Tonic Activity of Parasympathetic Efferent Nerve Fibers Hyperpolarizes the Resting Membrane Potential of Frog Taste Cells

Toshihide Sato¹, Kazuhisa Nishishita², Yuzo Kato², Yukio Okada¹ and Kazuo Toda¹

¹Divisions of Integrative Sensory Physiology and ²Oral Molecular Pharmacology, Nagasaki University Graduate School of Biomedical Sciences, 1-7-1 Sakamoto, Nagasaki 852-8588, Japan

Correspondence to be sent to: Toshihide Sato, Division of Integrative Sensory Physiology, Nagasaki University Graduate School of Biomedical Sciences, 1-7-1 Sakamoto, Nagasaki 852-8588, Japan. e-mail: toshi@net.nagasaki-u.ac.jp

Abstract

We investigated the relationship between the membrane potential of frog taste cells in the fungiform papillae and the tonic discharge of parasympathetic efferent fibers in the glossopharyngeal (GP) nerve. When the parasympathetic preganglionic fibers in the GP nerve were kept intact, the mean membrane potential of Ringer-adapted taste cells was $-40 \text{ mV}$ but decreased to $-31 \text{ mV}$ after transecting the preganglionic fibers in the GP nerve and crushing the postganglionic fibers in the papillary nerve. The same result occurred after blocking the nicotinic acetylcholine receptors on parasympathetic ganglion cells in the tongue and blocking the substance P neurokinin-1 (NK-1) receptors in the gustatory efferent synapses. This indicates that the parasympathetic nerve (PSN) hyperpolarizes the membrane potential of frog taste cells by $-9 \text{ mV}$. Repetitive stimulation of a transected GP nerve revealed that a $-9 \text{ mV}$ hyperpolarization of taste cells maintained under the intact GP nerve derives from an $-10 \text{ Hz}$ discharge of the PSN efferent fibers. The mean frequency of tonic discharges extracellularly recorded from PSN efferent fibers of the taste disks was $9.1 \text{ impulses/s}$. We conclude that the resting membrane potential of frog taste cells is continuously hyperpolarized by an average $-9 \text{ mV}$ by an $-10 \text{ Hz}$ tonic discharge from the parasympathetic preganglionic neurons in the medulla oblongata.

Key words: gustatory efferent synapse, membrane potential, parasympathetic nerve, slow hyperpolarizing potential, taste receptor cell

Introduction

The sensitivity of some sensory cells such as a stretch receptor and a hair cell is controlled by efferent fibers supplying the cells (Eyzaguirre and Kuffler, 1955; Furukawa, 1981). Efferent control of gustatory cell activities has been suggested by electron microscopical and electrophysiological studies (Nomura et al., 1975; Yoshie et al., 1996; Reutter et al., 1997; Sato et al., 2002, 2004). Our previous studies have revealed that electrical stimulation of efferent C-fibers in the lingual branch of the frog glossoopharyngeal (GP) nerve induces the postsynaptic-like slow hyperpolarizing potentials (HPs) in taste cells of the fungiform papillae (Sato et al., 2002). These slow HPs are likely to be generated in taste cells by releasing substance P as a neurotransmitter from the presynaptic terminals of the GP efferent fibers (Sato et al., 2004).

Recently, efferent nerve fibers supplying the taste cells in the frog fungiform papillae have been found to be the parasympathetic fibers alone in the lingual branch of the GP nerve (Sato et al., 2005). Sympathetic efferent fibers running along the lingual branch of the GP nerve in the frog do not innervate the taste cells in the fungiform papillae (Sato et al., 2005) but their arterioles (Inoue and Kitada, 1988).

Since the frog taste cells are hyperpolarized by stimulating parasympathetic nerve (PSN) efferent fibers (Sato et al., 2005), there is the possibility that the membrane potential of the taste cells is continuously controlled by tonic discharges from the parasympathetic preganglionic neurons originating in the medulla oblongata. We studied this possibility in the present experiment.

