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Review about relation between white sugar and retinopathy

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Abstract

The surging prevalence of vision impairment is increasingly linked to the high intake of refined white sugar, particularly in its forms as fructose and added sweeteners. Retinopathy is a condition that is often a result of this dietary pattern, and is an important factor that initiates and promotes the progression of retinopathy. The biochemical mechanisms by which excess sugar consumption leads to retinal damage are explored, focusing on the processes of inflammation, hyperglycemia-induced oxidative stress, and the negative effects of advanced glycation end products (AGEs). Current available experimental and epidemiological knowledge are combined and discussed in terms of clinical implications for prevention and clinical management. In conclusion, the findings underscore the importance of strategic dietary choices in minimizing the risk and impact of retinal damage.

Keywords: Sucrose; Added sweeteners; Fructose; Retinal damage; Diabetic retinopathy; AGEs; Oxidative stress

1. Introduction

The worldwide increase in refined sugar consumption, particularly HFCS and sucrose (white sugar), is a reflection of the increase in metabolic syndrome, including retinopathy, as a complication of these syndromes. In addition to its effect of causing hyperglycemia and insulin resistance, there are several other mechanisms by which excessive white sugar consumption can cause harmful effects on the delicate retinal vasculature and neural tissue . This review examines the latest evidence for a causal link between white sugar intake and retinopathy, highlighting the pathways and implications of this association.

2. Proposed Causal Mechanisms

2.1. Hyperglycemia and the Polyol Pathway:

Ongoing excess sugar consumption in the diet leads to a chronic increase in blood sugar. In the cells of the retina, this excess of glucose is shunted into the polyol metabolic pathway where the enzyme aldose reductase converts it to sorbitol and consumes NADPH. These metabolic changes create substantial cellular osmotic imbalance due to the accumulation of sorbitol and lead to a depletion of essential cellular antioxidants, such as glutathione. This combination causes intracellular osmotic stress because of sorbitol accumulation and loss of antioxidants, such as glutathione, inside the cells, making retinal pericytes and endothelial cells susceptible to oxidative damage.

2.2. Inflammation and Oxidative Stress

A high fructose diet—one that contains a lot of fructose, an important part of white sugar—can be strong inflammatory agents and factors contributing to oxidative stress throughout the body. Fructose is metabolized in the liver to produce uric acid and reactive oxygen species (ROS). These circulating pro-inflammatory molecules have the ability to disrupt

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the blood-retinal barrier so that leukocytes can be recruited, vascular permeability is increased and inflammatory cytokines, including VEGF (Vascular Endothelial Growth Factor) are secreted. Also, notably, VEGF is a prime catalyst for the abnormal angiogenesis characteristic of proliferative retinopathy.

3. The importance of Advanced Glycation End-Products (AGEs)

A non-enzymatic glycation process of refined sugar molecules with lipids and proteins forms AGE molecules, notably fructose which is very reactive. These compounds tend to become deposited in the retinal vascular system and cause the cross-linking of collagen, make the capillary walls more rigid, as well as decrease the available nitric oxide, all of which will ultimately hinder the proper vasodilation. In addition, AGEs bind to their receptor (RAGE) on retinal cells, activating pro-inflammatory and pro-apoptotic pathways; thereby promoting cellular dysfunction.3 Scientific Evidence supporting the Association

3.1. Epidemiological Studies

Consistent with this, high consumption of sugars has been found to be significantly associated with the occurrence or worsening of retinopathy in large cohort studies. Data shows that sweetened drink consumption in the top 25% of drinkers is strongly associated with a significantly increased risk of retinal complications compared to the lowest quarter of drinkers. This connection is even seen after accounting for overall calorie consumption and body mass index.

