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TECHNICAL REPORT

A workshop on 'Dietary Sweetness—Is It an Issue?'

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This report summarises a workshop convened by ILSI Europe on 3 and 4 April 2017 to discuss the issue of dietary sweetness. The objectives were to understand the roles of sweetness in the diet, establish whether exposure to sweetness affects diet quality and energy intake, and consider whether sweetness *per se* affects health. Although there may be evidence for tracking of intake of some sweet components of the diet through childhood, evidence for tracking of whole diet sweetness, or through other stages of maturity are lacking. The evidence to date does not support adverse effects of sweetness on diet quality or energy intake, except where sweet food choices increase intake of free sugars. There is some evidence for improvements in diet quality and reduced energy intake where sweetness without calories replaces sweetness with calories. There is a need to understand the physiological and metabolic relevance of sweet taste receptors on the tongue, in the gut and elsewhere in the body, as well as possible differentiation in the effects of sustained consumption of individual sweeteners. Despite a plethora of studies, there is no consistent evidence for an association of sweetness sensitivity/preference with obesity or type 2 diabetes. A multifaceted integrated approach, characterising nutritive and sensory aspects of the whole diet or dietary patterns, may be more valuable in providing contextual insight. The outcomes of the workshop could be used as a scientific basis to inform the expert community and create more useful dialogue among health care professionals.

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INTRODUCTION

ILSI Europe's 'Dietary Carbohydrates' and 'Eating Behaviour and Energy Balance' task forces convened a workshop on 3 and 4 April 2017 to discuss the subject 'Dietary Sweetness – Is It an Issue?'. The full programme and speakers' presentations are available at http://ilsi.eu/event/ilsi-europe-workshop-on-dietary-sweetness-is-it-an-issue/. Speakers addressed topics related to the evolution and biology of sweetness, dietary exposure to sweetness, diet quality, energy intake and the implications of sweetness on health.

Session 1: The roles of sweetness in the diet

The evolution and biology of sweetness. Beauchamp described sweetness as a human percept, commented on the comparative biology of sweetness within and between species, and addressed the human appeal for sweetness. 'Sweet' appears as a consistent basic taste descriptor across cultures and the ability to identify sweet foods via taste is common, if not universal, in plant-eating species. Many natural sugars are sweet and provide energy, although there are numerous natural non-caloric sweeteners, few of which are used commercially.[1] Sweetness is motivating and liking is innate. However, variation in individuals' liking for sweetness is apparent, 30–50% of which may have a genetic basis.[2] Very few experimental studies have attempted to alter the sensitivity or preference for sweetness. A single study revealed that reduction in simple sugars exposure increased perception of sweet intensity, but did not alter preference,[3] though much more research is needed before conclusions can be drawn. The human liking for sweetness may have evolved to aid the safe sourcing of nutrients, perhaps by opposition with bitter taste which often indicates toxicity. Consideration of the sweet–bitter ratio as a primary signal may help understand the power of sweetness.[4]

Session 2: Sweetness, diet quality and energy intake: what is the evidence that exposure to sweetness affects diet quality and energy intake?

The impact of frequent/persistent exposure to sweetness in earlier life stages on intake of sweet sugar-containing foods and drinks later in life. Nicklaus indicated that very little research has examined exposure to whole diet sweetness. In a recent study, Nicklaus *et al.* characterised the diet from a sensory perspective and highlighted an increase in sweetness exposure from 3–6 to 10–12 months of age.[5] The relationship between sweetness exposure in these early months and dietary intake at 9 years old is currently under investigation. Most studies examining the tracking of consumption of sweet elements in the diet have used sweet foods, sugar-sweetened beverages or dietary sugars, which could act to various degrees as proxies for exposure to sweetness. Many studies clearly document a tracking of dietary patterns particularly during

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childhood. However, evidence for tracking of sweet food consumption from childhood through to adolescence, or through adolescence itself, is less clear and several studies show that the learning aspect of determining food preferences is rather specific to a given food and may not be generalised.[6–8]

Further research could benefit from development of databases, with improved nutritional information, such as the free sugars content of foods, as well as standardised characterisation of sensory information,[9] all of which may help with between study and country comparisons. Research needs to consider frequency of exposure, intensity of sweetness, the amount consumed and sweetness with/without calories. The life stages of most importance for researching the tracking of sweetness need to be determined.

As most evidence is observational, causal relationships cannot be confirmed. Intervention trials may be difficult for ethical and practical reasons. However, intervention studies on sweetness reduction could be a feasible avenue of research.

