

Chemesthesis affects taste

The sensations of taste, which are divisible into several distinct qualities: sweet, sour, bitter, salty, umami, and possibly fatty, play an important role for accepting or rejecting food and serve to protect us from ingesting harmful substances, but also cause the behavioral preferences to some types of foods. Taste sensations begin with the stimulation of taste receptor cells located in taste buds, small clusters of specialized epithelial cells on the tongue and in the oral cavity. Taste cells release transmitters during gustatory stimulation to stimulate primary afferent sensory fibers that carry the gustatory information to the brain.

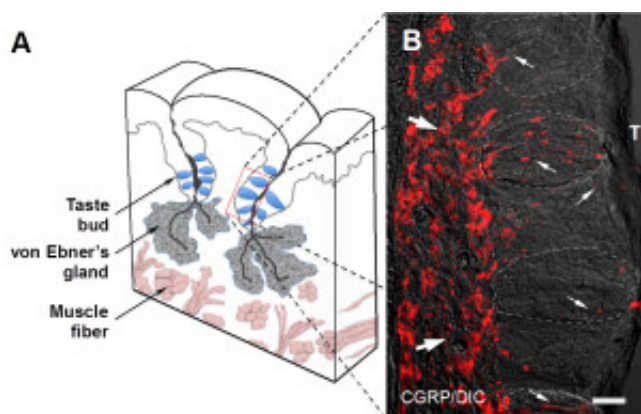


Fig. 1. CGRP immunohistochemistry in mouse vallate papillae. A, Schematic drawing of a lingual slice containing the vallate papilla. Red box represents approximate position of the taste buds in B.

In addition to transmitting gustatory responses to prototypic taste stimuli as part of a common chemical senses, taste transmitters are expected may contribute to regulating the function of adjacent taste cells. It is now being recognized there is a significant degree of information processing in the taste buds during gustatory stimulation. This information processing involves cell-to-cell communication within the taste bud using transmitters such as serotonin (5-HT), ATP, and γ -aminobutyric acid (GABA).

Sensory signals from the oral cavity are carried to the brain in chemosensory fibers that contribute to chemesthesis, the general chemical sensitivity of the skin and mucus membranes in the oronasal cavities and being perceived as pungency, irritation or heat. Taste perception alterations follow condition changes in the mouth, such as temperature. For instance, the pungency of foods and beverages is likely highly influenced by the temperature at which they are consumed, their acidity, and, for beverages, their carbonation. Indeed, electrophysiology revealed that taste perception is enhanced as the temperature of food and beverage products increases. The mechanism why/how this occurs is not fully known. Intriguingly, despite the profusion of work identifying mechanisms of chemosensation, little information has been generated regarding

irritant–taste interactions.

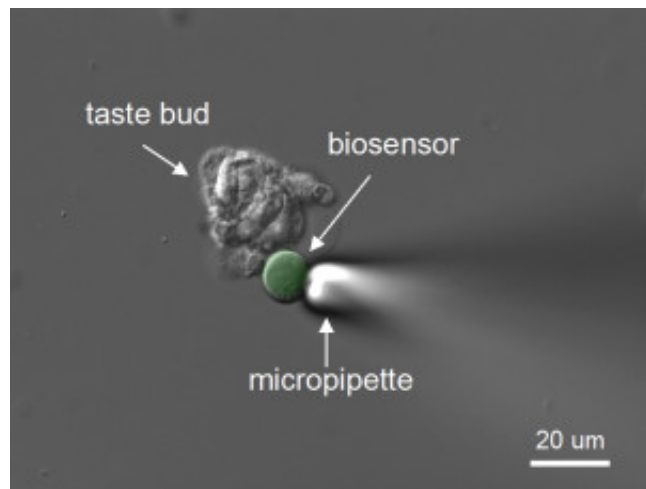


Fig. 2. Micrograph of a biosensor cell abutted against an isolated taste bud in a living preparation

My laboratory at the Southern Illinois University School of Medicine in Carbondale has innovated a powerful technique using genetically-engineered biosensors, to study whether and how taste bud cells release transmitters by real-time Ca^{2+} imaging. In the present article, by using 5-HT and ATP biosensors, we reported that calcitonin gene-related peptide (CGRP), which may be released from peripheral axon terminals, modulated taste-evoked ATP secretion from taste buds, which is a key transmitter for communication from taste buds to gustatory nerves. Unambiguously, our findings suggest that the complex interplay between taste cells and chemosensory neurons may play an important role during the processing of the complex stimuli involving both spicy and tasty components.

The characterization of local transmitters and their effects in the taste bud is essential not only for basic understanding of sensory afferent excitation but also for potential clinical applications. These include development of taste modifiers for potential use in management of obesity, compliance in taking medications (particularly in pediatrics), and pharmaceutical development in the context of diminished taste sensation or ageusia (lack of taste) related to age or disease chemotherapy. Indeed, taste perceptions are altered by medications that inadvertently also act on taste bud transmitters. For example, selective serotonin reuptake inhibitors such as Prozac have known taste side effects.

Publication

[Calcitonin Gene-Related Peptide Reduces Taste-Evoked ATP Secretion from Mouse Taste Buds.](#)

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