# Guidelines on 'added' sugars are unscientific and unnecessary

*Edward Archer*

In her recent Comment article (Guidelines to lower intake of added sugar are necessary and justified. *Nat. Rev. Cardiol.* **19**, 569–570 (2022))[1], Kimber Stanhope offers a rebuttal to criticisms that recommendations to reduce the consumption of 'added' sugars were based on "low-quality evidence" and "ill-informed opinions". Given the prominence (and misrepresentation) of my critique[2] in that rebuttal[1], I write to correct several misconceptions.

To begin, my colleagues and I established that the data underlying the US Dietary Guidelines were physiologically implausible[3,4], pseudo-scientific (non-falsifiable)[5,6] and "essentially meaningless"[5]. We further showed that, by obscuring established facts and causal mechanisms[7,8], these data created a "fictional discourse on diet–disease relations"[6]. Therefore, because the rebuttal[1] failed to address our conclusions, the critique that recommendations were "ill-formed" and based on "low-quality evidence" stands unopposed. However, more importantly, the author misrepresented the large body of rigorous evidence presented in my critique[2] and previous reviews[6,8].

First, sugars added to foods and beverages enter the same metabolic pathways as sugars intrinsic to foods and beverages[9]. This unequivocal fact is an a priori refutation of the position that 'added' sugars are unique and demonstrates that the term 'added sugar' has only rhetorical, not scientific, value[2,8].

Second, humans begin life consuming ~40% of their daily calories as dietary sugars — either in breast milk or infant formula. However, infant formula is an 'ultra-processed', sugar-sweetened beverage with 'added' sugar, 'added' salt and 'added' fat. Therefore, recommendations to limit 'added' sugar and 'processed' foods would prevent the proper feeding of most infants in industrialized nations. And contrary to anti-sugar rhetoric, nations with the highest rates of sugar-sweetened beverage (formula) consumption by infants (for example, Japan and Norway) also have the lowest rates of obesity, type 2 diabetes mellitus and cardiovascular disease[2,8].

Third, the medicinal use of sucrose (table sugar) for malnutrition and diarrhoeal diseases saves the lives of 600,000 children each year, and if every ill child were treated, another 500,000 would be saved. Therefore, so-called 'added' sugars have saved more lives than any pharmaceutical agent[2,8].

Fourth, the most comprehensive governmental reports on dietary sugars drew surprisingly similar conclusions[9,10]. The US report concluded that there was "no plausible evidence that the consumption of simple sugars" was related to the aetiology of obesity, type 2 diabetes or cardiovascular disease[9] and that "feeding normal human volunteers at levels of fructose approximating the 90th percentile intake levels of the U.S. population failed to demonstrate adverse effects on insulin sensitivity or glucose tolerance"[9].

The UK report concluded that "the consumption of sugars within the present range in the UK carries no special metabolic risks" and "played no direct causal role in the development of cardiovascular disease … essential hypertension, or of diabetes mellitus"[10]. Given these unequivocal findings, arguments that sugars and sugar-sweetened beverages are causal to obesity, type 2 diabetes and cardiovascular disease defy rigorous experimental and real-world evidence[9,10] while obscuring established facts and causal relationships[7,8].

Most importantly, because foods and beverages are often the only innately gratifying 'goods' that economically or socially disadvantaged people can purchase, proscriptions against sugars and fats are "regressive and unjust because they harm the most vulnerable members of our society while providing no personal or public health benefits"[2].

In conclusion, no-one — and especially not disadvantaged individuals — should be subjected to the fear and confusion caused by anti-sugar rhetoric or to recommendations based on physiologically implausible and pseudo-scientific dietary data.

There is a reply to this letter by Stanhope, K. L. *Nat. Rev. Cardiol.* https://doi.org/10.1038/s41569-022-00794-7 (2022).

*Edward Archer*
*EvolvingFX, Fort Wayne, IN, USA.*
*e-mail: archer1@evolvingfx.com*

