million deaths in males during 2019 in Europe, and 31% of all deaths globally [1, 2]. Moreover, CVD incidence is globally increasing due to population growth, rural-to-urban migration and the aging of the world's population [3]. CVD-related morbidity is associated with a gradual decrease in the quality of life and higher economic cost [4, 5], and it accounts for 24% of non-communicable diseases related disability-adjusted life years globally [5]. The promotion of healthy lifestyles to prevent CVD is an urgent need to reduce the current burden of this public health concern [6].

The promotion of healthy diets is probably one of the most cost-effective strategies to prevent CVD. Several dietary patterns have been proposed based on the cardioprotective effect of their components [7]. The Mediterranean Diet (MedDiet), the Alternative Healthy Eating Index or the Dietary Approaches to Stop Hypertension are well-known dietary patterns. They represent a priori defined high-quality diet scores inversely associated with the risk of CVD [8]. A common characteristic of these dietary patterns is a high consumption of fruits and vegetables, whole grains, nuts, legumes, vegetable oils, fish, and seafood; a moderate consumption of low-fat dairy products; and a low consumption of processed meat, sugar-sweetened beverages, and sodium [9].

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A score appraising Paleolithic diet and the risk of cardiovascular disease in a Mediterranean p

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Anthropological studies have hypothesized that hunter-gatherers had a slim build, and they were fit and free of chronic diseases such as CVD due to their diet [15, 16]. From a public health perspective, the challenge is to know the potential health benefits of a Paleo-style dietary pattern adapted to current lifestyles and food availability. Currently, the PaleoDiet is characterized by a high consumption of fruits, vegetables, tree nuts, eggs, fish, and unprocessed meats, and a low consumption of dairy products, cereals and grains, legumes, processed foods, and culinary ingredients (added salt, sugar and refined fats) [17]. However, there is a diverse interpretation about the PaleoDiet at the popular levels and these Paleo-style diets may have no clear definitions and there is scarce scientific evidence to promote them [17]. Some recent reviews have suggested an inverse association between the PaleoDiet and cardiovascular risk factors [17, 18]. However, there is scarce evidence on the long-term beneficial effect of the PaleoDiet on hard end-points of the most prevalent chronic diseases such as CVD. To the best of our knowledge, only two studies with large sample size have suggested potential benefits of the PaleoDiet on CVD mortality [19, 20].

In this study we aimed to assess the association between a score appraising a PaleoDiet pattern and the risk of CVD in a well-known Spanish cohort. Knowing the association between this dietary pattern and CVD risk in a country with a relatively high adherence to the MedDiet is of interest. For this reason, we also explored the relationship between the MedDiet and PaleoDiet. While the MedDiet emphasizes a high consumption of all plant-based foods, the PaleoDiet limits the intake of legumes, cereals and grains, and recommends higher intake of unprocessed meats. Our hypothesis was that the PaleoDiet could reduce CVD risk due to the high consumption of fruits, vegetables, tree nuts, eggs, fish, unprocessed meats, and the exclusion of ultra-processed foods. In addition, we hypothesized that the recommendation to reduce the consumption of whole grains and legumes within the PaleoDiet score might mitigate the inverse association between this diet and CVD risk.