The role of sweetness in dietary patterns: past and present. de Graaf contextualised the sweetness of the diet with an overview of sugars consumption from an historic perspective. Dietary sweetness (sugars intake) has increased: being relatively absent from the Neolithic and early agricultural starch-rich diets, used sparingly as a spice or condiment in the middle ages, and thereafter increased with sugar cane cultivation,[10] the industrial revolution, beet cultivation and overall increased availability of sugar.[11] However, de Graaf noted that total sugars intake has remained relatively stable in the Netherlands since the 1970/80's. Others noted a similar recent stability in total sugars intake in the UK, although in the US, intakes of added sugars have fallen since the millennium.[12] The relative stability in total sugars intake in recent years is apparent in all age groups in the Netherlands. However, sugars provide a higher contribution to the diet in younger age groups, perhaps reflecting a higher sweet preference which has been noted in youth. On discussion, it was noted that the types of food and beverages contributing to sugars intake may affect the sweetness of the diet due to the dissimilar sweet intensity of the various monosaccharides and disaccharides, as well as the use of low-calorie sweeteners (LCS).

In support of the appeal for the development of food and beverage sensory databases, de Graaf presented preliminary data which employed a sensory database of 481 foods (comprising 83% of the Dutch energy intake), categorised using cluster analysis into six taste groups ('fat', 'sweet/sour', 'neutral', 'salt/umami/fat', 'sweet/fat' and 'bitter'). This database was combined with dietary recall data from two independent observational studies in adults in the Netherlands.[13] Results revealed gender differences, where women consumed significantly more %energy from 'sweet/fat' and 'sweet/sour' foods than men. The relationships between body mass index (BMI) and the consumption of sweet foods were not consistent between surveys, but %energy from 'salt/umami/fat' foods was slightly higher in obese than normal weight individuals in both men and women in both surveys ($P < 0.05$), which agrees with previous findings.[14] de Graaf concluded that sweetness preference and intakes vary with age and gender, but do not appear to vary with weight status. Although sugars intake has increased since our hunter-gatherer predecessors, it may have stabilised, or fallen, in some countries. However, sugars intake may not reflect the sweetness of the diet. The development of standardized taste scales and food sensory databases will help to advance and consolidate the research in this area.

Sweetness and diet quality. Gibson proposed that sweetness could affect diet quality if sweet tasting diets were intrinsically nutrient poor, or if preference or appetite for sweetness encouraged consumption of less nutritious foods. It was noted that several indices of diet quality exist, based on a variety of subjective and objective nutrition criteria, making comparisons difficult.

Most data relate to the intake of sugars or use of LCS within diets, and do not address whole-diet sweetness. Evidence from observational studies provides support that diets which are very high in free or in added sugars have lower nutrient density (mg/MJ), though not necessarily absolute nutrient amount,[15] and tend to score lower on diet quality.[16] Such associations are not seen for total sugars, because intrinsic sugars are positively associated with diet quality.[17] This suggests that diet quality is not a function of sweetness but the selection of less nutritious sugar-containing foods. Sweet foods and beverages appear to be distributed among different dietary patterns,[18] rather than a single 'sweet' dietary pattern. Evidence that LCS-beverage consumption specifically is associated with higher indices of diet quality has been shown in some studies,[19 20], but not others.[21]

Data from intervention trials appear to support that maintaining sweetness in the diet via replacement of sugar-sweetened beverages (SSB) with LCS beverages does not seem to induce compensatory consumption of sweet foods.[22] A behavioural intervention to reduce SSB intake alone was shown to spontaneously induce other favourable dietary changes such as lower consumption of sweet coffee and increased intake of vegetables and whole grains.[23]

Gibson concluded that the evidence for a need to reduce the (non-sugar) sweetness of the diet was not compelling from a diet quality perspective, although a reduction in the intake of foods and beverages high in free sugars and low in nutrients may improve some markers of diet quality. Use of LCS does not appear to induce compensatory intake affecting diet quality. However, longer term studies need to examine effects of unsweetened versus LCS-sweetened diets, and of low sugar versus usual diets, on food choices in real life settings, with detailed measures of consumption. In addition, there is a need to establish if and how hedonics relate to actual consumption.[8] Other considerations include: the level of sweetness reduction, the vehicles for sweetness, individual differences, culture and context.

Exposure to dietary sweetness with calories: is there a learned association, and does sweetness without calories impact food intake patterns and energy balance?. Rogers noted that some authors have contended that exposure to sweetness without calories undermines sweetness as a cue for the learned control of energy intake, and that this risks increasing energy intake and body weight.[24] It is well established that animals can learn associations between flavour cues and post-ingestive consequences of nutrients, which in turn guides food choice and intake.[25] However, the evidence from studies on rats used to support the disruptive effect of sweetness without calories on appetite and weight control[24] has recently been cast into doubt.[26] In any case, in human diets, while sweetness predicts the sugar content of foods, it does not predict energy content.[27,28] Therefore, irrespective of the presence of sweet-tasting products without calories, sweetness *per se* may not be a useful cue for controlling energy intake. Still, there is a need to more fully understand the role sweetness may play in learning the nutritive value of consuming food at different life stages.

