Vita Columbia

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Sugar
and
Modern Disease

vitaSWEET: a natural alternative to reduce added sugar consumption

vitaSWEET

by Vita Columbia

An ancient alternative as sweet as sugar but without the metabolic concequences

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Sugar and Modern Disease: Substitution with vitaSWEET to reduce Added Sugar Consumption

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Grewal, VP. Vita Columbia Clinical Research Inc. *January 2nd, 2020*

In 2011, the United Nations (UN) general assembly adopted a resolution for the first time acknowledging chronic non-communicable diseases (CNCDs) as one the major socioeconomic challenges in the twenty-first century. The UN declared CNCDs such as heart disease, cancer, diabetes, and chronic respiratory diseases a threat to all countries, killing three in five people and contributing to 35 million deaths annually. The UN identified tobacco use, harmful alcohol use, and an unhealthy diet as the primary risk factors for the development of CNCDs.[1] While public health authorities have imposed significant regulation and educational efforts to minimize tobacco and alcohol use, the essentiality of food and complexity of nutrition has made regulation and education difficult.[2]

In recent years, researchers have identified added sugar consumption as one of the most significant dietary causes of CNCDs, in addition to neurodegenerative diseases, and even pediatric diseases such as autism and childhood obesity.[2] In the past 50 years, added sugar consumption has increased exponentially and become a staple of the western diet. Sugar is not only present in obvious foods such as deserts and sweetened beverages, it has become a hidden ingredient added to nearly all processed foods.[3] In recent years, financial incentives have driven multinational corporations to replace sugar with high-fructose corn syrup in certain products. In many regions of the world, people are consuming over 500 calories per day from added sugar alone.[4]

Both sugar and high-fructose corn syrup are comprised of equal parts of glucose and fructose and are very high on the glycemic index, meaning that they cause blood glucose levels to rise rapidly. Insulin is the primary hormone involved in the regulation of blood glucose levels, which binds to specific receptors on cells to facilitate glucose entry and utilization by cells. Repeated hyperglycemic episodes cause cells to remove insulin receptors from their membrane, in a phenomenon termed insulin resistance. The body is initially able to maintain normal blood glucose levels by increasing insulin secretion, but ultimately it is unable to compensate and type 2 diabetes mellitus (T2DM) develops.

The most serious manifestation of T2DM are vascular complications which include retinopathy, nephropathy, neuropathy, cerebrovascular diseases, coronary heart disease, and peripheral vascular disease, often leading to blindness, stroke, heart attack, and amputation of limbs. Vascular complications in T2DM increase in proportion to the duration of hyperglycemia.[5] Chronic hyperglycemia triggers endothelial dysfunction, the primary factor in the pathogenesis of diabetic vascular complications. Endothelial cells (ECs) line the surface of all blood vessels and serve as a barrier between circulating blood and tissues. ECs are particularly sensitive to changes in blood glucose levels, and hyperglycemic conditions induce acute endothelial inflammation and a prolonged pro-inflammatory state, leading to endothelial dysfunction. Endothelial dysfunction is a disruption in vascular homeostasis characterized by reduced nitric oxide production, increased endothelial permeability, increased expression of adhesion molecules, and increased apoptosis.[6,7]

In addition to the vascular complications of T2DM, endothelial dysfunction also plays a significant role in the pathogenesis of inflammatory disorders such as rheumatoid arthritis, asthma, cerebral small vessel disease, and autoimmune disorders such as inflammatory bowel disease.[8] Furthermore, researchers believe endothelial dysfunction is an early pivotal event preceding the development of coronary artery atherosclerosis and heart disease.[9] The risk of developing autoimmune diseases, diabetes, and atherosclerotic coronary artery disease is directly related to the number, intensity and duration of hyperglycemic events, with damage beginning years before the formal diagnosis of diabetes.[7]

Another mechanism by which uncontrolled hyperglycemia associated with T2DM leads to vascular complications is through the accumulation of advanced glycation end-products (AGEs). AGEs are a complex group of compounds consisting of proteins, lipids, or DNA which undergo glycation when exposed to sugars. The human body lacks enzymes to eliminate glycated products, which progressively accumulate as metabolic waste and cause functional deterioration of organs and tissues during the aging process.[10] The glycation reaction results in impaired protein function and reduced

elasticity of tissues such as blood vessels, skin, and tendons.[11,12,13] Many chronic diseases such as atherosclerosis, chronic kidney disease, rheumatoid arthritis, Alzheimer's disease, Parkinson's disease, vascular dementia, cataracts, other degenerative ophthalmic diseases, and many other diseases are associated with the accumulation of AGEs in tissues.[14,15,16] Furthermore, there is significant evidence that AGEs contribute to sarcopenia and the loss of bone density and muscle mass associated with aging.[17]

