

## AP Biology

### Cell Signaling Taste Lab

#### Background

**Chemoreception** is the process by which organisms sense chemicals in their environment. This is often regarded as the oldest sense and is universal among animals; it is even found in **bacteria** and other microorganisms. Organisms use chemoreception to accomplish a number of different tasks, including identifying suitable habitats, determining the quality of a food source, finding a mate, finding places to lay eggs, and/or monitoring their internal environments.

Animals receive chemical information with special receptor neurons called **chemoreceptors**.

Chemoreception can take a number of different forms, often depending on the environment an organism lives in. In humans and most terrestrial animals, chemoreception has been refined into the senses of smell and taste. In contrast, for many organisms that have evolved living in water, there is not a big difference between the senses of smell and taste.

**Gustation**, the sense of taste, is closely related to the sense of smell. In fact, much of what we think of as taste is actually smell. In terrestrial vertebrates, taste is sensed through **taste buds** that are found in the mouth. Some fishes have taste buds in their skin, which enhances their ability to sense the environment they live in.

A taste bud is actually a cluster of several cells:

- (1) chemoreceptor cells that occupy the center of the bud, detect the tastant (i.e., a molecule that stimulates a taste), and synapse with an afferent sensory neuron
- (2) support cells that form the outer wall of the taste bud, as well as some portions of the center.

In humans, approximately 10,000 taste buds are found in the epithelium of the tongue, many on the raised **papillae**. In contrast to smell, which is sensed by many different types of chemoreceptors, taste is sensed by only a few (4-5) – it is the combination of these receptors that leads to the variety of tastes we can sense. These taste receptors detect tastants that signal sweet, salty, sour, or bitter. Recently, a fifth taste, called umami, has been identified- the savory, meaty taste that originates from amino acids and we commonly associate with MSG (monosodium glutamate). Similar to smell, taste is sensed by the diffusion of specific molecules into the taste buds. For example “saltiness” results from the diffusion of  $\text{Na}^+$ , “sourness” results from  $\text{H}^+$ , and the other tastes result from a variety of organic molecules.

Contrary to a common misconception, the taste buds are NOT localized on the tongue into regions for the primary tastes, but rather all areas of the tongue are responsible for the primary tastes. Taste papillae can be seen on the tongue as little red dots, or raised bumps, particularly at the front of the tongue. These visible papillae on the front are actually called “fungiform” papillae because they look like little button mushrooms. There are three other kinds of papillae: foliate, circumvallate and the non-gustatory filiform papillae. A papilla is not a taste bud in itself. Rather many taste buds (which are not visible to the naked eye) are found on each papilla. At the base of each taste bud, an afferent sensory nerve invades the taste bud and branches extensively. Each afferent sensory neuron typically synapses with multiple chemoreceptor cells within the taste bud.

Receptor activation → Intracellular Signaling → Neurotransmitter Release → Gustatory Neuron  
( $\text{Ca}^{2+}$  entry) Activation

## Signal Transduction of Taste

The transduction of taste signals typically involves ion channels bound in the membrane of taste chemosensory axons, including voltage-gated  $\text{Na}^+$ ,  $\text{K}^+$ , and  $\text{Ca}^{2+}$  channels that produce depolarizing potentials when taste chemoreceptor cells interact with tastants.

The resulting chemoreceptor potentials raise  $\text{Ca}^{2+}$  to levels sufficient between the chemosensory cell and afferent sensory neuron, thus eliciting action potentials in the afferent sensory neuron (Purves, et al, 2008). In general, the higher the concentration of the tastant, the greater the depolarization of the taste cell will be.

- **Transduction of Salt Taste**

Sodium ( $\text{Na}^+$ ) ions enter the receptor cells via Na-channels. The entry of  $\text{Na}^+$  causes a depolarization, then calcium ( $\text{Ca}^{2+}$ ) enters through voltage-sensitive  $\text{Ca}^{2+}$  channels, transmitter release occurs and results in increased firing in the primary afferent nerve results. Sodium chloride ( $\text{NaCl}$ ), common table salt, tastes saltiest.

- **Transduction of Sour Taste**

Sour taste is due to acids and acids essentially are protons ( $\text{H}^+$ ). Many sour, naturally acidic foods are rich in vitamin C (e.g. lemons, oranges, tomatoes). There are three possible transduction mechanisms:

- $\text{H}^+$  ions block potassium ( $\text{K}^+$ ) channels.  $\text{K}^+$  channels are responsible for maintaining the cell membrane potential at a hyperpolarized level (close to the  $\text{K}^+$  equilibrium potential of around  $-85\text{mV}$ ). Blockage of these channels causes a depolarization,  $\text{Ca}^{2+}$  entry, transmitter release, and increased firing in the primary afferent nerve.
- $\text{H}^+$  ions enter the cell through ENaC channels or
- $\text{H}^+$  ions enter the cell through a cation channel – the PCKD channel (this is the latest suggestion of Huang et al (2006) Nature 442, 934-8).

- **Transduction of Sweet Taste**

There are receptors in the membrane of the taste chemoreceptor cell that bind glucose, sucrose (a combination of glucose and fructose), and other carbohydrates. Binding to these receptors activates adenylyl cyclase, thereby elevating cAMP. This causes a PKA-mediated phosphorylation of  $\text{K}^+$  channels, inhibiting them. Depolarization occurs,  $\text{Ca}^{2+}$  enters the cell through depolarization-activated  $\text{Ca}^{2+}$  channels, and transmitter is released increasing firing in the primary afferent nerve.

