proteinspotlight
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Issue 55, February 2005
www.proteinspotlight.org

The taste experience

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To what end do we need to taste? Just to enjoy a night out at the restaurant? Fish can also taste. But they do not go out for dinner. We – like all animals – taste because we have to be able to distinguish between what is good for us, and what is not. Nowadays, things are easy. All you have to do is saunter down the road to the nearest supermarket and pick out what you need. Most of us can read, and relate the word 'tomato', 'steak' or 'chocolate' to a taste. Thousands of years ago, though, there were no supermarkets (or chocolate) and our ancestors could only rely on their taste buds. As a consequence, they learned that sweet-tasting foods were probably edible, whilst bitter ones were probably not, because – more often than not – bitterness spelled poison. And the distinction we are able to make between sweet and bitter resides in taste receptors which are lodged in the recesses of our taste buds.

Nature has granted us five fundamental sensations – sweet, bitter, sour, salty and umami – the combination of which we call 'the taste of something'. Different tastes demand different types of receptor, and scientists are only beginning to untangle the complexities involved in their perception. To date, they have tracked down three receptors that seem to be directly involved in our perception of sweetness along with the fashionable umami taste[1]: T1R1, T1R2 and T1R3, which combine to form heterodimeric G-protein coupled receptor complexes. Which is hardly surprising since proteins of this sort are also known to play key roles in the pathway we use to perceive smells for example.

Taste receptors are lodged in the membranes of taste cells, which are found by the hundreds in our taste buds. Relays to our brain are then made via sensory nerve fibres. T1R1, T1R2 and T1R3 are about 800 amino acids long and wind in and out of each cell membrane seven times, where they form twosomes: T1R1 with T1R3, or T1R2 with T1R3. The T1R1/T1R3 dimer helps us to perceive sweet tastes whereas the T1R2/T1R3 dimer triggers off the perception of the umami taste. And without T1R3, the sensation of sweetness would be tasteless, so to speak. T1R3 has an amino terminal glycosylation site which – were it to be used – would add an appendage which in turn would interfere with T1R3's coupling to either T1R1 or T1R2. Consequently, this glycosylation site may have some kind of regulatory function in the taste experience.

Tongue Wags, Temple Parker

Courtesy of the artist

How do flavours bind to their receptors? There are 'small flavours' and 'larger flavours', i.e. small chemical entities such as carbohydrates or larger macromolecules like proteins. Small flavours probably slip into pockets formed by the heterodimers, thus triggering off the sensation of taste. Larger flavours – too big to squeeze into such pockets – may display 'sweet fingers' which do fit in neatly. However, this cannot account for the great increase in activity sparked off by a large sweet molecule, which can be as much as 100'000 times sweeter than the sensation procured by a smaller one! An

[1] See issue December 2001

altogether different mechanism must be involved. And what has been suggested is that larger molecules dock to the taste receptors via large interacting surfaces, which would not only stabilise the whole complex but would also account for the long-lasting and greater sensation of sweetness.

Sweetness comes in all shapes, sizes…and sweetness – some things taste sweeter than others. How do we make the difference? How can we distinguish a chocolate bar from a slice of chicken, for instance? Our tongue is an orchestra of receptors, which are there to distinguish between sweet, sour, bitter, salty and umami. When you eat a slice of salami, hordes of different molecules are released onto your taste buds firing off concertos of sensations, the combination of which will make you perceive something definitely on the sweet side though the background noise is filled with sensations of saltiness, perhaps even sourness, a little bitterness and who knows some umaminess.

Over the millennia, the mechanisms underlying the perception of taste have been fine-tuned for the purposes of survival. Sweet tastes, for example, are attractive because it usually means that we are consuming carbohydrates which are essential to the organism.

Apart from the academic desire to acquire knowledge on how and why we taste, the prospects of such research are particularly appetising for those immersed in food flavouring. Taste receptors can be switched off – or on – by designing molecules which mime the receptors' natural ligands. Nature has also devised her own little fib, since the sweet protein miraculin can trick the mind into believing that something bitter is in fact sweet[1]. The commercial prospects behind food flavouring are obvious but there are also serious illnesses related to sweetness, such as diabetes or hypolipemia – an abnormally low concentration of fats in the blood – that could also benefit from research in drug design, which could in turn offer those suffering from them a sweeter future.

Cross-references to Swiss-Prot

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Sweet taste receptor T1R2, *Homo sapiens*, (human) : Q8TE23
Sweet taste receptor T1R3, *Homo sapiens*, (human) : Q7RTX0

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Protein Spotlight (ISSN 1424-4721), http://www.proteinspotlight.org, is published by the Swiss-Prot group at the Swiss Institute of Bioinformatics (SIB).