Interaction between Gustatory Depolarizing Receptor Potential and Efferent-Induced Slow Depolarizing Synaptic Potential in Frog Taste Cell

Toshihide Sato · Kazuhisa Nishishita · Yukio Okada · Kazuo Toda

Abstract  Electrical stimulation of parasympathetic nerve (PSN) efferent fibers in the glossopharyngeal nerve induced a slow depolarizing synaptic potential (DSP) in frog taste cells under hypoxia. The objective of this study is to examine the interaction between a gustatory depolarizing receptor potential (GDRP) and a slow DSP. The amplitude of slow DSP added to a tastant-induced GDRP of 10 mV was suppressed to 60% of control slow DSPs for NaCl and acetic acid stimulations, but to 20-30% for quinine·HCl (Q·HCl) and sucrose stimulations. On the other hand, when a GDRP was induced during a prolonged slow DSP, the amplitude of GDRPs induced by 1 M NaCl and 1 M sucrose was suppressed to 50% of controls, but that by 1 mM acetic acid and 10 mM Q·HCl unchanged. It is concluded that the interaction between GDRPs and efferent-induced slow DSPs in frog taste cells under hypoxia derives from the crosstalk between a gustatory receptor current across the receptive membrane and a slow depolarizing synaptic current across the proximal subsynaptic membrane of taste cells.

T. Sato (✉) · Y. Okada · K. Toda
Division of Integrative Sensory Physiology, Nagasaki University Graduate School of Biomedical sciences, Nagasaki 852-8588, Japan  e-mail: toshi@net.nagasaki-u.ac.jp
Keywords Basic taste stimuli • Gustatory efferent synapse • Parasympathetic nerve • Receptor potential • Slow depolarizing synaptic potential • Frog taste cell

Introduction

Sensory output information is controlled by efferent fibers supplying the sensory cells. Efferent synaptic control of sensory cells is known in hair cells in auditory and vestibular organs (Furukawa 1981) and stretch receptor cells in crustacean muscles (Eyzaguirre and Kuffler 1955). The existence of efferent synapses on taste cells is suggested by electron-microscopic studies (Murray 1971; Nomura et al. 1975; Witt 1993; Yoshie et al. 1996; Reutter et al. 1997). Our previous studies (Sato et al. 2002, 2004, 2005, 2007b, c) showed that electrical stimulation of the parasympathetic nerve (PSN) efferent fibers in the frog glossopharyngeal nerve (GPN) induces either a slow hyperpolarizing synaptic potential (HSP) under normoxia or a slow depolarizing synaptic potential (DSP) under hypoxia. These potentials correspond to a slow inhibitory postsynaptic potential and a slow excitatory postsynaptic potential. At the normal velocity (1-1.5 mm/s) of capillary blood flow in the tongue, slow HSPs alone are elicited in frog taste cells, but at the declined blood flow velocity of < 0.2 mm/s slow DSPs alone are elicited (Sato et al. 2002, 2007b, c).
Under normal blood circulation in the tongue, all gustatory depolarizing receptor potentials (GDRPs) in frog taste cells evoked by four basic taste stimuli are depressed by adding PSN-induced slow HSPs (Sato et al. 2007a). Thus suppression is due to an inhibition of depolarizing receptor currents across the apical receptive membrane by slow hyperpolarizing synaptic currents across the proximal subsynaptic membrane of taste cells. On the other hand, when GDRPs are evoked during prolonged generation of a slow HSP, the GDRPs for NaCl and sucrose stimuli are enhanced, but the GDRPs for Q-HCl and acetic acid stimuli do not change (Sato et al. 2005). The enhancement of the NaCl and sucrose responses is due to an increase of the driving force for cation entry across the apical receptive membrane.

The objective of the present work is to examine the interaction between tastant-induced GDRPs and PSN efferent-induced slow DSPs in frog taste cells under hypoxia. The experiments were conducted in two issues: i) change in slow DSPs added to tastant-induced GDRPs and ii) change in the GDRPs added to slow DSPs.

