Cracking the Cardio Code pt IV

A Biogenetix Clinical Presentation

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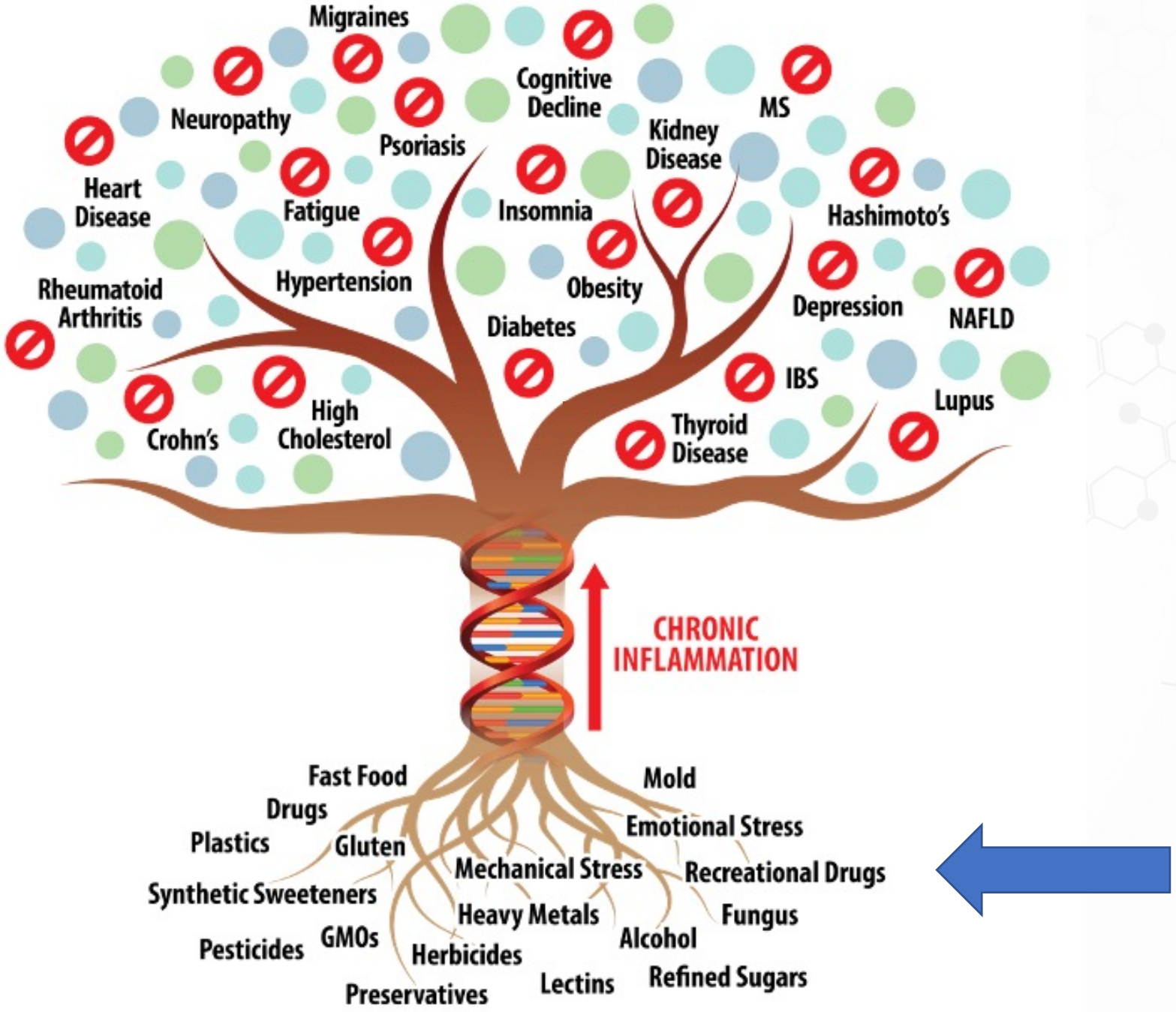
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*


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

Heart disease in the United States

In the United States:

- Heart disease is the **leading cause of death** for men, women, and people of most racial and ethnic groups. [1]
- One person dies **every 33 seconds** from cardiovascular disease. [1]
- About 695,000 people died from heart disease in 2021—that's **1 in every 5 deaths**. [1] [2]
- Heart disease costs about **$239.9 billion** each year from 2018 to 2019. [3] This includes the cost of health care services, medicines, and lost productivity due to death.

