Taste Cell Responses in the Frog Are Modulated by Parasympathetic Efferent Nerve Fibers

Toshihide Sato¹, Yukio Okada¹, Toshihiro Miyazaki², Yuzo Kato³ and Kazuo Toda¹

¹Division of Integrative Sensory Physiology, ²Division of Oral Cytology and Cell Biology and ³Division of Oral 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 studied the anatomical properties of parasympathetic postganglionic neurons in the frog tongue and their modulatory effects on taste cell responses. Most of the parasympathetic ganglion cell bodies in the tongue were found in extremely small nerve bundles running near the fungiform papillae, which originate from the lingual branches of the glossopharyngeal (GP) nerve. The density of parasympathetic postganglionic neurons in the tongue was 8000–11,000/mm³ of the extremely small nerve bundle. The mean major axis of parasympathetic ganglion cell bodies was 21 µm, and the mean length of parasympathetic postganglionic neurons was 1.45 mm. Electrical stimulation at 30 Hz of either the GP nerve or the papillary nerve produced slow hyperpolarizing potentials (HPs) in taste cells. After nicotinic acetyl choline receptors on the parasympathetic ganglion cells in the tongue had been blocked by intravenous (i.v.) injection of D-tubocurarine (1 mg/kg), stimulation of the GP nerve did not induce any slow HPs in taste cells but that of the papillary nerve did. A further i.v. injection of a substance P NK-1 antagonist, L-703,606, blocked the slow HPs induced by the papillary nerve stimulation. This suggests that the parasympathetic postganglionic efferent fibers innervate taste cells and are related to a generation of the slow HPs and that substance P is released from the parasympathetic postganglionic axon terminals. When the resting membrane potential of a taste cell was hyperpolarized by a prolonged slow HP, the gustatory receptor potentials for NaCl and sugar stimuli were enhanced in amplitude, but those for quinine-HCl and acetic acid stimuli remained unchanged. It is concluded that frog taste cell responses are modulated by activities of parasympathetic postganglionic efferent fibers innervating these cells.

Key words: efferent synapse, frog taste cell, parasympathetic postganglionic neuron, slow hyperpolarizing potential, tastant-induced receptor potential

Introduction

The efferent innervation of taste cells 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). Efferent fibers innervating taste cells are thought to modulate the gustatory sensitivity of taste cells situated in various living environments (Sato et al., 2002).

Parasympathetic ganglia, which have been observed in the tongues of various animals, are suggested to contribute to the physiological functions of the gustatory organs, lingual glands, and lingual blood vessels (Gairns and Garven, 1952; K.N. Bhargava and A.K. Bhargava, 1974; Jaeger and Hillman, 1976). Electron-microscopical studies on the frog tongue (Inoue and Kitada, 1991; Inoue et al., 1992) clarified that unmyelinated efferent fibers from the parasympathetic ganglia in the tongue are in close contact with the supporting cell and basal cell in the taste disk of the fungiform papillae. Our previous works (Sato et al., 2002, 2004) suggested that the frog taste cell elicits a slow hyperpolarizing potential (HP), which is similar to a slow inhibitory postsynaptic potential, in response to stimulation of unmyelinated efferent fibers of the glossopharyngeal (GP) nerve and that substance P is released from these unmyelinated efferent fiber terminals.

The present experiments were undertaken to investigate the anatomical properties of parasympathetic postganglionic neurons in the frog tongue and their modulatory effects on tastant-induced receptor potentials in the taste cells.
Materials and methods

Preparation

Twenty-nine bullfrogs (Rana catesbeiana) were used in the present experiments. All experiments were performed under the Guidelines for Animal Experimentation of Nagasaki University. The experiments using physiological approaches were carried out with the whole animal. The animals were anesthetized with intraperitoneal injection of a 50% urethane–Ringer solution (1–3 g/kg body weight). The tongue was pulled from the mouth and pinned on a silicone rubber plate. The blood supplies of lingual arteries and veins were kept normal as long as possible. The hypoglossal nerves on both sides were severed to remove the spontaneous contraction of the tongue. The GP nerves on both sides were separated free from the surrounding connective tissues, cut centrally, and immersed into mineral oil. The experiments were carried out at room temperature of 22–26°C.

