Sucrose and Fructose Effects on Atherogenesis: A Systematic Review

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**Proposed Project Title:** Sucrose and Fructose Effects on Atherogenesis: A Systematic Review

**Purpose:** The aim of this systematic review is to examine and describe evidence from diet-related interventional research to establish whether a relationship exists between sucrose and fructose consumption and atherosclerotic heart disease in the United States.

**Background:** “Diseases of the heart” is the leading cause of death in the United States (CDC, 2022). Of the many afflictions encompassed in this umbrella category, ischemic heart disease occupies the majority of cases. Ischemic heart disease – also known as coronary heart disease or coronary artery disease – is caused by plaque formation inside the lumen of the vessels supplying the heart. This makes it difficult for the heart to receive oxygen efficiently and puts the patient at risk for a heart attack – or myocardial infarction – caused by poor perfusion to the heart itself. Atherosclerosis is the term for such plaques, and atherogenesis is affected by multiple factors.

Atherogenesis is worst when it affects the coronary arteries, but it can occur anywhere in the vascular system, leading to heart attacks, clots, strokes, aneurysms, hypertension, cerebrovascular disease, peripheral artery disease, and general weakening of the vasculature. Though the cellular mechanisms are not fully understood, it is thought to be caused in large part by inflammation. The inflammation cascade causes leukocytes to adhere to low-density lipoprotein (LDL) molecules in the blood and attach to the cell wall when under longstanding vascular stress (Libby, 2000). When arteries experience shear force from episodes of hypertension or inflammatory damage, the normal response of the body would be to repair the microfractures or consume the foreign particle. When this process is initiated in the presence of oxidative forces, though, the LDL molecules that have been kept in the bloodstream by the normal proteoglycan proteins and monocytes oxidize and stick to the endothelial lining to form plaques. Because this proposed action depends so highly on oxidation and adhesion, consumption of certain foods that alter these pathways could affect atherogenesis. In the American diet, sugar is one powerful ingredient that predisposes a patient to inflammation, oxidative stress, and increased platelet adhesion (Libby, 2000).

**Project Goals:** In this review, I am motivated by the pressing nature of the cardiovascular health epidemic and the modifiable factors that could affect patient outcomes and general wellness. I seek to analyze the evidence surrounding dietary factors that affect atherogenesis. Foods that encourage inflammation or contribute to insulin resistance – like refined sucrose or high-fructose corn syrups – are thought to contribute to atherosclerotic plaque formation via inflammatory oxidation reactions of LDL in the vascular space. If there is sufficient evidence to support this claim, it will likely shift the current understanding of the disease process from the current emphasis on fatty acids. Though lipid intake can be altered to prevent some atherosclerosis, high sugar intake remains one of the strongest predictors of coronary heart disease (Howard & Wylie-Rosett, 2002). Nursing education regarding diet would require a shift in emphasis to control blood sugar nonpharmacologically.

To explore whether there is evidence in favor of low sugar diet-related changes to treat the typical atherosclerotic heart diseases, I will use the following question to focus my review of the literature: Are patients with high-sugar and fructose consumption as measured by diet reports, serum glucose, serum insulin, and inflammatory markers at an increased risk for atherosclerotic disease compared to patients with low-sugar diets? I will use relevant databases such as PubMed and Cochrane to structure a search for research articles. Using Medical Subject Headings (MeSH), I will create an algorithm that is reproducible to refine the articles to be used in the review, filtering for articles published in the past ten years that are meta-analyses or randomized-control trials. Once the evidence is compiled, I will condense the findings in an organized fashion and determine clinical implications for patient education.
Proposed Project Schedule:

1. Conduct background research on topic, identify gap in review literature (01/01/2023 – 02/01/2023)
2. Contact five most relevant journals for publication interest (02/01/2023 – 02/14/2023)
3. Conduct research on refined topic (05/01/2023 – 07/08/2023)

Cumulative GPA: 

References