Coronary artery disease (CAD)

- Coronary heart disease is the most common type of heart disease, killing 375,476 people in 2021. [2]
- About **1 in 20 adults** age 20 and older have CAD (about 5%). [2]
- In 2021, about 2 in 10 deaths from CAD happened in adults less than 65 years old. [1]

Heart attack

- In the United States, someone has a heart attack every 40 seconds. [2]
- Every year, about **805,000 people** in the United States have a heart attack. [2] Of these,
  - 605,000 are a first heart attack. [2]
  - 200,000 happen to people who have already had a heart attack. [2]
- About **1 in 5** heart attacks are silent—the damage is done, but the person is not aware of it. [2]

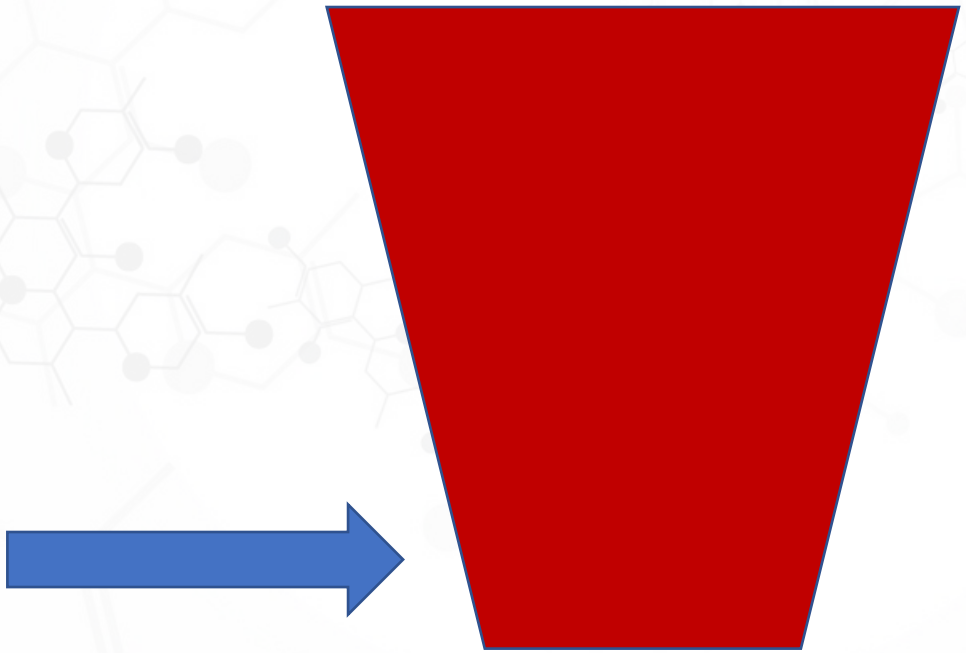
My doctor says it’s genetic…nothin’ you can do.

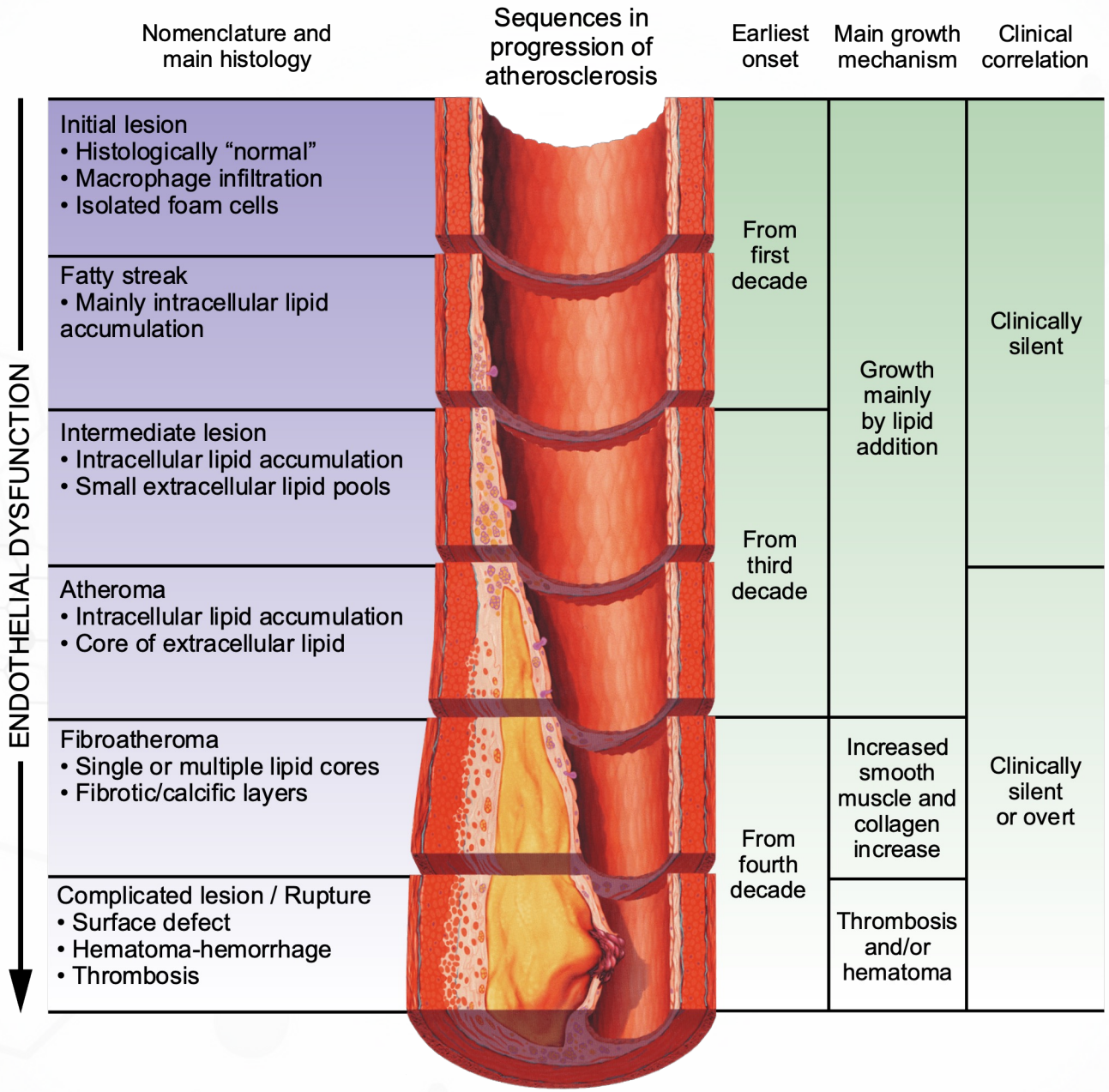
“It’s genetic.”

