

*The Transduction of Sweet Tastants in
Humans*

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ABSTRACT

This literature review will explain the variables that affect the taste system. This review will explain the interactions of sugar with bitter, salt and fat in the taste cell. The different transduction mechanisms will also be illustrated within this review. Differences of intensities and threshold will be displayed and explained through experiments in various research articles. The effect of Sweeteners will explain the concept of synergy. Prop Sensitivity will also be investigated, from this investigation the three types of tasters will be defined. Sweet taste transduction in humans is also explained in more detail by experiments that received similar result in animals. This generalization leads to the belief that taste systems and mechanisms in lab animals are similar to those in humans.

INTRODUCTION

Humans are suited with five basic senses that aid in survival. Through these five senses, humans are able to incorporate separate signals and use them as cues of our physical environment. Taste is one of these basic signals that is used to warn the body of toxins and other harmful substances as well as necessary nutrients that the body may encounter.

The tongue is the body's taste instrument. It can sense a single taste stimulus or a variety of taste stimuli and send neural signals about the presence of these stimuli to the brain. The taste system is able to produce neural signals by way of buds on the tongue. This process of taste transduction occurs in the taste buds that are comprised of taste receptor cells. These receptor cells sense the presence of chemicals on the tongue through different mechanisms whose overall effect produces intracellular changes within the cell. These intracellular changes result in depolarization and then release of neurotransmitters. These neurotransmitters are then received by three nerves, the chorda tympani, greater superficial petrosal, and the glossopharyngeal nerves that transmit the neural signals to the nucleus of the solitary tract where they converge in the brain stem. The signals

proceed to areas of the cortex where they are labeled as the qualities as sweet, sour, salty or bitter.

There are individual differences in taste perception and humans have been grouped into types of tasters; non-tasters, tasters and super-tasters (Drewnowski A *et al*, 1997). This classification is based on the ability to taste or not to taste Propylthiouracil (PROP). Non-tasters have a low ability to detect the PROP solution. Tasters detect the PROP solution more intensely than non-tasters, with the detection threshold is much lower for tasters than non-tasters. Super-tasters can taste the PROP solution even more intensely and have the lowest detection threshold. Super tasters also have a greater general taste sensitivity than the tasters or non-tasters. One important difference between the types of tasters is the number of taste receptor sites. Therefore humans with more taste receptor sites have higher detection capabilities for sweets and other tastants compared to those with less receptor sites.

Taste Transduction in Humans

Taste transduction is the process of a physical chemical stimulus becoming a neural signal. Taste transduction occurs through two main mechanisms, ion channels and metabotropically. Activation of ion channels are usually perceived as a salty or sour taste. Metabotropic transduction is usually perceived as a sweet or bitter neural taste.

Ionic transduction occurs when salty chemicals in food or drink enter the taste cell and depolarize the cell by making the cell more positive. Sour chemicals produce depolarization by blocking potassium(K⁺) ionotropic channels in the taste cells. These increases in voltage make the cell more positive until a specific voltage is reached at which neurotransmitters are released. Threshold is the level at which neural signals can be first produced.

Metabotropic transduction is a lock and key system that recognizes sweet or bitter chemicals in food or drink. These chemicals bind to metabotropic binding sites on taste receptor cells, causing the release of a G-protein. The G-protein then leads to the release of a neural signal. There has been evidence for taste cell specific G-proteins(3c).

Bitter tastants are transduced by metabotropic transduction. The taste of bitter is usually an undesirable taste at very high concentrations. Potassium (K+) channels are key in the transduction of bitter tastants. It can be concluded that these channels are key because bitter tastants are not as readily detected when these channels are being blocked. The bitter taste was either weaker or not detected when presented to the taste cells because the neural transmitter was not being released or being less frequently (Kinnamon SC, Getchell TV, 1991)

Sweet taste is modified by chemicals within the cell (Shiffman SS *et al*, 1994). These modifiers are GDP (guanosine diphosphate)GTP (guanosine triphosphate) cAMP, amilliride. Sweet taste transduction has many components responsible for G-proteins and neural signal to be released. Adenylate Cyclase has been implicated in the transduction of the sweet taste. Adenylate Cyclase is a

second messenger system (G-Protein) that uses cAMP. cAMP is an intracellular messenger that is elevated when sweeteners bind to cell surface receptors. Simultaneously when cAMP levels are elevated potassium channels are inactivated in the cell (Shiffman SS *et al*, 1993). This binding process causes GDP to move from the binding site to be replaced by GTP. GTP in turn binds with another chemical then binds to the adenylate cyclase. PKA (protein kinase) is the end product of this series of binding, which depolarizes the cell (Shiffman SS *et al*, 1994).