The blood and interstitial fluid of bullfrogs which do not hibernate are under hypoxia in winter season because of a great decrease in the respiratory and circulatory activity (Prosser and Brown 1965). In this situation PSN efferent activity from the medulla oblongata is likely to induce slow DSPs in taste cells. Therefore, the present study is of a physiological significance.

**Materials and Methods**

Preparation, recording and stimulation
Adult bullfrogs (*Rana catesbeiana*) were used. All experiments were carried out in accordance with the Guidance of Animal Experimentation in Nagasaki University. The methods of making preparation, electrical recording from taste cells, electrical stimulation of GPN and chemical stimulation of taste cells were the same as previously mentioned (Sato et al. 2002, 2004, 2005, 2007a). In brief, intracellular recordings from taste cells in the taste disks of fungiform papillae were made with a 3 M KCl filled-microelectrode. The fungiform papillae used were located at the apical and middle portions of the tongue. The microelectrode was put at the surface of the central area of taste disk and advanced into the lower portion of intermediate layer (Osculati and Sbarbati 1995) by monitoring appearance of three step-potential changes of the membrane potentials in taste disk cells (Sato et al. 2007a). Both GPNs were separated free from the connective tissues, cut centrally and immersed into mineral oil. The distal portion of cut GPN was mostly stimulated at 30 Hz by electric pulses of 0.1 ms-duration and 15 V-strength to obtain the maximal slow synaptic potential changes from taste cells. In some experiments the GPN was stimulated at 1-10 Hz. All experiments were carried out at room temperature of 23-26°C.

**Experimental procedure**

The heart rate and the velocity of blood flow in bullfrogs spontaneously declined after anesthesia with a urethane solution at a dose of 2-4 g/kg body weight. The blood flow velocity through the capillaries in the fungiform papillae was measured with the same method as described previously (Sato et al. 2007c). The velocity of the lingual capillary blood flow was at 0.7-1.5 mm/s for 4 h after start of anesthesia and PSN stimulation
induced slow HSPs alone. The velocity of lingual blood flow became at 0.2-0.7 mm/s for 1 h after 4 h-anesthesia and PSN induced biphasic slow potentials composed of slow DSPs and slow HSPs. Then, the velocity of the blood flow declined to < 0.2 mm/s after 5 h-anesthesia and PSN induced slow DSPs alone (Sato et al. 2007c). Estimated venous Po2 (oxygen tension) was < 5 mmHg (< 20% of the control) (Prosser and Brown 1965). All the experiments in the present work were carried out under this situation in order to examine the interaction between slow DSPs and GDRPs in taste cells.

To eliminate the large physicochemical junction potentials which are induced between a GPN-induced lingual saliva and a lingual surface solution (Sato et al. 2000), atropine sulfate (Sigma-Aldrich, St Louis, MO) was intravenously injected at a dose of 1 mg/kg before start of all the experiments.

Solutions

A frog Ringer solution was composed of 115 mM NaCl, 2.5 mM KCl, 1.8 mM CaCl₂ and 5 mM HEPES [4-(2-hydroxyethyl)-piperazine-1-ethanesulfonic acid]. The pH was adjusted to 7.2 by a Tris [tris(hydroxymethyl)aminomethane] buffer. Taste solutions used were 0.1-1 M NaCl, 0.1-1 mM acetic acid, 1-10 mM quinine-HCl (Q-HCl) and 0.1-1 M sucrose. The last two chemicals were dissolved in 0.1 M NaCl to avoid hyperpolarizing responses of taste cells by water as a solvent. The inhibitory effects of 0.1 M NaCl on Q-HCl and sucrose responses of frog taste cells are weak (Sato and Sugimoto 1979; Okada et al. 1992). The Ringer and taste solutions were flowed on the tongue surface at a rate of 0.05 ml/s through a polyethylene tube with a stimulus-delivering port. The tongue surface was always adapted to the Ringer
solution. Usually, one type of taste stimulus was applied to single taste cells. When various concentrations of a stimulus were tested, the lower ones were first applied.