Recordings and stimulations

The methods and criteria for intracellular recordings from taste cells in the fungiform papillae were the same as described previously (Sato et al., 2002, 2004). Shortly, a microelectrode was deeply inserted into the central part of the taste disk of the fungiform papillae in order to penetrate a taste cell of type II or type III (Osculati and Sbarbati, 1995). The fungiform papillae scattered in the apical and middle regions of the tongue were used. Mostly, a microelectrode would penetrate a type III cell because 80–90% of the total cells at the receptor and glia-like cell body layer (lower intermediate layer) of the taste disks is composed of type III cells in the apical and middle loci of the tongue (Osculati and Sbarbati, 1995; Li and Lindemann, 2003). Also, electrical stimulation of the GP nerve was performed with the previously mentioned method (Sato et al., 2002, 2004). In brief, the GP nerve and papillary nerve were electrically stimulated at 30 Hz with pulses of 0.1-ms duration and 15-V strength to obtain the maximal slow HPs from taste cells. When the parasympathetic postganglionic efferent fibers in the papillary nerve were electrically stimulated, the stalk of the fungiform papillae was sucked with a Ringer-filled glass suction electrode (tip internal diameter, 180–220 µm) into which a chlorided silver wire electrode was inserted. The other silver wire electrode, enamel coated except at its tip, was glued to an outer wall of the suction electrode tip. Cathodal pulses were applied at 30 Hz to the papillary nerve fibers.

For gustatory stimulation of frog taste cells, 1 M NaCl, 1 mM acetic acid, 10 mM quinine–HCl (Q-HCl), and 1 M sucrose were used. The first two were dissolved in deionized water. Since taste cell–depolarizing responses for Q-HCl and sucrose are small in the frog (Sato et al., 1995), these chemicals were dissolved in 0.1 M NaCl to remove a membrane hyperpolarization by water as a solvent. The inhibitory effect of 0.1 M NaCl on Q-HCl and sucrose responses of taste cells is weak (Sato and Sugimoto, 1979; Okada et al., 1992). Each taste solution was flowed on the tongue surface at a rate of 0.05 ml/s. The tongue was beforehand adapted to a frog Ringer solution and rinsed with the Ringer solution after taste stimulation. The frog Ringer solution consisted of 115 mM NaCl, 2.5 mM KCl, 1.8 mM CaCl2, and 5 mM 4-(2-hydroxyethyl)piperazine-1-ethanesulfonic acid (HEPES) (pH 7.2).

Drugs

To block the synapses between taste cells and parasympathetic postganglionic efferent fiber terminals (Sato et al., 2004), L-703,606 (cis-2-(diphenylmethyl)-N-[(2-iodophenyl)methyl]-1-azabicyclo[2.2.2]octan-3-amine) oxalate salt (an antagonist of substance P NK-1 receptor) was used. Also, 2004), L-703,606 was prepared with methanol and kept at –20°C. Aliquots of a stock solution were added into a frog Ringer solution to obtain desired concentrations when used. Atropine sulfate and D-tubocurarine chloride were directly dissolved in the Ringer solution. Amount of intravenously (i.v.) injected Ringer solution containing each drug was 2 ml/kg body weight.

Histological examination

The tongue with a pair of the lingual branches of GP nerves was dissected from the animal and immersed for 1–2 h into 0.05–0.1% methylene blue (Sigma, St Louis, MO) dissolved in a Ringer solution to stain the parasympathetic ganglionic cells and unmyelinated efferent fibers in the lingual branches. The extremely small, small, and large nerve bundles in the stained lingual branches (Figure 1A) were cut 3 mm long in various regions inside and outside the tongue. Also, electrical stimulation of the GP nerve was performed with the previously mentioned method (Sato et al., 2002, 2004). In brief, the GP nerve and papillary nerve were electrically stimulated at 30 Hz with pulses of 0.1-ms duration and 15-V strength to obtain the maximal slow HPs from taste cells. When the parasympathetic postganglionic efferent fibers in the papillary nerve were electrically stimulated, the stalk of the fungiform papillae was sucked with a Ringer-filled glass suction electrode (tip internal diameter, 180–220 µm) into which a chlorided silver wire electrode was inserted. The other silver wire electrode, enamel coated except at its tip, was glued to an outer wall of the suction electrode tip. Cathodal pulses were applied at 30 Hz to the papillary nerve fibers.