Sweeteners and inhibitors of the sweet taste were manipulated in pigs and in gerbils. The findings were concurrent with the findings of previous experiments. Adenylate cyclase can enhance or inhibit the perception of sweet tastants in both. Some of the modulators made the sweet tastant more sweet and others made the tastant less sweet. The author thought that these findings would be helpful humans that suffer from hypogusia because of its sweetening effect (Shiffman SS *et al*, 1994). The experiments in pigs and gerbils have similar findings allowing the inference to be drawn that the transduction mechanism works similarly in most animals.

Influence on the Perception of Sweet

Interactions of Bitter and Sweet

Bitter and sweet tastants compete with each other due to their proximal relationship on the taste cell. Bitter and sweet are both transduced metabotropically on the apical portion (receptor level) of the tongue. A less intense taste perception is detected due to this competition in the taste cells. Hyman & Frank found that the chorda tympani nerve response was smaller in mixtures of the sweet and bitter compounds opposed to when they are presented separately (Schiffman SS et al, 1994). This is because one stimulus with the absence of the other would have a stronger effect on a taste receptor cell due to the lack of competition. This provides evidence for the theory of competition.

Sweeteners have a greater effect on bitters that are supra-threshold. The higher the sweet intensity the bigger the impact the sweetener has on the detection level of the bitter compound. The intensity of a sweet concentration is rated much higher than most bitter concentrations at threshold. Sweeteners at threshold for certain bitter concentrations effected some bitter concentrations, but most were not.

Concentration of a sweetener is important to the taste quality of bitter. The higher the concentration of a

sweetener in a high potency sweetener, the bitter perception becomes more intense. A single compound can have both sweet and bitter qualities. Small structural changes can alter the sweet and bitter intensities (Shiffman SS *et al*, 1994). Quinine (bitter) and sucrose (sweet) is perceived as less sweet than the unmixed solution equimolar in sucrose and less bitter than a solution equimolar in quinine. It is believed that the brain sums the taste perceptions in its peripheral areas. These perceptual interactions of sweet and bitter tastants have been attributed to a peripheral phenomenon. This phenomena occurs due to neural inhibition at the receptor level of the tongue (Shiffman SS *et al*, 1994). High potency sweeteners tend to be more bitter with increasing concentration suggest that the same chemical properties that emit bitter qualities play a role in perceived potency. That is further illustrated when bitter perception is increased in sweet solution depending on the sweet potency. Bitterness ratings tend to decrease with concentration for sweeteners that were considerably less potent than sucrose. Studies have shown similar inhibition in chimpanzees because of nerve fibers responded to some sweeteners but did not respond to bitter qualities.

Interaction of Salts with Sweets

Selective inhibition occurs through the transduction of sweet neural signals by blocking channels with a sodium salt compound. Some compounds have been found to inhibit the sweet taste in humans. Na-PMP is 2-(4-Methoxyphenoxy) propanoic acid is one compound that has been found to inhibit the sweet taste in humans. Another example of selective inhibition comes from studies of bitter and sweet tastants (Schiffman SS *et al*, 1999). It is proven that these taste properties are independent of each other because single papillia produced bitter sensations from quinine salt, but didn't respond to sweet stimuli. A separate experiment was conducted to determine the effect of Na-PMP on sweeteners in humans. The experiment shows that Na-PMP had an inhibitory effect on all but three of the tastants in the experiment. In another study from the same experiment Na-PMP enhanced the taste of sweet when a 30 second pre-rinse of the Na-PMP solution was administered prior to exposure to the tastant. The data showed that some sweet tastants were excited and somewhere inhibited by Na-PMP. From this the conclusion is drawn that selective inhibition is occurring. The notion

of more than one transduction mechanism in the taste cells comes from this data. It can be concluded that the sodium salt compound Na-PMP is an inhibitor of sweet taste. The findings also illustrate that there must be at least two sweet receptors or mechanisms because different responses were elicited for similar tastants.