Statistics

All experimental data were expressed as means ± standard errors of means (SEMs). The level of significance was set at $P < 0.05$ with a Student’s $t$-test.

Results

Characteristics of Slow DSP

At declined capillary blood flow velocity of $< 0.2$ mm/s slow DSPs alone are evoked in taste cells (Sato et al. 2002, 2007c). Fig. 1A shows an example of slow DSP elicited by 5 s-stimulation of PSN at 30 Hz. The mean amplitude of resting potentials and slow DSPs was $-32 \pm 1$ mV ($N = 83$) (B) and $7.7 \pm 0.5$ mV ($N = 83$) (C), respectively. The resting potentials under hypoxia were the same as those under normoxia ($P > 0.05$, $N = 70-83$) (Sato et al. 2007a). The input resistance of taste cells during a generation of slow DSPs was always reduced as shown in reduction of the amplitudes of pulse trains superimposed on the membrane potentials (Fig. 1A). The mean reduction of input resistance during slow DSPs was $69 \pm 2\%$ ($N = 78$) (D). The latency and peak time when stimulated at 30 Hz for 5 s were $1.8 \pm 0.2$ s ($N = 83$) (E) and $4.9 \pm 0.9$ s ($N = 83$) (F), respectively. The shorter the latency, the shorter the peak time was. The fall time of slow DSPs became longer with increasing stimulation time of PSN. The fall time
observed by 5 s stimulation was $17 \pm 1$ s ($N = 45$).

Membrane resistance change and reversal potential of GDRP

Change in the input resistance and the reversal potential of GDRPs are unknown in taste cells at a hypoxic level. The cell input resistance was decreased to 50-60% of controls ($N = 8$) during 1 M NaCl stimulation, 70-89% ($N = 6$) during 1 mM acetic acid stimulation and 90-95% ($N = 6$) during 1 M sucrose. The input resistance was increased to 105-110% of control ($N = 9$) during 10 mM Q-HCl stimulation. The membrane potential was altered at a range of $\pm 50$ mV by intracellularly injecting constant currents. The reversal potentials were 9-18 mV ($N = 3$) for 1 M NaCl and 9-20 mV ($N = 3$) for 1 M sucrose. The reversal potential for 1 mM acetic acid ($N = 2$) and 10 mM Q-HCl ($N = 3$) were unmeasured.

Summation of GDRP and slow DSP

When a PSN-evoked slow DSP under hypoxia was added to a tastant-induced GDRP, both depolarizing responses were summated. First, the effect of slow DSPs elicited by PSN stimulation at 1-30 Hz on GDRPs was examined. As shown in an example of Fig. 2A, the amplitude of slow DSPs added to the 1 M NaCl-induced GDRP was gradually increased with increasing frequency of PSN stimulation. In Fig. 2B-E, the relationships between summated responses of GDRPs and slow DSPs and frequency of PSN stimulation are shown. When a slow DSP induced by 30 Hz PSN stimulation was added to the large amplitude of 1 M NaCl· and 1 mM acetic acid·induced GDRPs, the
percentage of the added slow DSP was 28% of the amplitude of either GDRP (Fig. 2B and C). On the other hand, when the slow DSP was added to the small amplitude of 10 mM Q-HCl and 1 M sucrose-induced GDRPs, the percentage of the added slow DSP was 84% and 137% of the respective GDRPs (Fig. 2D and E).