3.2. Experimental Studies

Laboratory studies using animals have been important and have given insights, such as the fact that laboratory animals with low-activity, sucrose- or fructose-rich diets display the key pathological features of retinopathy. These include the breakdown of retinal capillaries, leakage of vessels, and an increase in inflammatory markers, even in non-diabetic individuals. Additionally, experiments have shown that concentrated levels of either glucose or fructose cause immediate distress to retinal endothelial cells in vitro. This is seen as increased membrane permeability, increased oxidative strain and accelerated programmed cell death (apoptosis).

4. Clinical Implications

4.1. Retinopathy Onset and Progression.Onset and progression of retinopathy.

High sugar intake is a great controllable risk factor which can accelerate the onset of retinopathy and worsen its clinical course. Studies show that people with poor glycemic control who eat a diet high in refined sugars are significantly more likely to be at risk for progression from non-proliferative to more dangerous, vision-threatening proliferative diabetic retinopathy.

4.2. Synergistic Effects with Hypertension

Sugar mediated pathways can aggravate problem caused by systemic hypertension to the eyes. Advanced glycation end-products (AGEs) and oxidative stress are key factors that contribute to endothelial dysfunction. This interaction exacerbates the development of hypertensive retinopathy, and also increases the risk of developing retinal vein occlusions substantially in the clinic.

4.3. Impact on Macular Edema

Excessively high dietary sugar levels have a compelling effect in intensifying the enhancement of vascular permeability primarily through inflammatory response and VEGF upregulation. One of the changes in the retina that contribute to most common reason of vision loss in individuals affected by DR is Diabetic Macular Edema (DME). These underlying pathological factors contribute to increasing the amount of swelling in the retina in response to too high a blood sugar, causing worsening vision loss.

5. Treatment and prevention aspects

5.1. Sugar Limitation is a nutrition intervention.

The backbone of preventative care is a strategic decrease in the amount of added sugars and carbohydrates consumed that have a high glycemic index. Enriched in antioxidants, fibre and Omega 3 fatty acids, and low processed sweeteners,

this diet may help to enhance the overall metabolic profile and help slow the development of retinal disease and modify this in a clinical way.

5.2. Management of glycemia: strategies.Management of glycemia: strategies.

The most reliable way to prevent onset of retinopathy is by maintaining comprehensive blood glucose control using medication and good diet. Post prandial glucose levels are especially important to watch and manage, and directly worsened by consumption of refined sugars.

5.3. Pharmacological Innovations targeting Sugar-induced Damage.

Current scientific research on treatment of diseases is increasingly directed at blocking the specific molecular pathways that are activated by sugar. Investigations are being pursued for inhibitors of aldose reductase (polyol pathway), and for the RAGE inhibitors or AGE breaking compounds which seek to protect the retina, irrespective of overall glucose levels. Also, although anti-VEGF protocols are applied in advanced phases, they basically treat the late vascular effects of inflammation caused by an overconsumption of sugar.6. Future Research Directions

Future research should focus on understanding the differential effects of different sugar components (e.g., fructose vs. glucose) on the retina; establishing safe limits for sugar intake in vulnerable populations; and creating specific nutraceuticals or drugs that could prevent the retina from specific molecular damage from high-sugar diets.

Future Research Directions

Additional studies are needed on these relevant aspects of this critical health issue:

- Characterizing the specific biochemical effects of each of the different sugars (e.g. fructose versus glucose) on retinal tissue.
- Establish clear sugar-sugar guidelines and policies, particularly for vulnerable groups of population who have a greater risk of developing metabolic disorders.
- Innovative nutraceuticals and/or targeted pharmacological agents that will help to protect an over-consumed retina from the molecular damage inflicted.

6. Conclusion

Relevant evidence has been pieced together, supporting strong relationships between the increased incidence and the increased burden of retinopathy with overconsumption of refined and added sugar. This relationship is brought about by a complex network of inflammatory, oxidative and metabolic disturbances. Results highlight the critical role of an all-round approach to nutritional assessment and proactive nutrition counselling for routine management of patients with risk factors for vision loss. The key to long-term eye health is through holistic management both in terms of nutrition as a discipline and also the traditional medical way of treatment.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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