There are a number of different sweet receptors that respond to many organic compounds; sugars, saccharin, alcohols, some amino acids, and some lead salts such as those found in lead paint. These different receptors are what make it possible to have so many different artificial sweeteners

- **Transduction of Bitter Taste**

Bitter substances cause the second messenger ( $\text{IP}_3$ ) mediated release of  $\text{Ca}^{2+}$  from internal stores, therefore external  $\text{Ca}^{2+}$  is not required. The elevated  $\text{Ca}^{2+}$  causes transmitter release and this increases the firing of the primary afferent nerve. Bitter taste is believed to have evolved as a protective mechanism to avoid poisonous plants and their toxic compounds such as the alkaloids quinine, caffeine, nicotine, and strychnine and non- alkaloids such as aspirin.

- **Transduction of Umami Taste**

Umami, Japanese for “delicious”, the taste of certain amino acids (e.g. glutamate, aspartate and related compounds), was first identified by Kikunae Ikeda at the Imperial University of Tokyo first identified it in 1909. It was originally shown that the metabotropic glutamate receptor (mGluR4) mediated umami taste (Chaudari, *et al*, 1996; Kurihara & Kashiwayanagi, 1998). Binding to the receptor activates a G-protein and this may elevate intracellular  $Ca^{2+}$ . More recently it has been found that the T1R1 + T1R3 receptors mediate umami taste (Nelson, *et al*, 2002). Monosodium glutamate (MSG) added to many foods to enhance their taste (and the main ingredient of Soy sauce), beef, and aging cheese may stimulate the umami receptors. In addition, there are ionotropic glutamate receptors (linked to ion channels) on the tongue. When activated by umami compounds or soy sauce, non-selective cation channels open, thereby depolarizing the cell. Calcium enters, causing transmitter release and increased firing in the primary afferent nerve.

## Procedure

To determine how taste receptors work you will either drink a tea made from the Indian herb *Gymnema sylvestre* or chew a tablet from the *Synsepalum dulcificum* plant, known for its “miracle berries”. Your task is to determine what effect *Gymnema* or miracle berries have on taste perceptions and try to come up with a mechanism that explains this effect.

## Determining How Taste Receptors Work

1. Obtain a small package/sample of each of the following: salt, Equal®, raw broccoli, sugar, chocolate, lemon wedge, apple cider vinegar, and SweeTarts®.
2. Taste these samples **in the following order**:
  - Salt
  - Equal® (Aspartame)
  - Raw broccoli
  - Sugar
  - M & Ms
  - Lemon wedge
  - Apple cider vinegar
  - SweeTarts®
3. Rate each substance for the perception of **sweet, sour, bitter**, and **salt** on a scale from 0 to 10 in the table below. A rating of “0” represents no perceived taste whereas a rating of “10” represents a very intense taste.
4. Rinse your mouth with water between each substance in order to avoid aftertaste mixtures.
5. After your initial taste of each substance, one member of the group should get a sample of the *Gymnema* tea, and one should get a sample of the miracle fruit.
  - Swish one ounce of tea in your mouth for 30 seconds. Try to coat all areas of the mouth with the tea. Spit the tea into the sink when finished, and then rinse your mouth briefly with water.
  - Allow the tablet to dissolve on your tongue—do not chew. It may take 5min or more.
6. Beginning with salt, (and following the list above) re-taste each of the substances. Rate and record your perceptions of salty, sweet, bitter and sour for each substance on the following page.



**Table 1**

Rate each substance for the perception of sweet, sour, bitter, and salt on a scale from 0 to 10. A rating of “0” represents no perceived taste and a rating of “10” represents a very intense taste.

Tastant	Taste Ratings				
		Sweet	Sour	Salty	Bitter
Salt	Before:				
	After Tea:				
	After miraculin:				
Aspartame	Before:				
	After Tea:				
	After miraculin:				
Raw Broccoli	Before:				
	After Tea:				
	After miraculin:				
Sugar	Before:				
	After Tea:				
	After miraculin:				
M & Ms	Before:				
	After Tea:				
	After miraculin:				
Lemon Wedge	Before:				
	After Tea:				
	After miraculin:				
Apple Cider Vinegar	Before:				
	After Tea:				
	After miraculin:				
SweeTarts	Before:				
	After Tea:				
	After miraculin:				

## Analysis Questions

1. Based on the data you collected on yourself/your lab partner, what observations can you make about the effect of *Gymnema sylvestre* on the sense of taste? Which type(s) of taste does the tea alter?
2. Based on the data you collected on yourself/your lab partner, what observations can you make about the effect of miraculin, the active ingredient in miracle fruit, on the sense of taste? Which type(s) of taste does the glycoprotein alter?
3. For each tastant, compare your before and after tea answers.
  - Which (if any) tastants' flavors were not affected by *Gymnema* tea?
  - Which (if any) tastants' flavors were eliminated by *Gymnema* tea?
  - Which (if any) tastants' flavors were changed by *Gymnema* tea? In what way?
4. For each tastant, compare your before and after miraculin answers.
  - Which (if any) tastants' flavors were not affected by miraculin?
  - Which (if any) tastants' flavors were eliminated by miraculin?
  - Which (if any) tastants' flavors were changed by miraculin? In what way?
5. What might be the possible mechanism for the effect of *Gymnema* on the perception of taste? Explain in detail.
6. What might be the possible mechanism for the effect of miraculin on the perception of taste? Explain in detail.