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Editorial

Sugars and their Support

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We just ate Christmas. For many it is a time to let dietary inhibitions fall away and over-indulge, with cake, chocolates, desserts and sweet liqueurs on the menu. So, every year, at this time of year, people ask me if sweetness is a sin.

Most higher animals have taste perception systems that are similar to ours, with specific genes that code for receptors for each of the tastes: sweet, salty, sour, bitter, umami and, in some species, fat. Herbivores and omnivores like sweetness, because for them, the ability to taste and enjoy carbohydrate-rich foods is a matter of calories, and survival. For carnivores, sweetness is irrelevant, and most of them have mutations that disable the sweet receptors on their tongues [1,2]. This is why cats don't care about chocolate, while rats, dogs, pigs, monkeys and humans do.

But not everything that tastes sweet is a carbohydrate. In fact, the sweet sensors on our taste buds are amazingly promiscuous and react to many different kinds and shapes of molecule. Many sugars taste sweet but so do cyclamates (40 times sweeter than sugar, or 40X), di-peptides like aspartame (180X), diterpenes like the steviasides (250X), benzoic sulphamides such as saccharin (300X), and polyphenols such as the dihydrochalcones (350X).

There is group of far larger molecules that stimulate the sweet receptors even more intensely. These are the proteins monellin (from the Miracle berry, 1500X), thaumatin (from the Katemfe fruit, 1500X) and brazzein (from the Oubli fruit, 2000X). These do not activate the sweet receptor directly but act as a kind of molecular wedge, holding the sweet receptors open and activated.

Some poisons taste sweet too.

The antifreeze polyethylene glycol is one, lead acetate - formerly known as lead sugar - is another. Used to colour and extend the working life of paint, lead acetate in paints and wallpapers poisoned those children who accidentally discovered that the brightly coloured woodwork of their cots, or the wallpaper over them, tasted like candy. (This last phenomenon was reprised in Roald Dahl's 1964 children's novel Charlie and the Chocolate Factory).

Sweet receptors occurs not only on the tongue but in many other tissues also. You find them for example in the hypothalamus, the pancreas, and throughout the gut [3] where they probably act as energy sensors and contribute to the regulation of blood sugar levels, appetite and gut function. They are found in the testicles too, where they are probably involved in determining whether there is enough energy available to divert calories to testosterone, sperm production and sex. Sex is fun but not essential to life in the short term, which is why very low calorie diets tend to reduce libido.

If the intense sweeteners used in so many foods and beverages are able to act at sweet receptors on the tongue, it seems at least possible that they will also act at sweet receptors in some of the other tissues listed above. This is one reason why there has been so much research into their possible adverse effects; the other reason, of course, being the obvious financial interest of Big Sugar.

Big Sugar's involvement in nutritional research has been largely corrupt, and hugely corrupting. For example, there is extensive evidence that Big Sugar bribed leading scientists at Harvard and elsewhere to suppress evidence of the harmful effects of excessive sugar consumption, and to play up the supposedly harmful effects of intense sweeteners [4,5]. This is very much in line with Big Sugar's track record of lobbying, bribing and blackmailing politicians at federal and state level, to ensure favourable market conditions for their industry. They do this internationally too. In 2003, for instance, when the World Health Organization recommended that people reduce the amount of sugar they consume, American sugar companies threatened to appeal to Congress to cut the WHO's funding [6] and the WHO backed off.

You can see the damaging impact of the sugar industry in many places. These range from the truly appalling state of American public health and the body shape of the average American consumer, to the environmental destruction in sugar cane country (Florida, Louisiana, Texas and Hawaii) and worst of all, the composition of American processed and ultra-processed foods.

A can of sugar-sweetened soda contains roughly 40 grams or 10 teaspoons of sugar, but the sugar content of pasta sauces, ketchup and many other 'savoury' foods is not far behind, rendering them almost inedible to the unsuspecting European.

American white bread, for example, is now as sweet as brioche, sans the textural subtlety of its continental cousin; 'fruit' yoghurts are as sweet as ice cream, and pasta sauces contain as much sugar as cookies.

How can they eat this stuff?

There is evidence that as the sugar industry has pushed more and more sugar into processed foods the American palate has shifted, and now requires more sugar to experience the same level of sweetness [7]. The food engineers admit as much. Howard Moskowitz, trained in high math at Queens College and experimental psychology at Harvard (but who clearly never studied health or ethics), is largely responsible for the bliss-point research that has turned so many US consumers into diabetic, cancer-prone junk food junkies.

Sugar is a key component in the ultra-processed diet, and when consumed in large amounts, and in these foods, is extremely unhealthy. How do the intense sweeteners compare?