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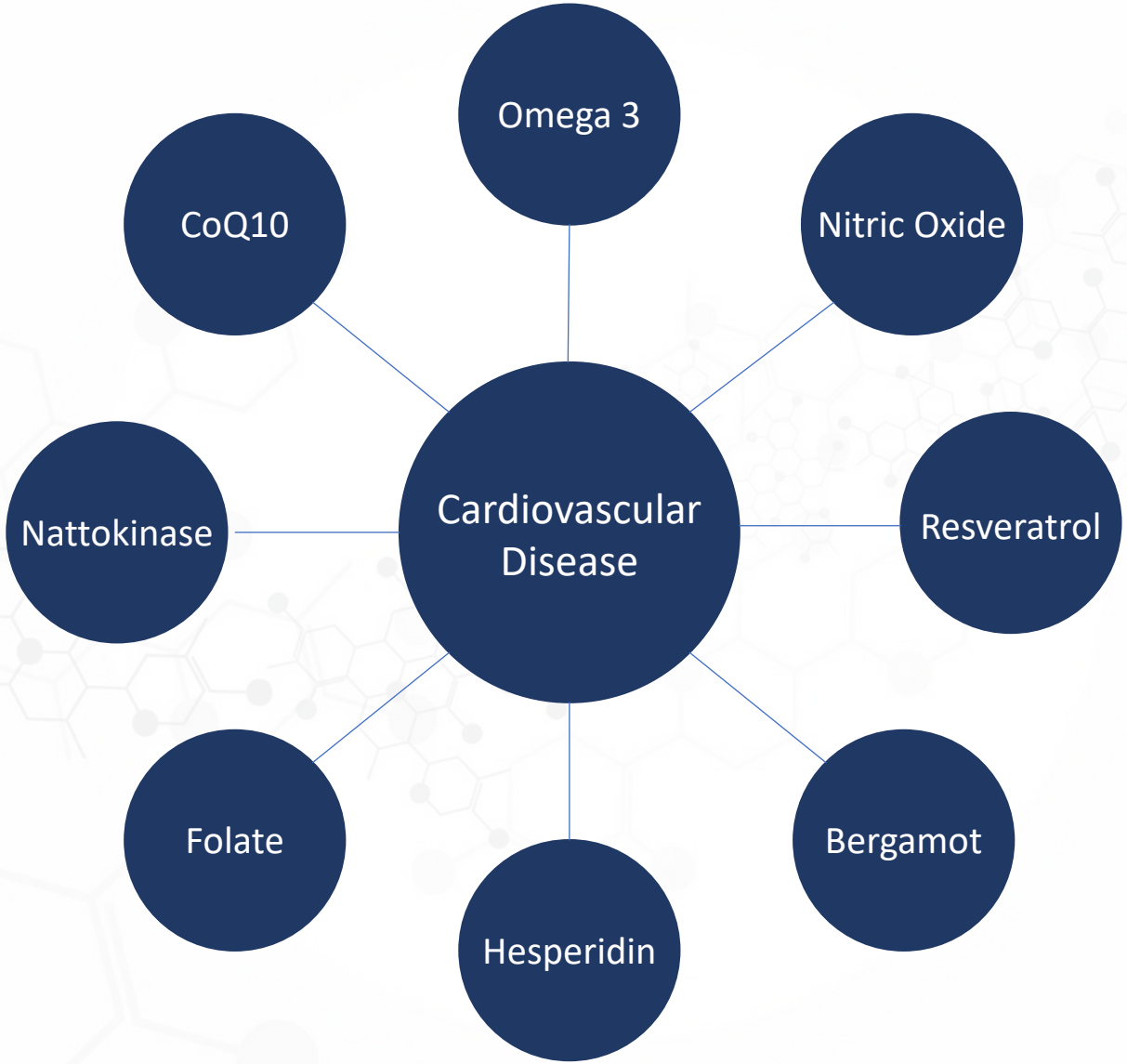
This prospective Mediterranean cohort study, conducted among young adult participants, showed an inverse association between higher adherence to the PaleoDiet and CVD risk. Among participants with the highest PaleoDiet score (upper quintile), a 55% relative CVD risk reduction was observed as compared to participants in the lowest quintile. This association was not significantly modified by sex, weight status or physical activity, and the results were robust in multiple sensitivity analyses aimed at controlling for residual confounding. A similar association was also observed when we alternatively excluded items one by one from the PaleoDiet score, although the consumption of fruits and vegetables, and the avoidance of ultra-processed foods may be key components of this diet. However, weaker inverse associations were also found when nuts or fish were excluded, suggesting the synergistic effect of all components within the PaleoDiet score. However, the prohibition of grains and cereals should be further explored since a stronger inverse association was found when this limitation was not part of the PaleoDiet score. Finally, in the joint analysis according to levels of adherence to PaleoDiet and MedDiet, the strongest inverse association with CVD was found among participants with the highest adherence to both dietary patterns.