Interaction of Fat with Sweet

Fat and sucrose come together and form a taste that is pleasing to the senses (Warwick ZS, Schiffman SS,1990). High concentrations of fats in liquids are not pleasing to the senses alone, but once sucrose is added to the liquid-fat solution the taste gets better. The more sucrose that is added the better the taste of the fat concentration. This more pleasant taste leads to overeating. The overeating of these high fat foods lead to obesity in humans (Warwick ZS, Schiffman SS,1990).

Influence of Synergy with Sweet

The intensity of stimuli can be increased without increasing its molarity by combining a similar stimulant. This process is called synergy. Synergy can occur in two different ways. The first way being that when the taste of

two tastants come together to form an intensity level higher than the sum of both tastants. Ex fructose would have an intensity of 4 and sucrose would have an intensity of 3. This compound has synergy when the intensity level is > 7 .

The second type of synergy is when two tastants come together to give a higher level of intensity than when tasted separately. (ex.) Salts intensity is rated at 5 and sugar's intensity is rated at 2. Synergy is when the two salt and sugar form a combined intensity > 6 . It is assumed that enhancement occurred at the receptor level.

When three tastants (Traids) are mixed then synergism is reduced (Shiffman SS *et al*, 1981). These ternary combinations do not have the same effect that binary combinations have on sweeteners. Two factors that effect binary combinations is the presence of a high potency sweetener in a mixture. The second factor is the concentration of competent sweeteners in the mixture. The less concentration of a tastant, the higher the synergy will be. This fact mimics the effects of pre-rinsing with a sub-threshold substance. The lower concentration makes the cell more positive requiring less of the next stimuli to cause a neural signal release.

Temp

It has been found that many things can affect the sweetness in taste stimuli (Shiffman SS *et al*, 2002). Temperature has been found to have a statistical difference in sweeteners. Heat has been found to enhance sweet stimuli while sweetness is significantly suppressed at cold temperatures.

PH

The pH had no statistical difference in the effect on sweetness intensity in humans (Shiffman SS *et al*, 2002). The pH levels were tested at five different levels. Sourness and astringency increased as the pH decreased with concentration.

Ions were thought to have a significant effect on perceived sweetness intensity (Shiffman SS *et al*, 2002). A study was conducted and no significant effect was found between the groups by the addition of ions. The sweetness intensity of sodium saccharin and sucralose had a longer delay to the onset of maximum sweetness intensity(Shiffman SS *et al*, 2002).

AGE

The bitter compound is transduced metabotropically through receptor sites on the taste buds of the tongue. When K⁺ ion channels are blocked by H⁺ ions on the apical

portion of the tongue the cell then becomes depolarized. The cell then becomes so positive that it signals a neural transmitter to be release. Fungiform pappilia are found on the anterior two-thirds of the apical portion of the tongue. They are a key aspect of the detection of bitter. This study also attributes the differences to the changes in the hippocampus and the amygdala. Another aspect of older people's lower detection level is that memory loss may make judgment more difficult on the magnitude scale harder. The difficulty is that older people forget the previous intensity level and fail to have anything to compare to the latest stimuli. (**FIX** NO SWEETENERS LOWERED

DETECTION OF THE THRESHOLD (SCHIFFMAN SS *ET AL*, 1994). SWEETENERS HAD NO EFFECT ON DETECTION THRESHOLD IN OLDER PEOPLE.) HUMANS HAVE SHOWN THAT THERE IS AN EFFECT ON AGE AND WEIGHT IN THE DETECTION OF FAT-SALT AND FAT-SUCROSE MIXTURES. SOLID FAT RICH STIMULI ALSO INCREASE IN PALATABILITY WHEN SUCROSE IS ADDED (WARWICK ZS, SHIFFMAN SS, 1990). IN THIS STUDY THE MEAN AGE OF THE YOUNG GROUP WAS 22.4 AND THE ELDERLY PERSONS MEAN AGE WAS 82.3. THE CRITERION FOR OVERWEIGHT PEOPLE IS 15% OVER THE IDEAL WEIGHT ACCORDING TO THE METROPOLITAN LIFE INSURANCE TABLE IN 1983(WARWICK ZS, SHIFFMAN SS, 1990).