https://doi.org/10.1038/s41569-022-00792-9

1. Stanhope, K. L. Guidelines to lower intake of added sugar are necessary and justified. *Nat. Rev. Cardiol.* **19**, 569–570 (2022).
2. Archer, E. & Arjmandi, B. Falsehoods and facts about dietary sugars: a call for evidence-based policy. *Crit. Rev. Food Sci. Nutr.* **61**, 3725–3739 (2021).
3. Ioannidis, J. P. A. Implausible results in human nutrition research. *BMJ* **347**, f6698 (2013).
4. Archer, E., Hand, G. A. & Blair, S. N. Validity of U.S. nutritional surveillance: National Health and Nutrition Examination Survey caloric energy intake data, 1971-2010. *PLoS One* **8**, e76632 (2013).
5. Archer, E., Pavela, G. & Lavie, C. J. The inadmissibility of what we eat in America and NHANES dietary data in nutrition and obesity research and the scientific formulation of national dietary guidelines. *Mayo Clin. Proc.* **90**, 911–926 (2015).
6. Archer, E., Lavie, C. J. & Hill, J. O. The failure to measure dietary intake engendered a fictional discourse on diet-disease relations. *Front. Nutr.* **5**, 105 (2018).
7. Archer, E., Pavela, G., McDonald, S., Lavie, C. J. & Hill, J. O. Cell-specific "competition for calories" drives asymmetric nutrient-energy partitioning, obesity, and metabolic diseases in human and non-human animals. *Front. Physiol.* **9**, 1053 (2018).
8. Archer, E. In defense of sugar: a critique of diet-centrism. *Prog. Cardiovasc. Dis.* **61**, 10–19 (2018).
9. Glinsmann, W. H., Irausquin, H. & Park, Y. K. Evaluation of health aspects of sugars contained in carbohydrate sweeteners. Report of Sugars Task Force, 1986. *J. Nutr.* **116**(Suppl.), S1–S216 (1986).
10. Committee on Medical Aspects of Food Policy. Report on health and social subjects: 37. Dietary sugars and human disease. *Department of Health* https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/743792/Dietary_Sugars_and_Human_Disease__1989_.pdf (1989).

**Acknowledgements**
The content is solely the responsibility of the author and does not necessarily represent the official views of EvolvingFX, LLC. No financial support was received for the preparation of this paper.

**Competing interests**
The author declares no competing interests.

# Reply to: 'Guidelines on 'added' sugars are unscientific and unnecessary'

*Kimber L. Stanhope*

I thank Dr Archer for his Correspondence (Guidelines on 'added' sugars are unscientific and unnecessary. *Nat. Rev. Cardiol.* https://doi.org/10.1038/s41569-022-00792-9 (2022))[1] on my Comment article (Guidelines to lower intake of added sugar are necessary and justified. *Nat. Rev. Cardiol.* **19**, 569–570 (2022))[2]. The stated purpose of Dr Archer's letter is to correct several misconceptions. However, he does not explain how any of the statements

I made in the Comment article represent misconceptions that require correction. Instead, he re-asserts that the data underlying the US Dietary Guidelines are "physiologically implausible, pseudo-scientific and essentially meaningless" and states that I failed to address these conclusions. Therefore, I will specifically explain why each of the five arguments contained in his letter (quoted below in this Reply), and also in his review[3], do not support his conclusions.

> *Sugars added to foods and beverages enter the same metabolic pathways as sugars intrinsic to foods and beverages*

This statement is true. However, it is also true that the concentration of sugar in the food affects the activity of these metabolic pathways. As stated in the Comment article, the metabolism of fructose is controlled by the enzyme fructokinase. Because fructokinase is unregulated by hepatic energy needs, approximately 85% of the fructose that we consume is taken up and metabolized in the liver. This substrate overload activates the metabolic pathways, leading to upregulated production of fat and uric acid and downregulated oxidation of fat. The direct effects are increased liver lipid content and circulating triglyceride and uric acid levels. Related effects include increased plasma levels of atherogenic lipoproteins and decreased insulin sensitivity. All these risk factors have been shown to be sensitive to the dose of fructose-containing sugar[4].

The dose of sugar is the most important difference between foods with added sugar and those with naturally occurring sugar. Food processors add a much higher concentration of sugar to processed foods such as candy, cookies, cakes, ice cream and pastries than nature adds to fruit — humans' main source of naturally occurring sugar. For example, the concentration of sugar in grapes, which is highest among all fruits, is 15% by weight, with the majority of fruits having <10% sugar by weight. The concentration of added sugar in the top-selling cookie in the USA (Nabisco Oreo) is 38%. Therefore, overloading the liver with fructose is easier when consuming sweetened processed foods than when consuming fruit.

Food matrix, defined as the nutrient and non-nutrient components of foods and their molecular relationships, can also affect the activity of the metabolic pathways. Fruits contain fibre and polyphenols[5] that interfere with and slow macronutrient absorption. Therefore, fruit consumption is likely to result in less fructose overload of the liver than consumption of the same amount of sugar in a low-fibre processed food. The polyphenols[5] and other micronutrients in fruit might also have direct effects on specific metabolic pathways that lead to health benefits, but this topic has not been well studied. However, three clinical dietary intervention studies have shown that the consumption of naturally sweetened orange juice lowers circulating uric acid levels compared with the consumption of a sugar-sweetened beverage[6,7].

> *Humans begin life consuming ~40% of their daily calories as dietary sugars — either in breast milk or infant formula. However, infant formula is an 'ultra-processed', sugar-sweetened beverage with 'added' sugar, 'added' salt and 'added' fat*

There are two reasons why this argument is not relevant to the US Dietary Guidelines on added sugar. First, the US Dietary Guidelines are specific to individuals aged >2 years, and most children aged >2 years are not consuming breast milk or infant formula. Second, breast milk and infant formula contain lactose, and lactose does not contain fructose. Therefore, the consumption of lactose does not lead to fructose overload in the liver, which (as described above) is the trigger for metabolic dysregulation caused by added sugar.