The effects of sweetness without calories (that is, LCS) on energy intake and balance in humans have been reviewed recently in a meta-analysis.[29] Preload test-meal studies support a reduced cumulative energy intake (preload plus test meal) for sweet preloads without versus with calories (sugar), and no difference for LCS preloads versus water. In sustained intervention trials, when comparing effects of diets which provided sweetness with (sugars) and without (LCS) calories, the consensus was a relative lower body weight in adults and children consuming LCS, most likely due to incomplete compensation for the lower dietary energy content achieved by consuming LCS in (partial) replacement of sugar. A relative lower body weight may also be apparent for sweetness without calories versus water, possibly because sweetness without calories helps satisfy desire for sweetness. However, more research is needed in support of the latter hypothesis. In any case, it is clear that substitution of sweetness without calories for sweetness with calories helps reduce energy intake. Sweetness without calories does not appear to increase energy intake (or body weight) compared to water.

Session 3: Health aspects of sweetness: does sweetness *per se* affect health?

Sweetness and chronic disease risk. Prior to examining the evidence on sweetness and health, Mattes proposed an integrative over a reductionist approach to future research. Because multiple internal and external factors impinge on the various determinants of food and beverage choices and consumption, it is improbable that one facet of taste sensation explains a substantial percent of the variance in chronic disease risk. Opposing purported mechanisms are often cited to explain observed relationships between dietary sweetness and body weight with associated changes in health outcomes. When sensory responses (for example, threshold, scaling, hedonic) are low, authors suggest there may be a compensatory increase in sweetener/energy intake to achieve a desired level of sensory stimulation. Alternatively, when higher sweetness indices are observed, the proposed explanation is that the sensory stimulation is rewarding and thereby promotes an increase in energy intake. Neither 'mechanism' has been validated at more than a descriptive level so both should be viewed as speculative.

The preponderance of evidence reveals no significant association between sweetness recognition thresholds, intensity ratings, or quality recognition and BMI.[30–33] Differences in taste function related to BMI are commonly generalized across multiple taste qualities indicating no sweet-specific disorder.[34,35] In addition, preference for sweetness has been positively,[36] negatively[30,36] and not associated.[31,32,35] with BMI Individuals with diabetes generally exhibit decreased taste responsiveness for all taste stimuli[37–40] with greater declines in individuals with uncontrolled diabetes or longer duration of the disease. This is consistent with an effect of peripheral neuropathy on taste responses[41] and not a specific defect in sweetness responsiveness. Again, most studies suggest sweetness preference is not different between individuals with diabetes and those free of the disease.[42,43] Therefore, there does not appear to be a causal relationship between indices of sweetness and the risk or manifestations of either obesity or diabetes.

Sweetness and glycaemic regulation. McLaughlin focused on gut-mediated effects, as it has been recently reported that human enteroendocrine cells express sweet taste receptors (STRs), the function of which is unknown.[44] Although increasing hexose sugar concentration slows gastric emptying dose dependently, equisweet solutions of various LCS do not appear to exert the same effects.[45,46] Therefore, sweetness *per se* does not appear to mediate gastric emptying. Intragastric administration of lactisole, an antagonist at the STRs, prior to a glucose infusion blunted postprandial glycaemic responses,[47] though results were not replicated in later studies where lactisole was administered simultaneously with glucose.[48,49] These results suggest that gut STRs exert no major acute effects on glycaemic regulation. However, in studies of gut peptide hormones, lower GLP-1 and PYY but not CCK release has been shown following lactisole.[48]

Acute effects of LCS consumption have been evaluated in numerous human studies, but varied in delivered dose and methodological designs. Despite the demonstration that LCS might trigger glucose absorption and gut-peptide release in rats through activation of STRs,[44,50] the majority of human studies find that consumption of LCS either alone[51,52] or combined with glucose[53,54] does not exert a major influence on postprandial glucose, insulin or gut-peptide responses, at least in lean subjects.

Data derived from human clinical trials are not consistent with the results from animal studies and human cell lines. Differences may reflect the inadequacy of models to analyse human gastrointestinal tract and neuroendocrine responses, or doses which may not be relevant to human consumption. The functionality of human gut STRs remains unclear. Acute consumption of LCS does not seem to have a major effect on glucose and hormonal responses. Evidence for chronic effects is lacking and clarity on any putative effects requires well-designed randomised controlled clinical trials evaluating gut-related effects of 'nutritionally' relevant LCS doses in humans.