Omega 3
CoQ10
Nitric Oxide
Nattokinase
Cardiovascular Disease
Resveratrol
Folate
Bergamot
Hesperidin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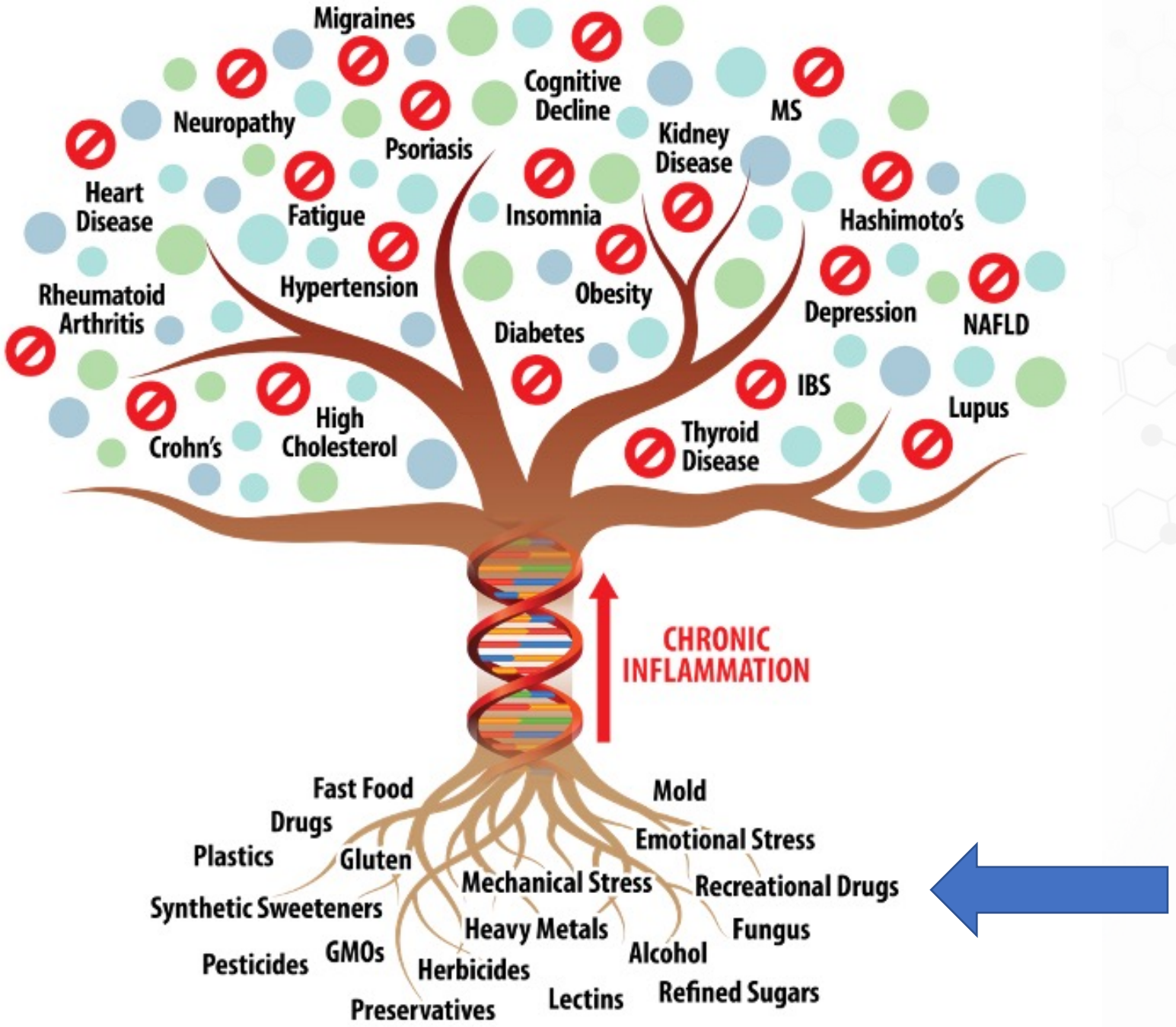
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