Sweetness

Sweetness is one of the so called basic tastes (sweet, sour, salty, and bitter). In other words, it is a gustatory experience devoid of olfactory or tactile elements. It is also hedonic and attractive, though this depends on the concentration of stimulus. Although most of the sweetness in our foods comes from sugars (mainly sucrose) and is therefore nutritive,¹ several thousand non-nutritive sweeteners are now known. The advantage of these latter types is that they are metabolically inert or make an unappreciable contribution to energy intake. Some are therefore useful for dietetic or diabetic foods and cheap (on a sweetness basis) compared with sugars. Sugars provide bulk, mouthfeel, and osmotic pressure (preservation power) in foods. Legally in the United Kingdom, however, they are not classed as sweeteners. Since 1983, 12 sweeteners are permissible for use in foods in the UK (Table),² divided into six bulk sweeteners—that is, hydrogenated sugars or polyols—and six intense sweeteners.

The bulk sweeteners are of about the same sweetness as sucrose (cane or beet sugar), whereas the intense sweeteners are hundreds or thousands of times sweeter. A recent conference organised by the International Life Sciences Institute at Geneva was devoted to chemical, psychophysical, sociological, and dietary aspects of sweetness.³

Although the desire for sweetness may be enhanced by overindulgence, the recognition of sweetness and its enjoyment seem to be inborn. Steiner has shown that human neonates display positive—that is, a relaxed satisfied smile—gustofacial responses to sweet solutions, easily distinguished from the responses to other basic tastes.⁴ Engen points out that taste elicits more definite hedonic reactions than odour in the newborn, as well as in children 3–7 years old.⁵ It is therefore interesting that, although

olfaction is normally considered to dominate gustation in flavour perception, taste may emerge as an earlier signal of what is ingested. The human infant is equipped with taste buds located in the tongue papillae, and some of these are present on the lips and even possibly the cheeks. Those outside the oral cavity decline in numbers with increasing age of the child; each taste bud contains about 60 taste cells on which are the receptors for all four basic tastes. The life span of a taste cell is about 10 days.

Basic chemoreception of sweetness

Sweet molecules may be either large—for example, proteins—or quite small—for example, simple sugars, polyols, and certain salts. Sweetness is thought to originate in a loose binding of a stimulus molecule with a receptor. The mechanism of this binding is probably hydrogen bonding, and a steric fit of stimulus molecule with receptor protein is therefore a necessary preliminary to an action potential at the taste neuron. Why then do some molecules taste thousands of times sweeter than the sugars? The answer seems to be twofold. Either the intense sweeteners accede better to receptors or they activate them better due to a better steric fit. Some molecules may do both, and a clue to the mechanism of sweet chemoreception comes from the peculiar temporal properties of many modern sweeteners. Intensely sweet substances seem to have a slow 'reaction time'—that is, time for the onset of sweetness sensation after tasting—and a pronounced persistence—that is, duration of taste. The new protein sweetener thaumatin, for example, persists for 20 minutes or more and is therefore quite unsuitable for use in foods as such. On the other hand, it is useful in chewing gum and medicines where it effectively masks other unpleasant tastes. Persistence is very likely caused by some organised, localised concentration of stimulus molecules at or near to the receptor. The sensation of sweetness then continues until these localised concentrations become depleted.^{6,7} It is now possible to enhance, abolish, or create sweetness by chemical means. Hough has recently reviewed the chemical aspects of sweetness.⁸

Taste dysfunction and disease

Henkin *et al* have listed four major causes of taste (and smell) dysfunction.⁹ These are:

Table Permitted sweeteners² in the United Kingdom

Sweetener	
Intense	Bulk
Acesulphame potassium	Hydrogenated glucose syrup
Aspartame	Isomalt
Saccharin	Mannitol
Sodium saccharin	Sorbitol
Calcium saccharin	Sorbitol syrup
Thaumatococcus	Xylitol

*Aspartame should not be used for patients with phenyl ketonuria.

- (1) post-influenza hyposmia (diminished sense of smell) and hypogeusia (diminished sense of taste);
- (2) idiopathic causes;
- (3) head injury;
- (4) allergic rhinitis.

Evidently hypogeusia or hyposmia, or both, are much more common in hospital patients (19% of sample tested) than dysgeusia and dysosmia (2%). Environmental or accidental exposure to lead, mercury, or cadmium (or parenteral administration of gold) has been associated with hypogeusia, hyposmia, dysgeusia, or dysosmia. Also decreased concentrations of body metals, particularly zinc and copper, produce severely decreased or disordered taste and smell function. It is therefore not surprising that the copper depleting drug, D-penicillamine, has been associated with the production of hypogeusia. Thiol containing compounds tend to inhibit taste function but several metal ions—for example, zinc, nickel, and copper—tend to restore normal functions. L-Histidine has the same effect. About two thirds of patients with taste and smell dysfunction do not exhibit zinc deficiency but, in those who do, treatment is consistent and takes about two to four months. After treatment with zinc patients usually note that the taste and smell of sweet is the first quality that returns toward normal and bitter is the last. In patients who do not exhibit zinc deficiency cyclic adenosine monophosphate may be involved. It is found that the drug aminophylline improves their hyposmia but not their hypogeusia.

Cancer may affect taste function, and Galili has pointed out that oropharyngeal irradiation produces devastating effects on gustatory perception.¹⁰ Patients with diabetes also show impairment of taste, though this may be specific for some sugars. Thus high thresholds for D-glucose and D-fructose have been reported in adult onset diabetes. This does not seem to affect other basic tastes, and there is apparently no correlation between blood glucose concentrations and taste thresholds. Patients with juvenile onset diabetes display no taste alterations.¹⁰ Patients with renal and hepatic disease may also show taste dysfunction, and sweet nausea may create a problem in such patients when an essential soluble energy intake is demanded. In these cases glucose syrups¹¹ constitute a useful alternative to

sugar and their hydrogenated forms¹² are of particular value to patients with diabetes.

Conclusions

Sweetness seems to be an inborn pleasurable sensation, but the chemical basis of sweetness is not yet fully understood. Medical observation of taste dysfunction will contribute to our understanding in this respect and, in the meantime, 12 sweeteners are permitted for general use in the UK, in addition to the normal food sugars, which are not regulated. Although many different classes of chemical cause sweetness, there is probably one molecular feature (a 'glycophore') common to them all. Elucidation of the mechanism of taste chemoreception is the goal in this field of endeavour.

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