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Experimental procedure

Strong electrical stimulation of GP nerve elicits a large slow potential on the lingual surface and in the taste disk cells.
This derives from the physicochemical junction potential generated between the saliva from parasympathetically innervated lingual glands and the lingual surface solution (Sato et al., 2000). This potential disturbs an analysis of slow HPs in a taste cell induced by GP nerve stimulation. Therefore, before the start of intracellular recordings from taste cells, atropine sulfate was injected i.v. at a dose of 1 mg/kg to completely block the slow junction potential (Sato et al., 2002, 2004).

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

Anatomical features of parasympathetic postganglionic neurons

The lingual branches of the GP nerves in the frog mostly innervate the whole tongue. The nerve bundles of the lingual branches run intramuscularly from the ventral side of the tongue to the dorsal side and reach the fungiform papillae, lingual glands, and lingual arterioles. The lingual branch is composed of extremely small, small, and large nerve bundles (Figure 1A). Figure 1B exemplifies methylene blue staining of the parasympathetic ganglion cells and unmyelinated efferent fibers in a small bundle of ~160 $\mu$m in diameter (a) and an extremely small bundle of ~20 $\mu$m in diameter (b). Only one cell body is seen in the small bundle (a), but 13 cell bodies are seen in the extremely small bundle (b). All the parasympathetic ganglion cells existed along the nerve bundles, which consisted of unstained myelinated afferent fibers and stained unmyelinated efferent fibers. The numerals in Figure 1A are the numbers of the parasympathetic postganglionic neurons per cubic millimeter of nerve bundles in varying regions of the tongue. The numbers of the parasympathetic postganglionic neurons in small and large nerve bundles (open circles) of the lingual branch were 1.6–12.8/mm$^3$ of these nerve bundles. On the other hand, the number of the parasympathetic postganglionic neurons in extremely small nerve bundles...
(filled circles), which approach the fungiform papillae on the dorsal surface, was 8000–11,000/mm³ of the nerve bundle. The number in the extremely small nerve bundles was on average 1500 times larger than that in larger nerve bundles. No parasympathetic ganglion cells were seen within the fungiform papillae.

The parasympathetic ganglion cell bodies in the tongue were a spheroid with a major axis of 12.7–30.3 μm (mean ± SEM = 21.0 ± 0.4 μm, n = 104) (Figure 2A). The ratio of the minor axis to the major axis was 0.35 ± 0.01 (n = 20). The nucleus of the ganglion cells was a spheroid with a major axis of 7–9 μm (n = 12). The parasympathetic postganglionic fibers had varicosities of 0.9–3.5 μm in diameter spaced 6–12 μm apart (n = 15). The length of parasympathetic postganglionic neurons (fiber and cell body), which was measured from the basement membrane of the taste disk to the cell bodies, was 0.16–3.94 mm (mean ± SEM = 1.45 ± 0.07 mm, n = 267) (Figure 2B). The parasympathetic ef- ferent fibers in the tongue ran parallel to myelinated afferent fibers. Both the parasympathetic pre- and postganglionic fibers were unmyelinated. The diameter of these efferent fibers was <1 μm.

**Effect of blocking activities of parasympathetic ganglion cells and taste cells on slow HPs**

Figure 3A shows a schematic arrangement of afferent and efferent fibers along the fungiform papilla and the lingual branch of the frog GP nerve and of the positions of recording and stimulating electrodes. The nerve fibers running through the papillary nerve of the fungiform papilla are composed of somatosensory and gustatory afferent fibers (Sato, 1976) and sympathetic and parasympathetic efferent fibers (Inoue and Kitada, 1988, 1991). When the GP nerve was electrically stimulated at 30 Hz (Stim A in Figure 3A), a slow HP appeared in taste cells [Figure 3B(a)]. Stimulation of the papillary nerve [Stim B in Figure 3A] elicited the same slow HP in taste cells [Figure 3B(b)]. The mean amplitudes of slow HPs evoked by Stim A and Stim B were −7.5 ± 1.1 mV (n = 22) and −7.7 ± 0.8 mV (n = 28), respectively (Figure 3C). No difference was found between the two values (P > 0.05, n = 22–28).