Suppression of slow DSP added to GDRP

In Fig. 3, as the amplitude of GDRPs for NaCl (A) and sucrose (B) stimuli was increased, the amplitude of slow DSPs added to GDRPs decreased. These relations are plotted in Fig. 4 A-D with various concentrations of 4 basic taste stimuli. Without taste stimulation, the amplitudes of control slow DSPs were 9-10 mV, which did not differ among taste cells with different sensitivities ($P > 0.05$, $N = 5-9$). The sizes of GDRPs in taste cells under hypoxia induced by 1 M NaCl, 1 mM acetic acid, 10 mM Q-HCl and 1 M sucrose were the same as those in taste cells under normoxia by the 4 stimuli ($P > 0.05$, $N = 5-11$) (Sato et al. 2007a). When the amplitude of slow DSPs added to GDRPs was plotted against the amplitude of the membrane potentials at plateau level of the GDRPs (dashed lines in Fig. 3), a linear relation was found between the two amplitudes. Figure 5 illustrates these representative relations in 4 taste cells in Fig. 4. The 4 leftmost points in this figure are control slow DSPs without taste stimulation and the other points are test slow DSPs added to GDRPs. The amplitudes of slow DSPs at the membrane potential of 20-22 mV are the slow DSPs added to 10 mV-GDRPs. It is seen that test slow DSPs added to GDRPs of equivalent 10 mV are larger for NaCl and acetic acid stimulations than for Q-HCl and sucrose stimulations.
Similar analyses from a total of 29 taste cells in Fig. 4 indicated that the amplitudes of slow DSPs added to 10 mV-GDRPs were suppressed to 61 ± 5% (N = 5) of control for NaCl, 63 ± 10% (N = 9) for acetic acid, 20 ± 1% (N = 9) for Q-HCl and 35 ± 8% (N = 6) for sucrose. The 4 straight lines through the experimental points in Fig. 5 indicate that the reversing points of slow DSPs added to the GDRPs elicited by Q-HCl and sucrose stimulations are -12--13 mV, but those of slow DSPs added to the GDRPs by NaCl and acetic acid stimulations are -2-0 mV. The same two different values of reversing points were obtained from the other taste cells in Fig. 4. The reversing point of -12--13 mV for slow DSPs during Q-HCl and sucrose stimulations was the same as the reversal potential measured by intracellularly injected electric currents (Sato et al. 2002, 2007a). However, the reversing point of -2-0 mV for slow DSPs during NaCl and acetic acid stimulations was quite different from that by the electric currents.

Suppression of GDRP added to slow DSP

As shown in Fig. 6, the amplitude of GDRP evoked by 1 M NaCl (A) during a prolonged generation of PSN-induced slow DSP became smaller than the amplitude of control without the slow DSP, but the amplitude of 1 mM acetic acid-induced GDRP (B) summated to the prolonged DSP remained unchanged. Fig. 7 summarized the control and test amplitudes of GDRPs for 1 M NaCl, 1 mM acetic acid, 10 mM Q-HCl and 1 M sucrose before and during generation of slow DSPs. The GDRPs for 1 M NaCl and 1 M sucrose were significantly suppressed by ~50% during slow DSPs (both
responses: $P < 0.05$, $N = 11$), but those for 1 mM acetic acid and 10 mM Q-HCl remained unchanged during slow DSPs (both responses: $P > 0.05$, $N = 11$). The suppression of GDRPs for both 1 M NaCl and 1 M sucrose became larger with increasing amplitude of slow DSPs (Fig. 8A), but the amplitudes of GDRPs for 1 mM acetic acid and 10 mM Q-HCl were independent of the amplitude of slow DSPs (Fig. 8B).

**Discussion**

Although the existence of efferent synapse in taste cells has been suggested since 1971 by electron-microscopical studies (Murray 1971), the first electrical recording of the postsynaptic potential at gustatory efferent synapse was made in 2002 from frog taste cells following the GPN stimulation (Sato et al. 2002).