A loss of bitter sensitivity can be seen with the increase of age. It is assumed that the loss of bitter sensitivity can be attributed to less fungiform papillae in older people. The loss is greater in non-tasters than it is in tasters. Threshold for older people is also higher than it is in younger people. Reasons for lowered detection level in elderly people can be attributed to lower levels of estrogen and testosterone, which leads to a decrease in receptor cell turnover rate (Schiffman SS *et al*, 1981).

The bonding of hydrogen atoms is also believed to have an effect on the perception of sweet taste (Shiffman SS *et al*, 1981). These different bonds allow for artificial sweeteners of similar chemical makeup to be made.

Artificial sugars allow the sweet taste of sugar without the some of the harmful effects. Artificial sweeteners have also been thought to cause mutagenicity. From these mutations in the body harmful effect occur such as cancer. It is believed that older people would get the brink of the mutations due to their diminishing taste threshold. The older person would have less fungiform papillae causing an overall lower detection of artificial sweeteners. It has been found that detection of artificial sweeteners in younger people is much greater than older people (Shiffman SS *et al*, 2002). Detection was different but the rank

orders in intensities were not different. Both groups young and old ranked the tastants in the same order.

In young, overweight people a significant effect was found in sucrose, but no significant effect was found in fat or fat-sucrose interaction. The fat-salt mixtures show that in younger obese subjects illustrates salt and fat combined had a significant effect on dietary intake (Warwick ZS, Shiffman SS, 1990).

In elderly people both overweight and normally weighted subjects detected palatability in fat-sucrose mixtures (Warwick ZS, Shiffman SS, 1990). A significant effect was not found in the fat or sucrose content in the mixture, nor to the interaction of fat and sucrose. The salt content was related to preference in fat-salt mixtures in older subjects. Fat content had no significant effect on fat-salt mixtures. The author thought that the reason for discrepancies in older and younger subjects was due to the discrimination of the various viscosities, rather than cues that are from sight or smell (Warwick ZS, Shiffman SS, 1990). It was found that fat has an effect on pleasantness in fat-sucrose mixtures in older people. The quantity of salt has the effect on fat-salt mixture in older people. In contrast, the interaction of fat-sucrose makes food more pleasant in fat-sucrose ratings in young people. Fat and

salt concentration cause a more pleasant perception of stimuli in fat-salt mixtures. It is assumed that no effect was found within either fat-salt or fat-sucrose interactions because both experiments were conducted suprathreshold.

WEIGHT

In normal weighted people fat-sucrose mixtures have a significant interaction. There is not a significant interaction with either sucrose or fat in conjunction to dietary intake. A significant effect was shown that preference for fat-salt mixtures depends on the concentration of salt and fat. There was not a significant effect between the fat-salt interactions in normal weighted people.

RACE

In previous studies African Americans were found to have had a greater preference for sweeter and saltier liquid solutions and food than adults and white children their age (Bacon AW *et al*, 1993). This study found that African Americans preferred more sweetness in fruit drinks. There was no difference in preference between the two races for bacon and Pork bellies (Bacon AW *et al*, 1993). One peculiarity was that all children tested lived in the

south, but some of the white parents were born in the Northern US. The difference may be from the child's upbringing and what the child is accustomed to eating in the home.

ERRORS IN TASTE

Adaptation is another phenomena in taste systems. A long lasting constant stimulus rapidly reaches its peak then gradually fades. Adaptation is also readily seen in the after effects of a long lasting stimulus, in that the sensitivity to that stimulus is lowered. This phenomenon occurs even when two separate tastants are transduced. (Froloff N et al, 1998). This is further proof for cross-adaptation because the taste stimulus is from different taste stimuli, but is binding to one taste receptor.

CONCLUSION

In conclusion, from these numerous research articles it can be concluded that the experiments conducted were very useful in the improvement of knowledge in the research of taste. These taste experiments have shown conclusively that many of the different systems and mechanisms are consistent throughout many of the taste systems. It has also shown that variables can change the perception of sweet. The information has been gained from the

experiments in lab animals and humans have advanced our knowledge of the transduction of the sweet taste tremendously.

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