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...

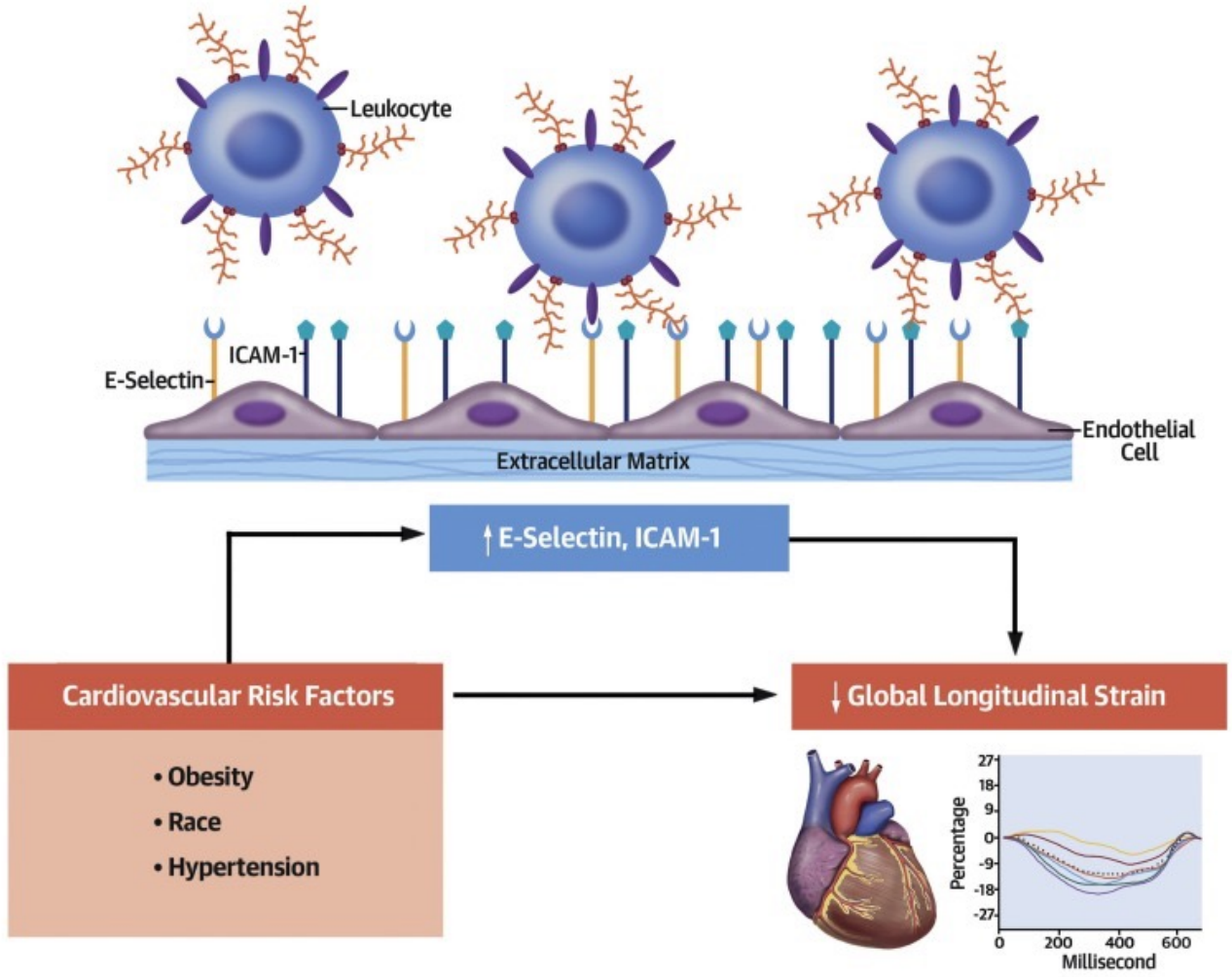
The Bucket Theory






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

CENTRAL ILLUSTRATION: Cardiovascular Risk Factors, Biomarkers of Endothelial Activation, and Cardiac Function



Patel, R.B. et al. J Am Coll Cardiol. 2020;75(17):2156-65.

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PMCID: PMC10663934
PMID: 38028000

Citrus flavonoids and adhesion molecules: Potential role in the management of atherosclerosis

Farnaz Ebrahimi,[a,b] Mohammad Mahdi Ghazimoradi,[c] Ghizal Fatima,[d] and Roodabeh Bahramsoltani[e,*]

1.2. Role of adhesion molecules in atherosclerosis

Adhesion molecules are a large group of mediators responsible for cellular interactions and their dysregulation is involved in the pathogenesis of several human diseases including gestational diabetes [7], fibrotic diseases [8], malignancies [9], autoimmune diseases [10], and cardiovascular disorders [11]. It has been demonstrated that upregulation of different adhesion molecules stimulates a cascade of several inflammatory processes and, if not being controlled properly, causes a chronic inflammatory status [10]. In other words, dysregulation of any of the categories of adhesion molecules results in specific inflammatory and immune-related disorders which can affect different tissues/organs and as mentioned above, has various clinical manifestations.

In the earlier stages of atherosclerosis, migration of monocytes through EC is associated with adhesion molecules present on the vascular endothelial cells. These important protein molecules are involved in the sticking of leukocytes to the surface of EC. Generally, adhesion molecules have five main families, i.e., immunoglobulin superfamily, selectins, integrins, cadherins, and others [8]. Three main groups of adhesion molecules in the plaque structure are integrins (β_1, β_2 Integrin), members of immunoglobulins superfamily including intercellular adhesion molecule 1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), and selectins (P, E, or L selectin) [12,13].

