The Mechanistic Action of Taste in the Tongue and Gut

Basic Question

When approaching the subject of taste, most people understandably begin to think of the tongue, recognizing it as the central hub for one of our five principle senses. However, recent research has signified a whole new branch of our taste responses and mechanisms that is found within our digestive tract. This novel division is deeply linked with the enteric nervous system and may share close associations with the activities and mechanisms of taste sensory cells within the tongue. In order to investigate this further, I will compare and contrast the mechanistic actions of taste sensory cells in both the tongue and within the gut.

Introduction

The enteric nervous system (ENS) has been shown to be a complex superhighway for gut-brain signaling, being called the “second brain” by some researchers (Mayer 2011). This comparison is easily made when comparing the size, complexity, and resemblance to the central nervous system, becoming a rapidly growing focus for many areas of research. One area of growing interest in ENS science is the ability for the digestive tract to transduce the sense of taste to the brain or other effectors, altering traditional thoughts on how the tongue is the major organ for taste reception.

The taste buds are the transducing elements of gustatory sensation within the tongue (Kinnamon 2011). They serve in the detection of nutrients in foods and guard
against the ingestion of toxic substances or rotten foods. To accomplish this, the taste buds distinguish between five basic taste qualities that have been identified in humans and mammals: sweet, sour, bitter, salty, and umami (amino acids). The sweet, salty, and umami qualities of taste are required by the body for energy balance, ionic homeostasis, and the build-up of proteins. More specifically, sweet taste detects sugars, salty taste helps detect ions such as sodium, and umami taste detects amino acids. On the other hand, the sour and bitter qualities of taste help the body detect acids in unripe fruit or spoiled foods, as well as detecting many plant alkaloids that can be toxic to the body (Kinnamon 2011; Sclafani, et al 2007). Therefore, the sense of taste has a unique ability to both provide the proper nutrition to the digestive tract, while simultaneously protecting it against harmful compounds.

Additionally, different taste qualities are triggered by both ionic and complex compounds and have developed different mechanisms for their detection. For example, salts and acids are primarily detected by ion channels, while chemicals eliciting bitter, sweet, and umami tastes are detected by G-protein-coupled receptors (GPCRs) and second messenger signaling pathways (Kinnamon 2011). There has also been significant progress in identifying the taste-signaling elements that mediate the response to sugars and amino acids. These include the T1R2/T1R3 sweet receptor, the T1R1/T1R3 umami receptor, the G-protein gustducin, and the calcium-activated cation channel TrpM5. Gustducin and TrpM5 also mediate the transduction of compounds that elicit bitter taste but not of compounds that elicit salty or sour taste qualities (Sclafani, et al 2007).

Moreover, many of the receptors and downstream second messengers involved
in taste detection and transduction have been shown to be involved in chemosensory functions of the gut (Kokrashvili et al 2009). More specifically, the enteroendocrine cells of the gut express these signaling elements, allowing the gut to function in concert with the ENS, the brain, and other effectors for taste reception and transduction.

**Current State of Knowledge**

The sense of taste depends on specialized receptor cells contained in taste buds located in the surface of the tongue. A specific taste is initiated by the interaction of a stimulus with the receptors and ion channels of taste receptor cells. Some taste transduction pathways convert chemical information into a cellular second messenger code (Kokrashvili et al 2009). This chemical mechanism of second messenger release occurs through the action of G-protein-coupled receptors (GPCRs). For example, two classes of taste GPCRs have been identified molecularly, T1Rs and T2Rs (Kinnamon 2011). T1Rs mediate sweet and umami taste, while T2Rs mediate bitter taste (Kinnamon 2011; Kokrashvili et al 2009).

T1R GPCRs are ‘C’ type receptors, with large N-terminal ligand binding domains that exhibit a venus fly trap ligand binding domain. These receptors are similar to the metabotropic glutamate receptors, such as the GABA_B receptor (Kinnamon 2011). Additionally, three different T1Rs have been identified: T1R1, T1R2 and T1R3. These receptors are functional only as heterodimers, where T1R3 must be one of the dimers for both the umami receptor (T1R1 + T1R3) and the sweet receptor (T1R2 + T1R3) (Kinnamon 2011; Sclafani, et al 2007). The sweet receptor heterodimer (T1R2 + T1R3) binds to molecules such as sugars, d-amino acids, synthetic sweeteners, and some sweet proteins. **END OF SAMPLE...**
References