After α-tubocurarine (an antagonist of nAChR) was i.v. injected at a dose of 1 mg/kg body weight, Stim A did not induce any slow HPs in taste cells but Stim B did. An example of recordings is shown in Figure 3B(c and d), and the mean amplitudes of the slow HPs are summarized in Figure 3C. A further i.v. injection (4 mg/kg) of a substance P NK-1 antagonist, L-703,606, following the α-tubocurarine injection blocked slow HPs in taste cells induced by the papillary nerve stimulation [Figure 3B(e and f), C]. Disappearance of GP nerve–induced slow HPs following α-tubocurarine injection might be due to a blocking of nAChRs on the parasympathetic ganglion cells (Wilson, 1979; Ganong, 2003). Also, disappearance of the papillary nerve–induced slow HPs following L-703,606 injection might be due to a blocking of substance P NK-1 receptors on the postsynaptic membrane in taste cells (Sato et al., 2004).

**Effect of slow HPs on gustatory receptor potential**

We investigated whether the amplitude of receptor potentials in taste cells is modulated by a hyperpolarization of the membrane potential due to a slow HP. An example of recordings is shown in Figure 4A, where the effect of a slow HP on a 1 M NaCl–induced depolarizing receptor potential in a taste cell was tested, while the duration of the slow HP was prolonged by a continuous 30-Hz stimulation of GP nerve.

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**Figure 2** Sizes of parasympathetic postganglionic neurons in the frog tongue. (A) Distribution of major axis of parasympathetic ganglion cell bodies. (B) Distribution of length of parasympathetic postganglionic neurons (fiber and cell body). The arrow in each graph shows the mean.
Figure 4B summarizes the control and test amplitudes of depolarizing receptor potentials induced by four basic taste stimuli of 1 M NaCl, 1 mM acetic acid, 10 mM Q-HCl, and 1 M sucrose, when the mean membrane potentials in taste cells were kept at −30 to 31 mV as controls and were hyperpolarized to −39 to 40 mV by GP nerve stimulation. After the resting potential of taste cells was significantly hyperpolarized by GP nerve–induced slow HPs (P < 0.05, n = 8–15), both depolarizing receptor potentials for 1 M NaCl and 1 M sucrose significantly increased (P < 0.05, n = 8–10) but those for 1 mM acetic acid and 10 mM Q-HCl did not change (P > 0.05, n = 11–15).

Discussion

In general, parasympathetic ganglia are situated near the corresponding organ. The parasympathetic ganglia have been found in the tongue of the frogs, birds, and mammals (Jaeger and Hillman, 1976). Particularly, the parasympathetic ganglion cells in the frog tongue have been observed...
along the nerve bundles of the lingual branches of the GP nerve since the 19th century (Biedermann, 1882; Gaupp, 1904; Jaeger and Hillman, 1976). In earlier histological investigations with light microscopy, the parasympathetic postganglionic fibers were thought to innervate and control the lingual glands (Gaupp, 1904), lingual arterioles (Siggins and Weiten, 1971), and taste organs (K.N. Bhargava and A.K. Bhargava, 1974). Electron-microscopical studies revealed that parasympathetic postganglionic neurons in the frog tongue make synapses with supporting and basal cells in the taste disk of the fungiform papillae (Inoue and Kitada, 1991; Inoue et al., 1992).

Our previous studies have suggested that frog taste cells are innervated by efferent fibers of C type (Sato et al., 2002) and that GP nerve–induced slow HPs in the taste cells are generated by releasing substance P from the efferent fiber terminals (Sato et al., 2004). We investigated the possibility that slow HPs in frog taste cells induced by GP nerve stimulation are generated by excitation of the parasympathetic postganglionic efferent fibers in the GP nerve. In the present study, slow HPs evoked by pulse-train stimulation of the GP nerve completely vanished by i.v. injection of d-tubocurarine. However, no slow HPs evoked by pulse-train stimulation of the papillary nerve, where no parasympathetic ganglion cells are present, vanished even after a d-tubocurarine injection. Since nAChRs on the parasympathetic ganglion cell bodies, which are richly present in the extremely small nerve bundles of the lingual branches of GP nerve, might be blocked by d-tubocurarine (Wilson, 1979; Ganong, 2003), slow HPs were not elicited in taste cells by the stimulation of the parasympathetic preganglionic fibers in the GP nerve but by stimulation of the parasympathetic postganglionic fibers in the papillary nerve. A further injection of a substance P NK-1 antagonist blocked slow HPs induced by the papillary nerve. This suggests that the parasympathetic postganglionic fibers in the papillary nerve are presynaptic fibers in gustatory efferent synapses of the frog taste cells and that substance P is probably released from the postganglionic fiber terminals. A principal transmitter released from the parasympathetic postganglionic fibers is generally acetylcholine, but noncholinergic transmitter agents such as neuropeptides are known to be released from these fibers (Ganong, 2003).