Materials and methods

Preparation

Bullfrogs (Rana catesbeiana) weighing 350–720 g were used in the experiments. All the experiments were conducted in accordance with Nagasaki University Guidelines for Animal Experimentation. The animals were anesthetized by intraperitoneal injection of a 50% urethane-Ringer solution at a dose of 1.5–4.0 g/kg body weight. The anesthetic solution
was initially injected at 1 g/kg and then gradually added. The intensity of anesthesia was weak or moderate, so that the heart rate and lingual blood circulation were almost normal during each experiment for 3 h. The mean heart rate at room temperature of 23–25°C was 58 ± 2 beats/min (n = 20) in control and 55 ± 2 beats/min (n = 20) 3 h after anesthesia. To remove spontaneous twitches of the tongue muscles, the hypoglossal nerves were cut bilaterally. Both GP nerves were separated free from the surrounding connective tissues and kept intact in an unsectioned state. In some experiments, the GP nerve was transected and the papillary nerve in the fungiform papillae was crushed with a pair of fine forceps. The GP nerves were immersed into mineral oil, and the tongue was pulled from the mouth and pinned on a silicone rubber plate. The room temperature in our laboratory was maintained at 23–25°C during all experiments.

Electrical recording and stimulation

Intracellular electrical recordings from taste cells were made with the same methods and criteria as mentioned previously (Sato et al., 2002, 2004). Briefly, a 3-M KCl-filled microelectrode of 20–70 MΩ was inserted deeply into the central part of the taste disk of the fungiform papillae to impale a taste cell of type II or type III (Osculati and Sbarbati, 1995). An indifferent electrode of chlorided silver wire was inserted into the forelimb muscles. The tongue surface was always adapted to the frog Ringer solution. The magnitude of the membrane potentials in taste cells was measured at the stationary level 20–30 s after cell penetration. We could extracellularly record electrical activities from the PSN fiber terminals supplying the taste disk (Inoue et al., 1992) when a microelectrode of 3–5 MΩ (tip diameter, 1–3 μm) was inserted into the plexus at the basal layer of the taste disk in the fungiform papillae. An indifferent electrode was put on the submaxillary muscle to remove the electrocardiogram. Probably, the extracellular microelectrode tip touched the enlarged terminals (Inoue et al., 1992) of the PSN fibers in the taste disk in case of successful recordings. All electrical signals were amplified with a microelectrode amplifier (MEZ-8101, Nihon Kohden, Tokyo) and recorded on a pen recorder.

The GP nerve and papillary nerve were electrically stimulated at 1–50 Hz with a strong pulse of 0.1 ms in duration and 15 V in strength to obtain the slow HP from taste cells innervated by the PSN efferent C-fibers. Electrical stimulation of low threshold-myelinated afferent fibers in the GP nerve does not induce any slow HPs in taste cells (Sato et al., 2002). When the papillary nerve was electrically stimulated, the stalk of the fungiform papillae was sucked with a suction electrode and cathodal pulses were given to the nerve (Sato et al., 2005).

To block spontaneous tonic impulses from PSN fibers in the GP nerve, a pair of anodal and cathodal stimulating electrodes (silver wires) was located on the GP nerve distally and proximally, respectively, and the GP nerve was stimulated at 200 Hz with a strong pulse of 0.1 ms in duration and 15 V in strength. Cathodally elicited impulses in all types of fibers conducted antidromically and orthodromically. The antidromically conducting impulses along the parasympathetic preganglionic efferent C-fibers collided with the spontaneous tonic impulses orthodromically conducting along the same efferent C-fibers. All the orthodromically conducting efferent impulses, which were elicited at a cathode by electrical stimulation, were blocked at an anode by anodal polarizations.

Taste receptors in the tongue surface were stimulated by deionized water, 0.5 M NaCl, 1 mM acetic acid, or 10 mM quinine–HCl (Q–HCl). These solutions were all made up in deionized water. The taste solution was flowed on the tongue surface at a rate of 0.05 ml/s, and it was rinsed with a frog Ringer solution at the same rate after the end of taste stimulation. The Ringer solution was composed of 115 mM NaCl, 2.5 mM KCl, 1.8 mM CaCl₂, and 5 mM 4-(2-hydroxyethyl)-piperazine-1-ethanesulfonic acid, and the pH was adjusted to 7.2 by a Tris [tris(hydroxymethyl) aminomethane] buffer.