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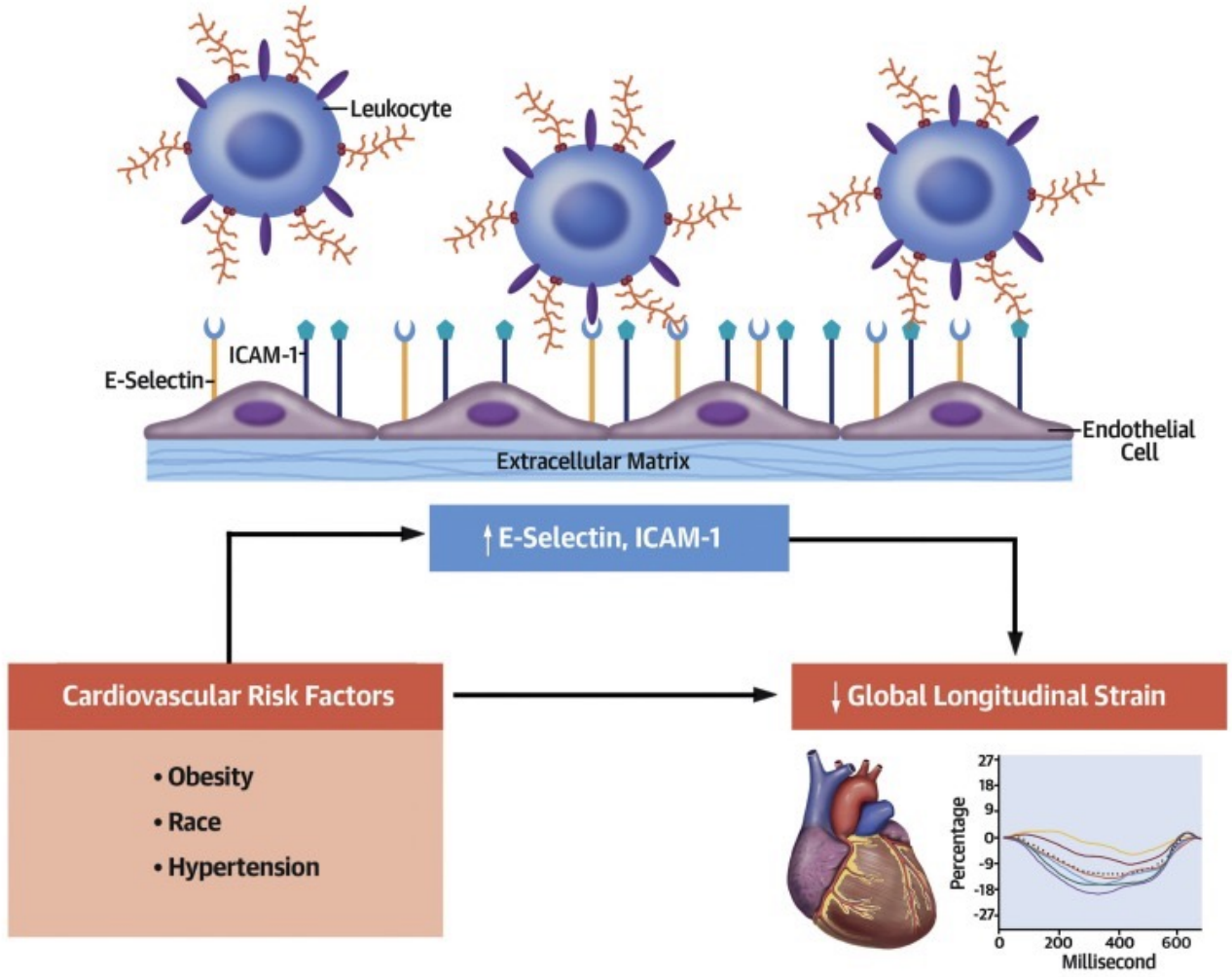
Expression of adhesion molecules due to ox-LDL-C accumulation occurs in EC. First of all, selectins cause rolling and tethering of leukocytes. Then, firm attachments take place with ICAM and VCAM expression and as a result, leukocytes enter the intima. Identifying and evaluating the expression of these molecules in atherosclerotic lesions in different