In addition to the parasympathetic preganglionic fibers, there are the somatosensory and gustatory fibers and the sympathetic postganglionic fibers in the lingual branches of the GP nerve of the frog (Sato, 1976; Inoue and Kitada, 1988). Their relationships to the slow HPs must be evaluated. In the present work, after blockage of nAChRs on the parasympathetic ganglion cells, no changes in the membrane potential were elicited in taste cells by strong electrical stimulation of the whole fibers present in the GP nerve. Application of d-tubocurarine might not block activities of sensory and sympathetic postganglionic fibers traveling the lingual branch of the GP nerve because these fibers lack nAChRs (Duncan, 1964; Morimoto and Sato, 1982), but their excitation did not cause slow HPs in taste cells (Figure 3). Therefore, the sensory and sympathetic efferent fibers in the lingual branch of the GP nerve are unlikely to relate to the generation of slow HPs in taste cells. The sympathetic efferent fibers are suggested not to innervate the taste disk but the arterioles in the fungiform papillae (Inoue and Kitada, 1988).

Slow HPs in the frog taste cells induced by GP nerve stimulation may correspond to slow inhibitory postsynaptic potentials at the synaptic membrane. The slow HPs are accompanied by a decrease in the membrane conductance and have a reversal potential of −13 mV (Sato et al., 2002). These characteristics suggest that the slow HPs may be elicited by closing nonselective cation channels, predominantly permeable to K⁺ and Na⁺, existing in the postsynaptic membrane of a taste cell after substance P released from the parasympathetic postganglionic fibers binds to NK-1 receptors on the postsynaptic membrane (Sato et al., 2004). Activation of the NK-1 receptors by substance P causes the excitatory postsynaptic potentials as depolarizing potentials (DPs) in many central neurons (Lewis and Travagli, 2001) but also causes the inhibitory postsynaptic potentials as HPs in some neurons (Ogier and Raggenbass, 2003; Vergnano et al., 2004). As mentioned in our previous study (Sato et al., 2002), slow DPs accompanied by an increase in the membrane conductance are induced in frog taste cells by stimulating GP nerve when blood circulation in the tongue is greatly reduced. This slow DP is blocked by a substance P NK-1 antagonist, L-703,606, suggesting that the slow DP is initiated by substance P from the parasympathetic postganglionic fibers (T. Sato, Y. Okada, and K. Toda, unpublished data).

Since G protein–coupled receptor and IP₃ and DAG as second messengers are involved in transduction cascades following binding of substance P to NK-1 receptors (Ganong, 2003), nonselective cation channels at the postsynaptic membrane of frog taste cell are probably closed in generating slow HPs and opened in generating slow DPs via IP₃ and/or DAG functions. The precise mechanisms for generating the two potentials are still unknown.

In Necturus taste bud, there is a bidirectional synaptic interaction between taste cells and basal cells (Ewald and Roper, 1994). It is assumed that a basal cell activated by excitation of a taste cell releases serotonin (5-HT), which causes both a hyperpolarization of the taste cell accompanied by an increase in the membrane resistance and an enhancement of the amplitude of receptor potential. This evidence seems to suggest that the generation of GP nerve–induced slow HPs in frog taste cells is possibly due to an excitation of basal cells by substance P released from the parasympathetic postganglionic fibers. However, since we penetrated a microelectrode into a taste cell situated in the central part of the taste disk, the distance between the impaled taste cell and the basal cells located at the periphery of the taste disk would be more than 80 μm, so that the possibility that a slow HP in the frog taste...
cell is induced via activation of basal cells is very low. Moreover, even after i.v. injection of 5-HT₁, 5-HT₂, and 5-HT₃ receptor antagonists, no slow HPs in taste cells are blocked (Sato et al., 2004). Therefore, these data suggest that a GP nerve-induced slow HP is directly initiated in the postsynaptic membrane of the taste cell by substance P from the parasympathetic postganglionic fiber terminals.