Drugs

To block nicotinic acetylcholine receptors (nAChRs) on the parasympathetic ganglion cell bodies in the tongue (Sato et al., 2005), d-tubocurarine chloride was injected intravenously (i.v.). L-703,606 oxalate salt (an antagonist of substance P NK-1 receptor) was injected i.v. to block the efferent synapses of taste cells (Sato et al., 2004, 2005). To block the lingual glands richly present on the tongue surface, atropine sulfate was injected i.v. All drugs were purchased from Sigma–Aldrich Co. (St Louis, MO). d-tubocurarine chloride and atropine sulfate were dissolved into the frog Ringer solution. A stock solution from L-703,606 oxalate salt was prepared with methanol and added into the Ringer solution when used. The amount of i.v. injected Ringer solution containing a drug was 2 ml/kg body weight.

Experimental procedure

Electrical repetitive stimulation of the GP nerve with strong voltage intensities produces a large slow potential on the tongue surface and in taste disk cells (Sato et al., 2000). This derives from the physicochemical junction potential generated between the saliva released from abundant lingual glands and the lingual surface solution. This potential disturbs an analysis of physiological slow HPs in taste cells elicited by GP nerve stimulation. Therefore, before the start of intracellular recordings, atropine was injected i.v. at a dose of 1 mg/kg to completely block the slow physicochemical junction potential. The injection effect lasted >7 h.

Statistics

All data were expressed as means ± SEMs. The level of significance was set at P < 0.05 with a Student’s t-test.
Results

Hyperpolarization of resting membrane potential in taste cell by tonic PSN activity

Of the sympathetic and parasympathetic efferent fibers running along the lingual branch of frog GP nerve, the latter alone innervate the taste cells of the taste disk (Sato et al., 2004, 2005). When the parasympathetic preganglionic fibers in the GP nerve were kept intact, the mean membrane potential of taste cells recorded from the four tongues was significantly reduced to $-39.8 \pm 1.8 \text{ mV (n = 90)}$ (Figure 1). On the other hand, when the preganglionic fibers of the GP nerve was sectioned, the mean membrane potential of the taste cells recorded from the four tongues was significantly reduced to $-31.0 \pm 1.9 \text{ mV (n = 85, P < 0.05)}$. Mechanically crushing the parasympathetic postganglionic fibers in the papillary nerve supplying the taste disk also significantly reduced the membrane potential of taste cells to $-30.7 \pm 1.3 \text{ mV (n = 24, P < 0.05)}$.

These large reduction in the membrane potentials occurred 3 min after mechanical blocking of the PSN fibers. After $\mathrm{d}$-tubocurarine had been injected i.v. at 1 mg/kg body weight to block the nAChRs on the parasympathetic ganglion cells in the tongue, the mean membrane potential of taste cells under the intact GP nerve was reduced to $-30.8 \pm 1.7 \text{ mV (n = 21)}$ within 15 min. After L-703,606 (an antagonist of substance P NK-1) had been injected i.v. at 4 mg/kg to block the synapses between taste cells and PSN efferent fibers (Sato et al., 2004, 2005), the mean membrane potential of taste cells under the intact GP nerve was reduced to $30.8 \pm 1.6 \text{ mV (n = 37)}$. To confirm the control blocking effects of the two drugs, the membrane potential change in taste cells induced by the drug injection was measured under transected GP nerve. Since the membrane potential after the treatment of $\mathrm{d}$-tubocurarine (1 mg/kg) and L-703,606 (4 mg/kg) was $-31.2 \pm 2.0 \text{ mV (n = 14)}$ and $-30.8 \pm 2.1 \text{ mV (n = 18)}$, respectively, each drug was suggested to act on the target receptor alone. No statistical differences existed among the membrane potentials of taste cells recorded from the papillary nerve-crushed, GP nerve-sectioned, $\mathrm{d}$-tubocurarine-treated, and L-703,606-treated tongues ($P > 0.05, n = 21–85$). Since either mechanical or pharmacological blocking of the parasympathetic pre- and postganglionic fiber activities equally reduced the taste cell membrane potential by 9 mV, no tonic discharges will arise from the postganglionic neurons themselves. These results suggest that the membrane potentials of frog taste cells are continuously hyperpolarized by an average $-9 \text{ mV}$ by a tonic discharge from the parasympathetic preganglionic neurons via the GP nerve.

At the horizontal bar of Figure 2, spontaneous tonic C-fiber discharges of PSN influencing the membrane potential in a taste cell were blocked by a collision with antidromically conducting C-fiber impulses evoked by strong 200-Hz stimulation of the GP nerve. After the PSN block, a slow depolarizing potential (DP) appeared in the taste cell because the tonic PSN discharge was not transmitted to the taste disk and returned slowly to the baseline following the removal of the blocking. The slow DPs had the amplitude of $6.8 \pm 0.4 \text{ mV (n = 18)}$, the latency of $1.9 \pm 0.3 \text{ s (n = 18)}$, and the rise time of $5.0 \pm 0.6 \text{ s (n = 18)}$.