studies is used as an indicator of the effectiveness of various anti-thrombotic drugs [5,21].

CENTRAL ILLUSTRATION: Cardiovascular Risk Factors, Biomarkers of Endothelial Activation, and Cardiac Function



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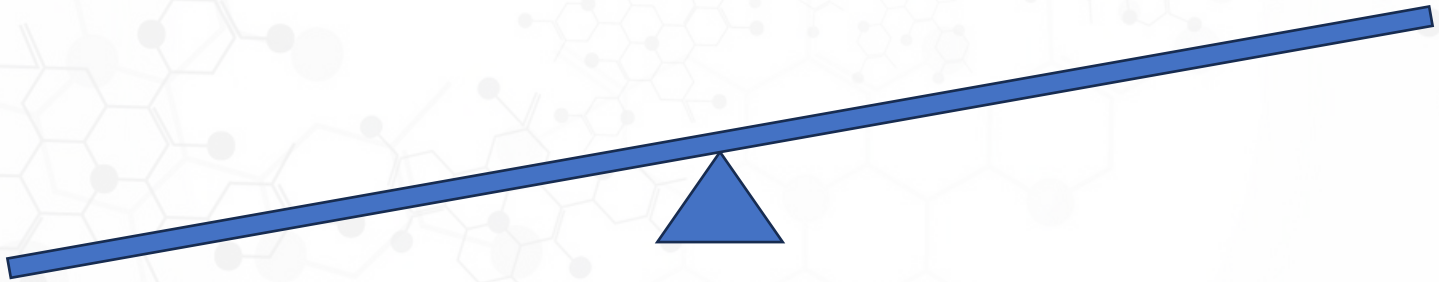
Risk & Inflammation (adhesion molecule expression)



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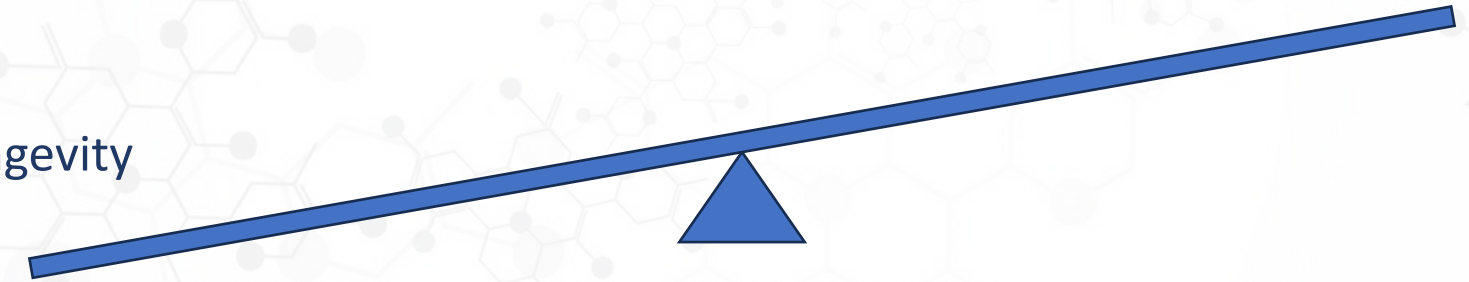