Science has only recently accepted the role of insulin in the brain, which includes neuronal development, synapse formation, learning and memory, glucoregulatory function and feeding behavior. Researchers suspect that the high concentration of insulin found in the brain is the result of local production in the brain and active transported across the blood-brain-barrier via a specific transporter, affected by hyperglycemic states.[18,19] Emerging studies have correlated hyperglycemia with having a direct role in the development of neurodegenerative diseases, such as Alzheimer's disease (AD).[20]

The connection between Alzheimer's Disease (AD) and hyperglycemia is so close, that researchers have begun referring to Alzheimer's as "type 3 diabetes".[21,22] Recent stuides have revealed insulin resistance to have a significant effect on the accumulation of both beta-amyloid (Aβ) plaques and neurofibrillary tangles (NFTs), the two main features believed to be associated with the development of AD. Several studies have shown a significant correlation between insulin resistance and Aβ synthesis, aggregation, and clearance mechanisms.[20,23] Additionally, researchers found that insulin resistance causes excessive enzymatic activation of glycogen synthesis, leading to leading to the formation of neurofibrillary tangles responsible for the cognitive changes in AD.[20,24]

In addition to neurodegenerative disease in the geriatric population, emerging research has linked prenatal hyperglycemia and gestational diabetes mellitus (GDM) as a significant risk factor for long-term neuropsychiatric morbidity in children, in particular Autism spectrum disorder (ASD).[25,26] The prevalence of ASD has increased dramatically since the 1960s and and continues to rise annually at an exponential rate. ASD diagnoses in children have increased from 1 in 150 in the year 2000, to 1 in 59 in 2018, with autism prevalence increasing by 119.4% in this time period.[27] ASD is a neurodevelopmental disorder characterized by impairments in socialization, communication and language, and repetitive or unusual behaviors. ASD typically appears during the first 5 years of life and persists into adulthood. Individuals with ASD often have co-exiting neuropsychological conditions such as epilepsy, depression, anxiety, and attention deficit hyperactivity disorder (ADHD). On average, autism costs families $60,000 USD per year. Data from observational studies suggests that as many as 45% of GDM cases may be preventable by adherence to a healthy lifestyle.[28]

Added sugar consumption is also a primary factor for many detrimental health conditions during childhood including obesity, dental caries, asthma, altered lipid profiles, hypertension, and cancer. Preventing childhood obesity in particular is vital due to its association with lifelong obesity, metabolic disorders such as T2DM and hypertriglyceridemia, obesity-related cancers, in addition to psychosocial repercussions and decreased educational attainment.[29,30,31,32,33]

Infants and children around the world exhibit a heightened preference for sweetness, most likely a biological underpinning to confer advantage in environments of scarcity. Moreover, research has shown significant similarities in neurochemistry and behavior between consumption of added sugars and drug-like effects, including bingeing, craving, tolerance, withdrawal, cross-sensitization, cross-dependence, reward and opioid effects. Sugar addiction appears to develop as a result of natural endogenous opioids released upon sugar consumption. The opioid-like pain reducing properties of sugar are evident from birth. A sweet solution placed in a newborns mouth, elicits facial relaxation, often accompanied with a smile. Furthermore, something sweet placed on the tongue of a crying newborn produces an instantaneous calming effect.[34] In another study, sucrose consumption delayed pain reporting in children aged 8 to 11 years old undergoing a cold-induced pain stimulus test, whilst having no effect in adult.[35] Evolutionary driven taste preferences, coupled with heightened response to natural endogenous opioids produced upon sugar consumption make infants and children more vulnerable to sugar addiction than adults.