Steady maintenance of membrane potential

We examined whether the membrane potential of a taste cell hyperpolarized by PSN fluctuates or not. Figure 3A illustrates a 20-min intracellular recording of the membrane potential from a taste cell under the intact GP nerve where no

![Figure 1](image1.png) 

**Figure 1.** Amplitudes of membrane potentials of frog taste cells before and after mechanical and pharmacological blocks of PSN activities. The membrane potentials of taste cells were recorded from tongues under intact GP and papillary nerve (control), sectioned GP nerve, crushed papillary nerve, and intravenous injections of $\mathrm{d}$-tubocurarine (1 mg/kg) and L-703,606 (4 mg/kg). Vertical bars in the columns are SEMs, and numerals below the bars are the number of taste cells tested. Tongue surface was adapted to Ringer solution in all the experiments.

![Figure 2](image2.png) 

**Figure 2.** Effect of blocking tonic discharge from a PSN fiber on membrane potential of a taste cell. A pair of cathodal and anodal stimulating electrodes was put on intact GP nerve proximally and distally, respectively, and 200-Hz stimulation (stim) with a pulse of 0.1 ms in duration and 15 V in strength was given for a period of the horizontal bar. A microelectrode was inserted into a taste cell via three steps of membrane potential deflection. The membrane potential was $-41.8 \text{ mV}$. 

Membrane Potential of Frog Taste Cell 309
fluctuation of the membrane potential was seen. Since an intracellular recording from a taste cell was incapable of a long holding for more than 30 min, the stability of the membrane potentials was statistically analyzed by continually measuring the membrane potentials from many taste cells in an intact tongue. Figure 3B shows that the amplitudes of the membrane potentials in taste cells were stable at \(-25.5\) mV during a 3-h observation and depolarized to \(-30.5\) mV after a transection of PSN fibers in the GP nerve. Consequently, the hyperpolarization of the taste cell membrane potential by a tonic discharge of PSN fibers is continual and stationary.

**Relationship between frequency of GP nerve stimulation and magnitude of slow HPs**

To disclose what frequency in the tonic discharge of the PSN induces the \(-9\)-mV hyperpolarization in taste cells, we investigated the relationship between the stimulus frequency applied to a GP nerve and the magnitude of slow HPs induced in taste cells. The amplitude of slow HPs is dependent on the membrane potential level (Sato et al., 2002), so the data were obtained from seven taste cells having the mean membrane potential of \(-31.4\) mV (a range of \(-28.3\) to \(-34.7\) mV) under the transected nerve and from eight taste cells having that of \(-40.2\) mV (a range of \(-37.1\) to \(-43.2\) mV) under the intact nerve. As shown in the solid curve of Figure 4, the taste cell membrane potentials under the GP nerve transection were gradually hyperpolarized as the stimulus frequency of para-sympathetic preganglionic fibers in the GP nerve was increased up to 30 Hz.

It is seen that \(-10\)-Hz stimulation hyperpolarized the membrane potential by \(-9\) mV. This suggests that the mean \(-9\)-mV elevation of the membrane potential in taste cells under intact GP nerves derives from an \(-10\)-Hz tonic discharge from the parasympathetic preganglionic fibers. Only small hyperpolarizations in the membrane potential of taste cells were evoked by pulse-train stimulation of the unsectioned GP nerve, as seen in the dashed curve in Figure 4. Thirty-hertz stimulation elicited only a \(-2\)-mV hyperpolarization in taste cells. This is because the membrane potential of taste cells had already been hyperpolarized by \(-9\) mV by the tonic discharge from PSN fibers. Repetitive stimulation of the parasympathetic postganglionic fibers in the distal portion of crushed papillary nerve induced a gradual increase of slow HPs in taste cells with increasing frequencies. The stimulus frequency–slow HP response curve was similar to the solid curve of Figure 4 (\(n = 4\), data not shown).