When the velocity of capillary blood flow under taste disks of the frog fungiform papillae is at 0.7-1.5 mm/s, PSN-induced responses in taste cells are slow HSPs alone. On the other hand, slow DSPs alone appear when the blood flow velocity declines to $< 0.2$ mm/s (Sato et al. 2002, 2007c). Our previous studies suggested that a slow HSP might be generated by closing a nonselective cation channel via a second messenger diacylglycerol (DAG). On the other hand, a slow DSP might be generated by opening another nonselective cation channel via inositol 1,4,5-trisphosphate (IP$_3$) because of lacking in O$_2$ under hypoxia (Sato et al. 2007c). The physiological characteristics of GDRPs in frog taste cells under hypoxia evoked by basic taste stimuli are almost the same as those obtained at the normoxic level (Akaike et al. 1976; Sato et al. 1995,
2007a). For example, the resting potential, size of GDRP, change in input resistance and reversal potential for 4 basic stimuli are equivalent between taste cells under normoxia and hypoxia. Slow synaptic potentials in taste cells induced by PSN efferent stimulation are very sensitive to a $P_{O_2}$ level, but GDRPs are relatively resistive to a low $P_{O_2}$ level. Therefore, the data of GDRPs investigated under normoxia are useful in discussing the mechanisms underlying suppression of slow DSPs added to GDRPs as well as suppression of GDRPs added to slow DSPs.

We have proposed ionic mechanisms underlying a generation of receptor potentials in frog taste cells for basic taste stimuli (Sato et al. 1994, 1995). When NaCl and acetic acid induce receptor potentials in frog taste cells, cation channels at the receptive membrane are opened. The part of these receptor currents is carried by Na$^+$ (Miyamoto et al. 1988, 1993; Okada et al. 1994, Sato et al. 1995). It has been proposed that slow DSPs in taste cells are induced by opening nonselective cation channels permeable to Na$^+$ and K$^+$ of ~30 pS on the proximal process membranes of taste cells (Fujiyama et al. 1993; Sato et al. 2002, 2007c). When a slow DSP appears during generating a tastant-induced GDRP, an interaction will occur between a gustatory receptor current across the apical receptive membrane and a slow depolarizing synaptic current across the proximal subsynaptic membrane of taste cells. Therefore, the reversing point of slow DSPs during NaCl and acetic acid stimulations is likely to be greatly shifted to the direction of Na$^+$ equilibrium potential as an electromotive force of their receptor potentials. On the other hand, it has been proposed that Q-HCl responses in frog taste cells are induced by Cl$^-$ pump at the receptive membrane (Okada et al. 1988) and that sucrose responses are induced by H$^+$ entry across the receptive membrane (Okada et al. 1992). Therefore, the reversing points of slow DSPs
added to GDRPs for Q·HCl and sucrose (Fig. 5) were not influenced by the Na⁺ equilibrium potential, and were equivalent to the reversal potential of $-11 \pm 2$ mV measured by electrical currents (Sato et al. 2002).

The amplitude of a slow DSP added to a GDRP of 10 mV was suppressed to 60% of the control when stimulated with NaCl and acetic acid but to 20-30% when stimulated with Q·HCl and sucrose. These data derive from two different reversing points of slow DSPs added to GDRPs (Fig. 5). This suggests that the crosstalk between receptor current passing the apical membrane and the slow depolarizing synaptic current passing the proximal subsynaptic membrane is weak when slow DSPs are summated to GDRPs of Q·HCl and sucrose but strong when slow DSPs are summated to GDRs of NaCl and acetic acid.

Slow DSPs added to GDRSs may be related to the enhancement of gustatory neural responses. This effect is larger for NaCl and acetic acid stimulations than for Q·HCl and sucrose stimulations.

On the other hand, a GDRP added to a sustained slow DSP is suppressed when stimulated with 1 M NaCl and 1 M sucrose, but remains unchanged when stimulated with 1 mM acetic acid and 10 mM Q·HCl. The amplitude of gustatory receptor current across the receptive membrane is a function of the membrane potential minus the equilibrium potential of gustatory receptor potential (Kuffler and Nicholls 1977). The equilibrium potential of strong NaCl and sucrose stimuli-induced GDRPs is ~20 mV (Okada et al. 1992; Miyamoto et al. 1993; Sato et al. 1995). Therefore, a decrease of the membrane potential in a taste cell by a slow DSP results in a decrease of the GDRPs for NaCl and sucrose because of a reduction of the driving force for Na⁺ entry. Because the reduction of GDRPs for NaCl and sucrose depends on the size of the slow DSPs, a
larger reduction of GDRPs added to a slow DSP is due to a larger decrease of driving force of cation passing through the receptive membrane. On the other hand, the equilibrium potential of Q-HCl-induced GDRP does not exist (Okada et al. 1988) and that for acetic acid-induced GDRP is larger than 90 mV (Miyamoto et al. 1988; Sato et al. 1995). Therefore, the GDRPs for Q-HCl and acetic acid may not be affected by a small decrease of the membrane potential by a slow DSP.