Longevity

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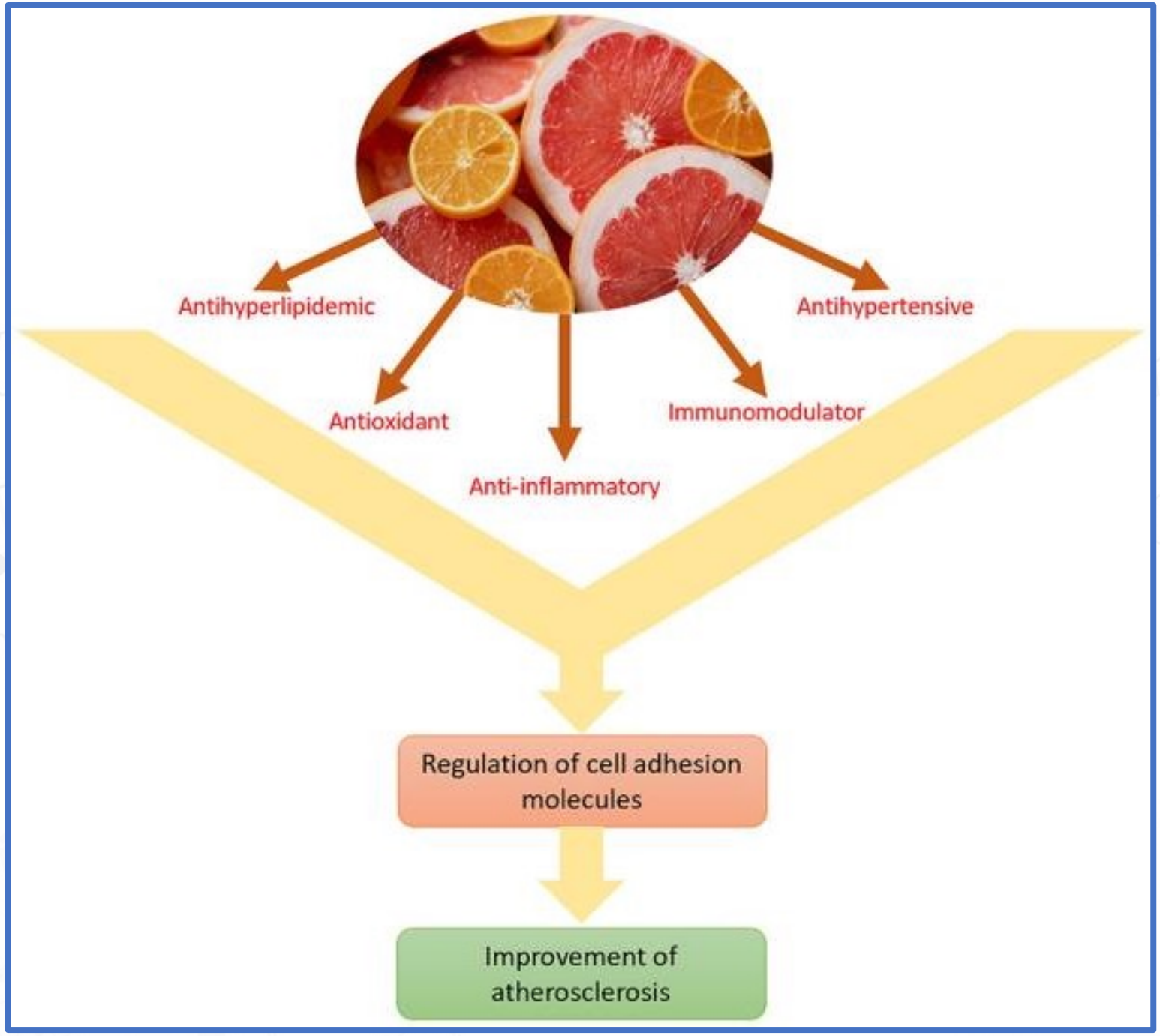
Farnaz Ebrahimi,[a,b] Mohammad Mahdi Ghazimoradi,[c] Ghizal Fatima,[d] and Roodabeh Bahramsoltani[e,*]