**Tonic discharge frequency in PSN fibers**

We tried to detect tonic impulses from the PSN fibers with extracellular microelectrodes. When the microelectrode was advanced into the basal plexiform layer of a taste disk, we could pick up two types of spike potential deflection, negative going and positive going. Figure 5A shows an example of discharges simultaneously recorded from two different axon
terminals within the taste disk. The small positively deflected spike potentials in one axon terminal did not respond to gustatory stimuli of water, 0.5 M NaCl, 1 mM acetic acid, and 10 mM Q-HCl applied to the tongue, but the large negatively deflected spike potentials in the other axon terminal responded to 0.5 M NaCl and 1 mM acetic acid. Therefore, the large negative spike potentials are an afferent discharge from the gustatory fiber. The large positive spike potentials (asterisks) induced by the NaCl and acetic acid stimuli are a well-known antidromic gustatory discharge traveled through branched fibers from the neighboring fungiform papillae (Rapuzzi and Casella, 1965; Sato, 1976; Kitada, 1978).

Since unmyelinated efferent fibers innervating the taste disks of the frog fungiform papillae are PSN fibers alone (Inoue and Kitada, 1991; Inoue et al., 1992; Sato et al., 2005), it is suggested that the small positive spike potentials are an efferent tonic discharge from PSN fibers supplying the taste disk. In the preparations where either the parasympathetic preganglionic fibers in the GP nerve was transected or the parasympathetic ganglia in the tongue were blocked by intravenous injection of D-tubocurarine at 1 mg/kg, the small positive spike potentials were no longer detected from the taste disks (n = 36).

Of neural activities of 28 fibers recorded from the basal plexus of the taste disks under the intact GP nerve, 19 fibers (68%) showed tonic discharges of PNS fibers alone with small positive spike potentials, and nine fibers (32%) showed gustatory discharges alone with both large negative (orthodromic) and large positive (antidromic) spike potentials. In eight recordings, the small positive spike potentials in one PSN fiber appeared simultaneously with the large negative and large positive spike potentials in the other gustatory fiber. A histogram of tonic discharges in 19 PSN fibers of the taste disks is shown in Figure 5B. The mean frequency of tonic discharges was 9.1 ± 0.6 impulses/s (n = 19). Discharge frequencies of negative spike potentials in the gustatory fibers were 1.5 ± 0.2 impulses/10 s (n = 9) in a resting state, 9.5 ± 0.8 impulses/10 s (n = 9) by 0.5 M NaCl, and 14.0 ± 0.7 impulses/10 s (n = 9) by 1 mM acetic acid.

**Discussion**

Sympathetic nerves and PSNs periodically discharge tonic impulses in resting states to maintain the basal activities of visceral organs. The tonic discharge frequency is usually as low as 1–4 Hz (Mirgorodsky and Skok, 1969; Su, 1999; Su et al., 2003). On the other hand, the tonic discharge at a higher frequency of 10–20 Hz has been found in the parasympathetic ciliary ganglion cells (Melnitchenko and Skok, 1970; Johnson and Purves, 1983). The present studies indicated that the membrane potential of the frog taste cells at rest is hyperpolarized by −9 mV by ~10-Hz tonic discharges from PSN efferent fibers in the GP nerve. Consequently, it is clear that the frog taste cells periodically receive a high frequency of tonic discharges from PSN.

In gustatory afferent synapses in frogs, a taste cell is a presynaptic cell, and an afferent fiber is a postsynaptic fiber. Synaptic transmissions between pre- and postsynaptic cells are modulated presynaptically and postsynaptically (Eccles, 1973; Kuffler and Nicholls, 1977; Ganong, 2003). The presynaptic modulation is composed of two types: presynaptic inhibition (Eccles, 1973; Kuffler and Nicholls, 1977) and presynaptic facilitation (Ganong, 2003). An efferent fiber terminal in the frog taste disk is suggested to make synaptic