Saccharin is the oldest. First discovered in 1879, it had a clear run until the 1970's when studies on lab rats found an association between high doses of saccharin and bladder cancer [8]. Subsequent work found that the bladder cancer was caused by a mechanism that does not occur in humans [9] and that saccharin actually has modest chemo-protective properties, via blockade of the cancer-enabling enzyme carbonic anhydrase [10].

It is not just saccharin that has been accused of causing cancer. Every other intense sweetener has been similarly accused at one time or another, from the cyclamates to aspartame and from sucralose to stevia [11]; and each one has come out clean.

But still, a nagging doubt remains. If the intense sweeteners really do substitute for sugar they should help weight loss and improved metabolism – yet the results are mixed [12-15]. And, to return to the ubiquity of the sweet receptor, it turns out that many bacteria have receptors very like ours; and that they react to intense sweeteners in ways that might theoretically impact on our health [16,17].

And yet, and yet…

The safety of the intense sweeteners currently approved in the U.S. and Europe (stevia, acesulfame-K, aspartame, neotame, saccharin and sucralose) is supported by literally hundreds of pre-clinical and clinical studies. A relatively small group of studies have shown adverse effects, but most of these, including the microbiome work, are severely limited due to effects shown only in animals or in vitro, small sample size, excessive doses, statistically non-significant or borderline significant results, and, in some cases, shockingly poor study design. And in the background, the evidence linking sugar consumption to disease continues to grow [5].

In the end, something will kill you. It could be kinetic, metabolic, infectious or genetic, or a combination of any of those. And while intense sweeteners may be somewhere on that list, they are way, way down the pecking order, and in all likelihood far lower than sugar, let alone tobacco, alcohol, television and over-done steaks.

The fact that so many otherwise sane'ish people are so against sweeteners is more to do with nutri-Puritanism than science.

Bibliography

1. Li X., *et al*. "Pseudogenization of a sweet-receptor gene accounts for cats' indifference toward sugar". *PLOS Genetics* 1.1 (2005): 27-35.
2. Jiang P., *et al*. "Major taste loss in carnivorous mammals". *Proceedings of the National Academy of Sciences of the United States of America* 109.13 (2012): 4956-4961.
3. Lee AA and Owyang C. "Sugars, Sweet Taste Receptors, and Brain Responses". *Nutrients* 9.7 (2017): 653.
4. Kearns CE., *et al*. "Sugar Industry and Coronary Heart Disease Research. A Historical Analysis of Internal Industry Documents". *JAMA Internal Medicine* 176.11 (2016): 1680-1685.
5. Kearns CE., *et al*. "Sugar industry sponsorship of germ-free rodent studies linking sucrose to hyperlipidemia and cancer: An historical analysis of internal documents". *PLOS Biology* 15.11 (2017): e2003460.
6. Sarah Boseley. "Sugar Industry Threatens to Scupper WHO". *The Guardian* (2003).
7. Bartolotto C. "Does Consuming Sugar and Artificial Sweeteners Change Taste Preferences?" *The Permanente Journal* 19.3 (2015): 81-84.
8. Price JM., *et al*. "Bladder tumors in rats fed cyclohexylamine or high doses of a mixture of cyclamate and saccharin". *Science* 167.3921 (1970): 1131-1132.
9. Weihrauch MR and Diehl V. "Artificial sweeteners–do they bear a carcinogenic risk?" *Annals of Oncology* 15.10 (2004): 1460-1465.
10. Sonmez F., *et al*. "*In vitro* inhibition effect and structure-activity relationships of some saccharin derivatives on erythrocyte carbonic anhydrase I and II". *Journal of Enzyme Inhibition and Medicinal Chemistry* 29.1 (2014): 118-123.
11. https://cspinet.org/eating-healthy/chemical-cuisine#stevia
12. Azad MB., *et al*. "Nonnutritive sweeteners and cardiometabolic health: a systematic review and meta-analysis of randomized controlled trials and prospective cohort studies". *Canadian Medical Association Journal* 189.28 (2017): E929-E939.
13. Patel L., *et al*. "Low-Calorie Beverage Consumption, Diet Quality and Cardiometabolic Risk Factors in British Adults". *Nutrients* 10.9 (2018): 1261.
14. Miller PE and Perez V. "Low-calorie sweeteners and body weight and composition: a meta-analysis of randomized controlled trials and prospective cohort studies". *The American Journal of Clinical Nutrition* 100.3 (2014): 765-777.
15. Reid AE., *et al*. "Early Exposure to Nonnutritive Sweeteners and Long-term Metabolic Health: A Systematic Review". *Pediatrics* 137.3 (2016): e20153603.
16. Suez J., *et al*. "Artificial sweeteners induce glucose intolerance by altering the gut microbiota". *Nature* 514.7521 (2014): 181-186.
17. Wang Q-P., *et al*. "Non-nutritive sweeteners possess a bacteriostatic effect and alter gut microbiota in mice". *PLoS One* 13.7 (2018): e0199080.

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