Much of illness afflicting modern society can be attributed to changes in eating patterns. In the past 160 years the amount of sugar consumed by society has exploded, attributed to the mechanization of sugar production. This trend is not limited to the developed world, but has impacted almost every country on the planet. Emerging research has identified added sugar consumption as a significant risk factor for the development of CNCDs including heart

disease, diabetes, cancer and chronic respiratory diseases, neurodegenerative diseases such as Alzheimer's disease, childhood autism secondary to GDM, childhood obesity and associated diseases, in addition to autoimmune conditions such as arthritis, inflammatory bowel disease, multiple sclerosis, and vasculitis. These illnesses combined pose enormous socioeconomic implications for every community and nation in the world.

One simple method to reduce dietary sugar consumption is through the substitution of sugar for an all-natural alternative such as vitaSWEET, which is as sweet as sugar, zero calories, has no effect on blood glucose levels, and is safe during pregnancy and for children.

vitaSWEET is a natural alternative to table sugar and high fructose corn syrup, that is 0 on the glycemic index, meaning that it has no effect on blood glucose levels and therefore is not associated with the metabolic consequences of sugar. vitaSWEET is formulated with two simple ingredients: monk fruit and erythritol.

Monk fruit or luo han guo is native to southern China and northern Thailand, and has almost 300 times natural sweetness than that of sugar. It has been used for centuries in traditional Chinese medicine, first mentioned in the records of 14th century Chinese monks. Erythritol is a naturally occurring sugar alcohol derived from fruits and plants. Vita Columbia's unique blend of both monk fruit and erythritol is as sweet as sugar and can be used as a 1-to-1 replacement in recipes.

vitaSWEET

An ancient alternative as sweet as sugar but without the metabolic concequences

References:

1. General Assembly resolution 66/2, Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases, A/RES/66/2 (19 September 2011)
2. Joint WHO/FAO Expert Consultation. Diet, Nutrition and the Prevention of Chronic Diseases WHO Technical Report Series 916 (WHO; 2003).
3. Vio, F. & Uauy, R. in Food Policy for Developing Countries: Case Studies (eds Pinstrup-Andersen, P. & Cheng, F.) No. 9–5 (2007).
4. Lustig, R., Schmidt, L. & Brindis, C. The toxic truth about sugar. *Nature.* 482, 27–29 (2012).
5. Silambarasan, M, Tan, JR, Karolina, DS, et al. MicroRNAs in Hyperglycemia Induced Endothelial Cell Dysfunction. *Int. J. Mol. Sci.* 2016;17(518).
6. Li, J, Huang, W, Tu, R. et al. Resveratrol rescues hyperglycemia-induced endothelial dysfunction via activation of Akt. *Acta Pharmacol Sin.* 2017;38:182–191.
7. Hansen NW, Jon Hansen A, Sams A. The endothelial border to health: Mechanistic evidence of the hyperglycemic culprit of inflammatory disease acceleration. *IUBMB Journals.* 2017;69(3):148-161.
8. Fishman, S.L., Sonmez, H., Basman, C. et al. The role of advanced glycation end-products in the development of coronary artery disease in patients with and without diabetes mellitus: a review. *Mol Med.* 2018;24(59).
9. Caballero, AE. Endothelial Dysfunction in Obesity and Insulin Resistance: A Road to Diabetes and Heart Disease. *Obesity Research.* 2003;11(11):1278-1289.
10. Brownlee M. Advanced protein glycosylation in diabetes and aging. *Annu Rev Med.* 1995; 46: 223-34.
11. Nguyen HP, Katta R. Sugar Sag: Glycation and the Role of Diet in Aging Skin. *Skin Therapy Lett.* 2015; 20:1-5.
12. Pageon H, Zucchi H, Rousset F, Monnier VM, Asselineau D. Skin aging by glycation: lessons from the reconstructed skin model. *Clin Chem Lab Med.* 2014; 52: 169-74.
13. Jakus, V., Rietbrock, N. Advanced glycation end-products and the progress of diabetic vascular complications. *Physiol. Res.* 2004; 53, 131-142.
14. Shuvaev, V.V., Laffont, I., Serot, J.M., Fujii,J., Taniguchi, N., Siest,G. Increased protein glycation in cerebrospinal fluid of Alzheimer's disease. *Neurobiol. Aging.* 2001; 22,397-402.
15. Kim, C.S., Park, S., Kim, J. The role of glycation in the pathogenesis of aging and its prevention through herbal products and physical exercise. *J Exerc Nutrition Biochem.* 2017; 21(3): 055-061.
16. Stopper H, Schinzel R, Sebekova K, Heidland A. Genotoxicity of advanced glycation end products in mammalian cells. *Cancer Lett.* 2003; 190: 151-6.