---

**Figure 5** Tonic discharge of PSN fibers innervating taste disk. (A) Simultaneous recording of small and large positive (upward deflected) and large negative (downward deflected) spike potentials from two different types of nerve fiber within a taste disk under intact GP nerve. Trace (a) shows spontaneous discharges before gustatory stimulation (control). Traces (b) and (c) show spontaneous discharges and gustatory responses for 0.5 M NaCl and 1 mM acetic acid, respectively. Large negative and large positive (asterisk) spike potentials responded to gustatory stimuli but small positive spike potentials did not. The former are from a gustatory fiber, and the latter are from a PSN fiber. The three traces were obtained from the same two fibers in a taste disk. (B) Histogram of tonic discharges of 19 PSN efferent fibers recorded from 19 taste disks under intact GP nerve.
contacts with a taste cell as a presynaptic cell because the membrane conductance of the taste cell is changed by electrical stimulation of PSN fibers in the GP nerve (Sato et al., 2002). In the case of the presynaptic inhibition, a depolarization occurs at the presynaptic cell terminals and thereby a transmitter release from the terminals is reduced (Eccles, 1973), but in the case of the presynaptic facilitation, a hyperpolarization occurs at the presynaptic cell terminals and a transmitter release from the terminals is facilitated (Mendell and Wall, 1964; Hodge, 1972; Ganong, 2003). Consequently, the presynaptic modulation in a frog taste cell by gustatory efferent fibers is a type of the presynaptic facilitation. Since PSN efferent fibers in the GP nerve induce a slow HP in the presynaptic taste cell terminals, the amplitude of taste-induced receptor potentials in frog taste cells is enhanced (Sato et al., 2005). Hence, it is supposed that a transmitter release from taste cell terminals is facilitated and that an impulse frequency is enhanced in a gustatory fiber in the frog.

Frog taste cells generate spike potentials by opening of Na\(^{+}\) channels. Usually the Na\(^{+}\) channels in the frog taste cell are mostly inactivated at the membrane potential of \(-30\) mV, but their action is partially restored at \(-40\) mV (Suwabe and Kitada, 2004). Therefore, the excitability in the taste cells which is restored under a gustatory efferent fiber-induced hyperpolarization of \(-9\) mV also will facilitate a synaptic transmission between a taste cell and a gustatory afferent fiber. The taste receptor cells in frog taste disks are morphologically estimated to be type II and type III cells (Osculati and Sbarbati, 1995). Recently, the taste receptor cells are physiologically suggested to be type III cells alone having voltage-gated Ca\(^{2+}\) channels (Suwabe and Kitada, 2004). The presynaptic cells in the taste disk which are concerned with presynaptic facilitation are likely to be type III cells.

In the auditory and vestibular organs, efferent fibers innervate sensory hair cells or afferent fibers (Flock, 1971; Bloom and Fawcett, 1975), and they usually inhibit auditory and vestibular impulses (Flock, 1971; Furukawa, 1981). Therefore, it is suggested that an inhibition of auditory and vestibular impulses by the efferent synapse at a hair cell is due to a presynaptic inhibition and that an inhibition of those by the efferent synapse at an auditory or vestibular afferent fiber terminal is due to a postsynaptic inhibition (Flock, 1971).

Extracellular recordings of discharge from the axon terminals of the taste disks in the intact tongue showed two types of spike potential. One is the positive-going spike potentials and the other is the negative-going spike potentials. When a recording microelectrode reaches the plexiform layer of a taste disk, negative spike potentials are likely to derive from between negativity of an unmyelinated gustatory axon terminal due to spontaneous or tastant-induced excitation and positivity of an unexcited axon near the terminal. On the other hand, small positive spike potentials are likely to derive from between positivity of an unexcited parasym pathetic postganglionic axon terminal and negativity of an excited axon near the terminal due to arrival of a tonic discharge from the PSN. Also, the large positive spike potentials will come from between positivity of an unexcited gustatory axon terminal and negativity of an excited axon near the terminal due to an antidromic gustatory discharge from the neighboring fungiform papillae.

All the gustatory nerve fibers in the frog fungiform papillae are myelinated (Hanamori and Ishiko, 1981; Sato et al., 1989) but lose the myelin sheaths below the taste disk (Rapuzzi and Casella, 1965; Düring and Andres, 1976). Single unmyelinated gustatory axons frequently ramify underneath and within the taste disk and innervate many taste receptor cells (Gaupp, 1904; Düring and Andres, 1976). Miyamoto et al. (1985) suggested that gustatory impulses in the frog fungiform papillae are firstly initiated at the first node of Ranvier of the myelinated nerve fibers and that tastant-induced postsynaptic potentials at gustatory axon terminals spread along the unmyelinated axonal part but do not initiate the impulses there. However, the present experiment clearly demonstrated that unmyelinated gustatory axon terminals in the taste disk of the fungiform papillae are capable of initiating the gustatory impulses.

References


Accepted January 17, 2006