A taste cell is a presynaptic cell and a gustatory afferent fiber is a postsynaptic neuron. Since the PSN efferent fiber makes synaptic contact with the presynaptic taste cell in frogs (Sato et al. 2005), the suppression of NaCl- and sucrose-induced GDRPs in taste cells by slow DSPs may be a type of presynaptic inhibition that is well-known in the central nervous system (Eccles 1964). With decreasing Po2 in blood due to a decrease in the capillary blood flow velocity of < 0.2 mm/s, most of the oxygen-sensitive ion channels are gradually inhibited to reduce tissue activities. This results in avoiding a loss of metabolic energies under hypoxia (López-Bameo et al. 2001). Suppression of GDRPs by PSN-induced slow DSPs under hypoxia also plays an important role in avoiding energy loss in taste organ.

In conclusion, interaction between a tastant-induced GDRP and a PSN-induced slow DSP in a taste cell is derived from the crosstalk between a gustatory depolarizing receptor current across the apical receptive membrane and a slow depolarizing synaptic current across the proximal subsynaptic membrane. Addition of a slow DSP to NaCl- and acetic acid-induced GDRPs induces stronger interaction between the two currents, but addition of NaCl- and sucrose-induced GDRPs to a prolonged slow DSP induces stronger interaction between the two currents.
**Acknowledgements** This work was supported by Japan Society for the Promotion of Science (17570064).

**References**


JC Eccles The Physiology of Synapses, Springer, Berlin, 1964

Eyzaguirre C, Kuffler SW (1955) Processes of excitation in the dendrites and the soma of single isolated sensory nerve cells of the lobster and crayfish. J Gen Physiol 39: 87-119


SW Kuffler, J Nicholls From Neuron to Brain, Sinauer Associates, Sunderland (MA), 1977


Murray RG (1971) Ultrastructure of taste receptors. In Beidler LM (ed), Handbook of


CL Prosser · FA Brown Comparative Animal Physiology, 2nd ed, Philadelphia, WB Saunders


Sato T, Nishishita K, Mineda T, Okada Y, Toda K (2007a) Depression of gustatory receptor potential in frog taste cell by parasympathetic nerve-induced slow hyperpolarizing potential. Chem Senses 32: 3-10

potential in frog taste cell induced by parasympathetic efferent stimulation under hypoxia. Chem Senses 32: 329-336


Legends

**Fig. 1** Characteristics of PSN-induced slow DSPs in frog taste cells. (A) A slow DSP in a taste cell. To measure membrane resistance hyperpolarizing pulse trains were superimposed on membrane potential. ES, Electrical stimulation of PSN at 30 Hz. Resting potential was –30 mV. (B)-(F) Histograms of resting potentials in taste cells inducing slow DSPs (B), PSN-induced slow DSPs (C), input resistance changes during generation of slow DSPs (control, 100%) (D), latencies of slow DSPs (E) and peak times of slow DSPs (F). Means ± SEMs were as follows: Resting potential, -32 ± 1 mV (N = 82); Slow DSP, 7.7 ± 0.5 mV (N = 83); Input resistance, 67 ± 2% (N = 78); Latency, 1.8 ± 0.2 s (N = 83); Peak time, 4.9 ± 0.9 s (N = 83). Tongue was adapted to frog Ringer solution.