MAIN CONCLUSIONS

- Much of the current evidence on the effect of exposure to sweetness on dietary behaviour is based on exposure to sweet elements in the diet rather than the relative sweetness of the whole diet.
- Sugars consumption has changed drastically since the early diets of our predecessors. However, sugars intake may have become more constant in recent years (last decades) at least in some countries, and fallen in others.
- Observational evidence exists for tracking of estimated consumption of sweet elements in the diet, particularly in childhood.
- Sweetness itself does not appear to affect diet quality, except where food choices result in a high intake of free sugars, which may lower nutrient density. It was proposed that, in naturally occurring foods, sweetness is mainly indicative of the sugars rather than the energy content of foods.
- Cross-sectional studies on low-calorie beverage consumption reveal some evidence of improved diet quality among LCS consumers compared to consumers of sugar-sweetened beverages. Intervention trials generally find that sweetness without calories reduces energy intake when it replaces sweetness with calories.
- There is no apparent relationship between single measures of taste perception or preference and BMI or type 2 diabetes.

SUGGESTIONS FOR FUTURE AVENUES OF RESEARCH

- Future research should adopt a whole diet or dietary patterns approach, using standardised databases which combine complete nutritive and sensory data on foods and beverages, with improved methods of estimating consumption, and which examine tracking through important periods of maturity.
- Further research is needed to more fully understand the role sweetness may play in learning the nutritive value of consuming food at different life stages.
- A reductionist approach has been employed in many of the studies to date, for example, relating measures of taste perception to disease risk. However, a multifactorial integrated approach may better address outstanding research questions.
- There is a need to understand the relevance of sweet taste receptors in the gut and elsewhere in the body.
- The effects of chronic intake of sweeteners on metabolic responses need to be examined; including the effects of individual sweeteners and employing realistic doses.
- More randomised controlled intervention trials are warranted to understand the effects of reducing sweetness in the diet (caloric and non-caloric sources) on the dimensions of sweetness, and the persistence thereof.

- A pre-requisite for all research studies is that they are appropriately powered, undertaken in different population groups, and with better controls and measures.

CONFLICT OF INTEREST

AW has previously worked for the World Sugar Research Organisation, and PepsiCo. NSS has received a BBSRC DTP CASE studentship funded by Cargill. GKB receives no personal funds from any private company. Ajinomoto provides a consulting fee to the Monell Center that is used to support a small portion of GKB's research program. All of GKB's published work described here was supported by the NIH or other US federal granting agencies, except for one study (Wise *et al.*) which was supported by PepsiCo conducted through an investigator-initiated grant. AB is employed by Cargill. Over the past 3 years, SG has received research funding from: Sugar Nutrition UK, PepsiCo, Coca-Cola, Cereal Partners Worldwide, Nestle and European Soft Drinks; and has received honoraria/travel from the International Sweeteners Association, PepsiCo and UNESDA. CdG received research funding from the Netherlands Sugar Foundation for a study on the brain response to the taste of sugars and low-energy sweeteners, published in *Neuroimage* 2011; CdG received honoraria/travel from the International Sweeteners Association, Mars, Pepsico, Kellogs; CdG is a member of the Scientific Advisory Board of Sensus, and received research funding directly or indirectly through NWO (Netherlands Organization for Scientific Research) or TIFN (Top Institute of Food and Nutrition) from Unilever, FrieslandCampina, Nestle, Heineken, Danone and Mars. JCGH has received research funding from the American Beverage Association, Astra Zeneca and Bristol Meyers Squib and has been an advisory board member for Novo Nordisk and Orexigen. RDM is an advisory board member for ConAgra and the Grain Foods Foundation, has received research support from the Almond Board of California, the California Walnut Commission and Ajinomoto, and has received multiple speaking honoraria. JM has received funding from the BBSRC, including a CASE PhD studentship funded by Cargill. DJM is employed by Unilever. Over the past 5 years, SN has received funding for research from Blédina S.A. (a branch of Danone Early Life Nutrition), received speaker's fees from Danone Global Affairs, Nestlé France, and from the French Beverage Alliance (Unijus) and provided consultancy service for Nestlé Research Center. SN is currently Co-Executive Editor of Appetite. PR has received funding from Sugar Nutrition UK, provided consultancy services for Coca-Cola Great Britain, and received speaker's fees from the International Sweeteners Association and the Global Stevia Institute. IAM has received grants/research support from Unilever, Mars and the UK Government and is on the Scientific Advisory Board of Mars, IKEA and Nestle. He is also involved in peer review work for Waltham Centre for Pet Nutrition and was in the speakers bureau of the UK Nutrition Society, UK Association for the Study of Obesity and Federation of European Nutrition Societies; is a member of the UK Scientific Advisory Committee on Nutrition, and he is also the Editor of *IJO*. The remaining authors declare no conflict of interest.

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