The mechanisms underlying the generation of receptor potentials in frog taste cells by four basic tastants have been investigated in detail by Sato et al. (1994a, 1995). Receptor currents in frog taste cells induced by the basic taste stimuli are assumed to principally flow through the apical taste receptor membrane of ~1 μm in diameter and the lateral membrane of the dendritic process close to the receptive membrane. On the other hand, slow inhibitory postsynaptic currents relevant to slow HPs in frog taste cells induced by GP nerve stimulation may preferentially flow through the basal processes of taste cells. Interaction between the receptor currents and the slow postsynaptic currents is assumed not to be strong because of the small length constant of taste cells (Ewald and Roper, 1992). The receptor current in taste cells is a function of both the membrane conductance and the membrane potential minus the equilibrium potential of the receptor potential (Kuffler and Nicholls, 1977). A slow hyperpolarization induced by the slow inhibitory postsynaptic current flowing across the basolateral membrane of taste cells will influence a motive force of ion movements across the apical receptive membrane and the lateral membrane in generating a tastant-induced receptor potential. In frog taste cells, depolarizing receptor potentials for strong concentrations of NaCl and sucrose stimuli have a reversal potential of ~+30 mV (inside positive) (Okada et al., 1992; Miyamoto et al., 1993; Sato et al., 1995). Shifting the membrane potential in the hyperpolarizing direction by a GP nerve-induced slow HP increases the electromotive force for cation influx through ion channels of the receptive membrane. Therefore, the amplitude of receptor potentials for NaCl and sucrose would be significantly increased (Figure 4).

Our studies on the generation of receptor potential in the frog taste cells by acid stimuli indicate that 70% of the whole amplitude of 1 mM HCl-induced receptor potential comes from an action of proton-gated cation channels at the receptive membrane and the remaining 30% from an action of proton pump at the receptive membrane (Miyamoto et al., 1988; Okada et al., 1993). The reversal potential for the acid response is as large as +80 mV. Therefore, the amplitude of acid-induced receptor potential under an ~10-mV hyperpolarization by GP nerve stimulation was not significantly increased because of a gentle slope in the relation between membrane potentials and receptor potentials (Figure 4).

Ozeki (1971) first studied the mechanism underlying the generation of a receptor potential in rat taste cell by quinine. He proposed that the quinine-induced receptor potential in a rat taste cell is generated by blocking the resting potassium conductance and obtained a reversal potential of ~–80 mV for the quinine response. Then, three different mechanisms have been proposed for quinine responses in amphibian taste cells: (i) inhibition of voltage-activated K⁺ channels (Akaike et al., 1976; Avenet and Lindemann, 1987; Kinnamon and Roper, 1988; Sugimoto and Teeter, 1991), (ii) active chloride transport (Okada et al., 1988; Sato et al., 1994b), and (iii) activation of cation channels (Tsunenari et al., 1996; Tsunenari and Kaneko, 2001). Although an inhibition of voltage-activated K⁺ channels situated at the apical receptive membrane is proposed as the mechanism of quinine response, no reversal points for quinine responses are measured in intact gustatory preparations (Akaike et al., 1976, Okada et al., 1988; Sato et al., 1994b). Okada et al. (1988) found that the amplitude of quinine-induced taste cell response in frogs does not change depending on K⁺ concentration in the superficial fluid outside the apical receptive membrane but increases depending on a decrease in the superficial Cl⁻ concentration and on an increase in intracellular Cl⁻. Therefore, they proposed active chloride transport as a quinine response mechanism.

In patch clamp studies with isolated frog taste cells, a large depolarizing receptor potential of 60 mV and a large inward current of 2000 pA are obtained by quinine stimulation (Tsunenari et al., 1996). This large DP accompanied by a large increase in the membrane conductance is thought to be generated by quinine-activated cation channels. In the study of quinine responses in taste cells with intact frog preparations, it has been found that the response amplitude is several millivolts, the membrane conductance is always decreased, and no reversal point for quinine responses appears (Okada et al., 1988). These quinine response characteristics are quite different from those obtained from isolated taste cells and patch membranes by Tsunenari et al. (1996). Therefore, it is very likely that the large receptor potential and current found in frog taste cells originate from quinine activation of the cation channels existing in the basolateral membrane. Since no reversal potential of quinine-induced depolarization in frog taste cells is found in our study, it is reasonable that the amplitude of quinine responses does not change either under the resting membrane potential or under the ~10-mV-hyperpolarized membrane potential evoked by GP nerve stimulation.

In conclusion, slow HPs in frog taste cells, which resemble the behavior of slow inhibitory postsynaptic potentials, are induced by activities of parasympathetic efferent fibers in the lingual branch of the GP nerve. Depolarizing receptor potentials in taste cells for salts and sugars are enhanced when the membrane potential in the taste cells is hyperpolarized by slow HPs.

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