.17. Odetti, P., Rossi, S., Monacelli, F., Poggi, A., Cirnigliaro, M., Federici, M., Federici, A. Advanced glycation end products and bone loss during aging. *Ann. N.Y. Acad. Sci.* 2005; 1043,710-717
18. Ho L, et al. Diet-induced insulin resistance promotes amyloidosis in a transgenic mouse model of Alzheimer's disease. *FASEB J.* 2004;18(7):902–4.
19. Derakhshan F, Toth C. Insulin and the brain. *Curr Diabetes Rev.* 2013;9(2):102–16.
20. Takeda S, Morishita R. 2017. Diabetes and Alzheimer's Disease. In: Yamagishi S, editor. Diabetes and Aging-Related Complications. Singapore: Springer Nature Singapore Pte Ltd. p. 101-111.
21. de la Monte, S. M., & Wands, J. R. (2008). Alzheimer's Disease is Type 3 Diabetes—Evidence Reviewed. *Journal of Diabetes Science and Technology.* 2(6), 1101–1113.
22. de la Monte SM. 2019. The Full Spectrum of Alzheimer's Disease is Rooted in Metabolic Derangements that Drive Type 3 Diabetes. In: Nakabeppu Y, Ninomiya T. Diabetes Mellitus. Singapore Pte Ltd. p. 45-83.
23. Stohr O, et al. Insulin receptor signaling mediates APP processing and beta-amyloid accumulation without altering survival in a transgenic mouse model of Alzheimer's disease. *Age (Dordr).* 2013; 35(1):83–101.
24. Tepper K, et al. Oligomer formation of tau protein hyperphosphorylated in cells. *J Biol Chem.* 2014;289(49):34389–407.
25. Mengying, L.M., Fallin, D., Riley, A. et al. The Association of Maternal Obesity and Diabetes with Autism and Other Developmental Disabilities. *Official Journal of the American Academy of Pediatrics.* 2016. 137(2).
26. Xu, G., Jing J., Browers K., et al. Maternal Diabetes and the Rising Autism Spectrum Disorders in the Offspring. A Systematic Review and Meta-Analysis. *Journal of Autism and Developmental Disorders*. 2013. 44(4): 766-775.
27. Elsabbagh, M., Divan, G., Koh, Y.J., et al. Global prevalence of autism and other pervasive developmental disorders. *Autism Res.* 2012. 5(3): 160–179.
28. Amendah, D., Grosse, S.D., Peacock, G., & Mandell, D.S. (2011). The economic costs of autism: A review. In D. Amaral, D. Geschwind, & G. Dawson (Eds.), Autism spectrum disorders (pp. 1347-1360). Oxford: Oxford University Press.
29. Herrick KA, Fryar CD, Hamner HC, et al. Added Sugars Intake among US Infants and Toddlers. *Journal of the Academy of Nutrition and Dietetics.* 2019.
30. Nutrition and the Prevention of Chronic Diseases: Report of a Joint WHO/FAO Expert Consultation. World Health Organization, Geneva, Switzerland; 2004
31. Millen, B.E., Abrams, S., Adams-Campbell, L. et al. The 2015 Dietary Guidelines Advisory Committee scientific report: Development and major conclusions. *Adv Nutr.* 2016; 7: 438–444
32. Johnson, R.K., Appel, L.J., Brands, M. et al. Dietary sugars intake and cardiovascular health: A scientific statement from the American Heart Association. *Circulation.* 2009; 120: 1011–1020
33. Thornley, S., Stewart, A., Marshall, R. et al. Per capita sugar consumption is associated with severe childhood asthma: an ecological study of 53 countries. *Prim Care Respir J.* 2011; 20, 75–78.
34. Ventura, A.K., Mennella, J.A. Innate and learned preferences for sweet taste in childhood. *Current Opinion in Clinical Nutrition and Metabolic Care.* 2011. 14(4): 379-384.
35. DiNicolantonio, J.J., O'Keefe, J.H. Sugar addiction: is it real? A narrative review. *British Journal of Sports Medicine.* 2017. 52(14): 910-913

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