> *The medicinal use of sucrose (table sugar) for malnutrition and diarrhoeal diseases saves the lives of 600,000 children each year, and if every ill child were treated, another 500,000 would be saved*

No-one, including those of us who support the US Dietary Guideline recommendation to lower consumption of added sugar, is advocating that physicians should not use the best possible treatment for a child with malnutrition or diarrhoea. However, the fact that sucrose is a component of the enteral therapy for malnutrition or diarrhoea does not exculpate sucrose, or other fructose-containing sugars, from contributing to the development of metabolic diseases when overconsumed in the typical Western diets of adequately nourished or over-nourished individuals.

> *The most comprehensive governmental reports on dietary sugars had surprisingly similar conclusions. The US report concluded that there was "no plausible evidence that the consumption of simple sugars" was related to the aetiology of obesity, type 2 diabetes or cardiovascular disease and that "feeding normal human volunteers at levels of fructose approximating the 90th percentile intake levels of the U.S. population failed to demonstrate adverse effects on insulin sensitivity or glucose tolerance"*

The reports that Dr Archer cites were written in 1986 and 1989. Obviously, they do not evaluate the studies on added sugar that have been conducted since 1989 and can no longer be considered comprehensive. As stated in the Comment article, reports from 21 clinical dietary intervention studies on added sugar have been published since 2000. The number of epidemiological studies conducted since 1990 on the relationship between added sugar and health might be as high as 400. Therefore, although the conclusions of these reports might have been valid as based on the scientific evidence available in 1985 and 1989, they are not valid now.

> *Most importantly, because foods and beverages are often the only innately gratifying 'goods' that economically or socially disadvantaged people can purchase, proscriptions against sugars and fats are "regressive and unjust because they harm the most vulnerable members of our society while providing no personal or public health benefits"*

To support the statement that proscriptions against sugar provide no personal health benefit, Dr Archer cites his own review[3]. There are no studies cited in this review that provide evidence that proscriptions against sugars cause no personal benefit. On the contrary, there are six recent dietary intervention studies (three from the past 3 years[8–10]) that show that adolescent and adult research participants had health benefits when they reduced their consumption of added sugar.

*Kimber L. Stanhope*
*Department of Molecular Biosciences, School of Veterinary Medicine, University of California – Davis, Davis, CA, USA.*
*e-mail: klstanhope@ucdavis.edu*

https://doi.org/10.1038/s41569-022-00794-7

1. Archer, E. Guidelines on 'added' sugars are unscientific and unnecessary. *Nat. Rev. Cardiol.* https://doi.org/10.1038/s41569-022-00792-9 (2022).
2. Stanhope, K. L. Guidelines to lower intake of added sugar are necessary and justified. *Nat. Rev. Cardiol.* **19**, 569–570 (2022).
3. Archer, E. & Arjmandi, B. Falsehoods and facts about dietary sugars: a call for evidence-based policy. *Crit. Rev. Food Sci. Nutr.* **61**, 3725–3739 (2021).
4. Sigala, D. M. et al. The dose–response effects of consuming high fructose corn syrup-sweetened beverages on hepatic lipid content and insulin sensitivity in young adults. *Nutrients* **14**, 1648 (2022).
5. Hanhineva, K. et al. Impact of dietary polyphenols on carbohydrate metabolism. *Int. J. Mol. Sci.* **11**, 1365–1402 (2010).

6. Büsing, F. et al. High intake of orange juice and cola differently affects metabolic risk in healthy subjects. *Clin. Nutr.* **38**, 812–819 (2019).
7. Price, C. A. et al. A pilot study comparing the effects of consuming 100% orange juice or sucrose-sweetened beverage on risk factors for cardiometabolic disease in women. *Nutrients* **13**, 760 (2021).
8. Khodami, B. et al. Effects of a low free sugar diet on the management of nonalcoholic fatty liver disease: a randomized clinical trial. *Eur. J. Clin. Nutr.* **76**, 987–994 (2022).
9. Schwimmer, J. B. et al. Effect of a low free sugar diet vs usual diet on nonalcoholic fatty liver disease in adolescent boys: a randomized clinical trial. *J. Am. Med. Assoc.* **321**, 256–265 (2019).
10. Simons, N. et al. Effects of fructose restriction on liver steatosis (FRUITLESS); a double-blind randomized controlled trial. *Am. J. Clin. Nutr.* **113**, 391–400 (2021).

**Acknowledgements**
The author thanks Bettina Hieronimus (Institute for Physiology and Biochemistry of Nutrition, Max Rubner-Institut, Germany) and Desiree Sigala (Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis, CA, USA) for discussion, insight and editing before submission.

**Competing interests**
K.L.S. has received honoraria for speaking from numerous organizations (since 2016: American Diabetes Association, Columbia University, Harvard School of Medicine, Swedish Medical Center and University of Missouri) and from CrossFit for serving as academic organizer of a 2017 conference and lead author of the conference summary report.