Atherosclerosis as a chronic inflammatory disorder is accompanied with oxidative stress which causes a high morbidity and mortality. Adhesion molecules, including intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), P-selectin, and E-selectin, are amongst the most important contributors in atherosclerosis. In such cases, dietary interventions with functional foods containing natural antioxidant and anti-inflammatory constituents are of a great interest. Citrus fruits are rich sources of flavonoids as natural pigments with potent antioxidant and anti-inflammatory activities. This study aims to review current evidence regarding the role of citrus flavonoids in the management of atherosclerosis with a focus on their effect on adhesion molecules. Electronic databases including PubMed, Scopus, and Web of Science were searched with the names of adhesion molecules and flavonoids from inception until January 2023. The included articles highly support the beneficial effects of citrus flavonoids in preclinical models of atherosclerosis. Quercetin, naringin and naringenin, hesperidin and hesperetin, nobiletin, rutin, luteolin, apigenin, and kaempferol are the most common flavonoids in citrus fruits which could modulate adhesion molecules including ICAM-1, VCAM-1, E-selectin, and P-selectin. Additionally, markers of chronic inflammation such as interleukins, tumor necrosis factor-α, nuclear factor-κB, and nitric oxide signaling, as well as oxidative stress markers like superoxide dismutase and glutathione were all normalized upon administration of citrus flavonoids. Conclusively, this review confirms the modulatory role of flavonoids on adhesion molecules in atherosclerosis based on the preclinical evaluations. Thus, citrus fruits can be further studied in atherosclerotic patients regarding their activity in reducing adhesion molecules.


Antihyperlipidemic
Antihypertensive
Antioxidant
Immunomodulator
Anti-inflammatory
Regulation of cell adhesion molecules
Improvement of atherosclerosis

Front Nutr. 2023; 10: 1177708.
Published online 2023 Jul 12. doi: 10.3389/fnut.2023.1177708
PMCID: PMC10369082
PMID: 37502716

The effects of hesperidin supplementation on cardiovascular risk factors in adults: a systematic review and dose–response meta-analysis

Atie Sadat Khorasanian, [1] Sahand Tehrani Fateh, [2] Fatemeh Gholami, [3] Niloufar Rasaei, [3] Hadis Gerami, [4,5] Sayyed Saeid Khayyatzadeh, [4,6] Farideh Shiraseb,[✉3,*] and Omid Asbaghi[✉7,8,*]

Hesperidin is a naturally occurring bioactive compound that may have an impact on cardiovascular disease risks, but the evidence is not conclusive. To investigate further, this study aimed to explore the effects of hesperidin supplementation on cardiovascular risk factors in adults. A comprehensive search was conducted up to August 2022 using relevant keywords in databases such as Scopus, PubMed, Embase, Cochrane Library, and ISI Web of Science for all randomized controlled trials (RCTs). The results showed that hesperidin supplementation had a significant effect on reducing serum triglyceride (TG), total cholesterol (TC), low-density cholesterol (LDL), tumor necrosis factor-alpha (TNF-α), and systolic blood pressure (SBP), whereas weight was increased. However, no significant effect was observed on high-density cholesterol (HDL), waist circumference (WC), fasting blood glucose (FBG), insulin, homeostatic model assessment for insulin resistance (HOMA-IR), C-reactive protein (CRP), interleukin-6 (IL-6), body mass index (BMI), and diastolic blood pressure (DBP). The study also found that an effective dosage of hesperidin supplementation was around 1,000 mg/d, and a more effective duration of supplementation was more than eight weeks to decrease insulin levels. Furthermore, the duration of intervention of more than six weeks was effective in decreasing FBG levels.

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PMCID: PMC10937595
PMID: 38486739

4.2. Cardiovascular diseases (CVD)

Following myocardial infarction (MI), collagen-producing myofibroblasts are activated, contributing to the gradual development of replacement fibrosis. Myocardial fibrosis undoubtedly affects left ventricular remodeling. Myocardial fibroblast activation is triggered by myocardial fiber stretching and inflammation. Myocardial fibrosis is regulated by inflammation, various cell types, paracrine mechanisms (such as TGFs) and collagen-degrading enzymes (such as MMPs) [97].

Hesperidin inhibits both caspase-3 and myeloperoxidase and smooth muscle actin alpha (α-SMA) and MMP-2 play crucial roles in preventing cardiac dysfunction and myocardial remodeling following MI by inhibiting collagen deposition and fibroblast migration [98]. Hesperidin and hesperetin effectively prevented hypertension and cardiac remodeling in a rat model of N(ω)-nitro-L-arginine methyl ester (L-NAME)-induced hypertension, as evidenced by the reduction in left ventricular wall thickness, cross-sectional area (CSA), fibrosis, vascular remodeling and expression of transforming growth factor β1 (TGF-β1) and TNF-1 proteins [99].

Hesperidin upregulates serum and hepatic SOD and GSH-Px in low-density lipoprotein (LDL) receptor-deficient ($LDLR^{-/-}$) mice fed a high-fat diet, which suggests an increased endogenous defense against oxidative stress [100]. Furthermore, hesperidin-treated animals exhibited less severe atherosclerosis than their control counterparts, as hesperidin-treated animals had lower serum oxidized (OX)-LDL, IL-6 and TNF levels than controls [100].

Therefore, hesperidin and hesperetin safeguards cardiovascular health by exhibiting anti-inflammatory and antioxidant properties, as well as other pharmacological effects.


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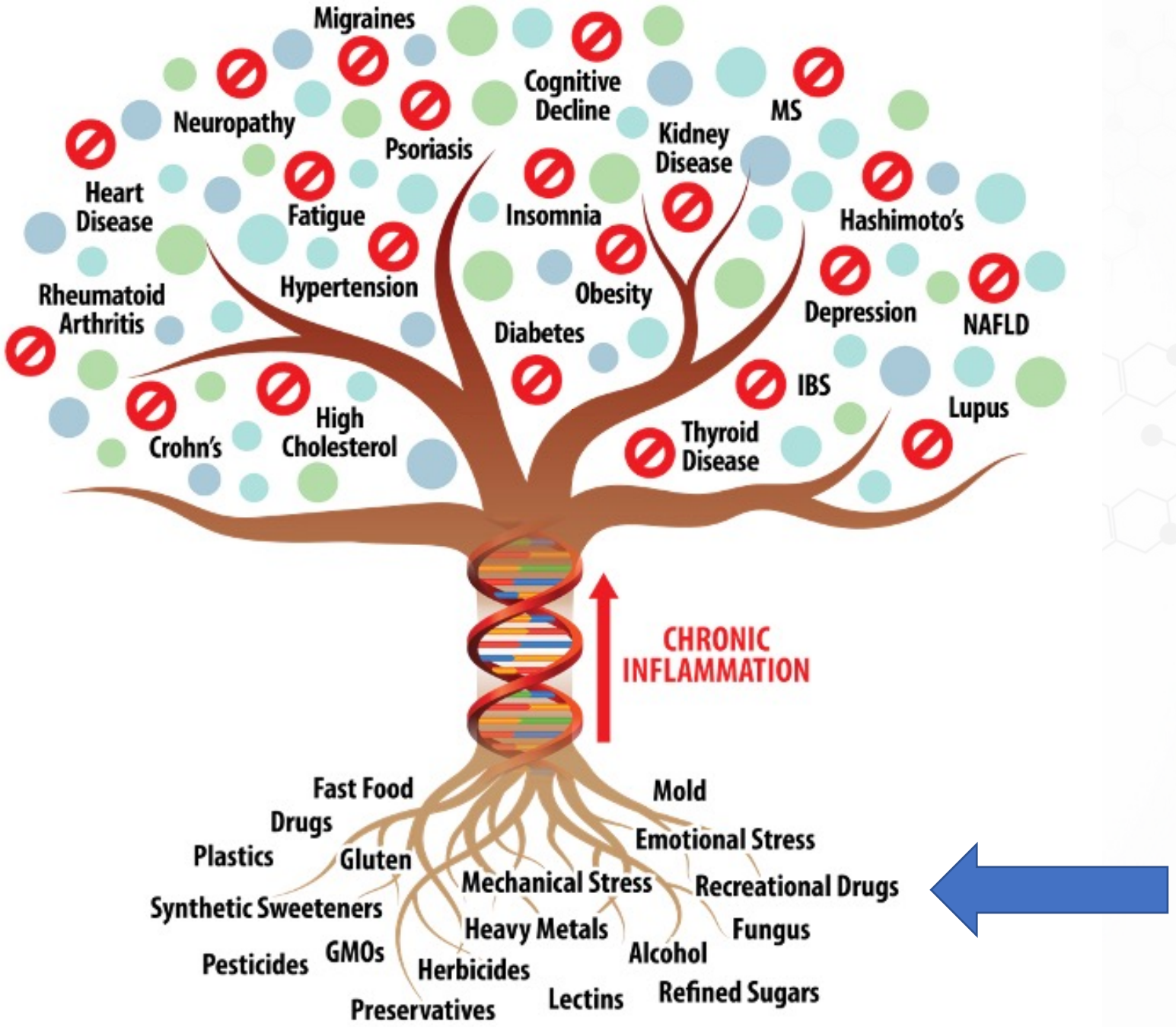
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