**Fig. 2** Effects of slow DSPs evoked by PSN stimulation at various frequencies on GDRPs. (A) PSN-induced slow DSPs summated to 1 M NaCl-evoked GDRPs in a taste cell. Stimulus frequencies applied to PSN were 30, 10, 5, 1, 0 Hz from top of slow DSP to bottom in traces. ES: electrical stimulation of PSN. Resting potential was –32 mV. (B)-(E) Effects of slow DSPs elicited by PSN stimulation on GDRPs induced by 1 M NaCl (B), 1 mM acetic acid (C), 10 mM Q-HCl (D) and 1 M sucrose (E). Horizontal dashed lines denote means of GDRPs. Vertical bars in this and other figures are SEMs. Resting potentials: –30 ± 1 mV (N = 9) (B), –32 ± 1 mV (N = 6) (C), –32 ± 1 mV (N = 6) (D) and –30 ± 1 mV (N = 9) (E).
**Fig. 3** Amplitude of slow DSPs added to GDRPs induced by different concentrations of stimuli. (A) NaCl. (B) Sucrose. All data were obtained from two taste cells. ES, electrical stimulation of PSN at 30 Hz. Horizontal dashed lines indicate receptor potential level. Resting potential was −29 mV (A) and −32 mV (B).

**Fig. 4** Effects of different concentrations of taste stimulus on GDRPs and slow DSPs added to the GDRPs. (A) 0.1, 0.3, 1 M NaCl. (B) 0.1, 0.3, 1 mM acetic acid. (C) 1, 3, 10 mM Q·HCl. (D) 0.1, 0.5, 1 M sucrose. Left ordinate is amplitude of GDRP and right ordinate is amplitude of slow DSPs added to GDRPs. Resting potential was −30 ± 0 mV (N = 5) (A), −32 ± 1 mV (N = 9) (B), −31 ± 0 mV (N = 9) (C) and −31 ± 1 mV (N = 6) (D).

**Fig. 5** Relationship between amplitude of membrane potential at plateau level of tastant-induced GDRP and amplitude of slow DSP added to the GDRP. Each straight line was obtained from data of single taste cells in Fig. 4. Taste stimuli and concentrations used are the same as mentioned in Fig. 4. Resting potential was −32 mV (X) and −30 mV (O, ▽, □).

**Fig. 6** GDRPs induced during prolonged generation of slow DSP. (A) Control (left) and test (right) GDRPs for 1 M NaCl. (B) Control (left) and test (right) GDRPs for 1 mM acetic acid. (A) and (B) were obtained from different taste cells having resting potential of −30 mV and −31 mV. ES: electrical stimulation of PSN at 30 Hz.
**Fig. 7**  GDRPs induced by 4 basic taste stimuli before (control) and during prolonged generation of slow DSPs. Numerals above columns are number of taste cells tested. Resting potential was –31 ± 1 mV (N = 22). Slow DSPs induced by PSN stimulation at 30 Hz were 8.0 ± 0.9 mV (N = 22).

**Fig. 8**  Relationship between slow DSPs and GDRPs added to the slow DSPs. (A) Relationship between amplitude of prolonged slow DSPs induced by PSN stimulation at 30 Hz and amplitude of GDRPs for 1 M NaCl and 1 M sucrose added to the slow DSPs. (B) Relationship between amplitude of slow DSPs and amplitude of GDRPs for 10 mN Q·HCl and 1 mM acetic acid added to the slow DSPs. Resting potential was –31 ± 1 mV (N = 22). Slow DSPs were 8.0 ± 0.9 mV (N = 22).
Fig. 1
Fig. 2

A

ES

1 M NaCl

20 s

10 mV

B

N = 9

1 M NaCl

C

N = 6

1 mM acetic acid

D

N = 6

10 mM Q-HCl

E

N = 9

1 M sucrose

Stimulus frequency (Hz)

Summation of GDRP and slow DSP
Fig. 3
Fig. 4
Membrane potential (mV)

-35 -30 -25 -20 -15 -10 -5 0 5

Slow DSP (mV)

-5 0 5 10 15 20

Fig. 5

- NaCl
- acetic acid
- Q-HCl
- sucrose
Fig. 6
Fig. 7

GDRP (mV)

- 1 M NaCl
- 1 M sucrose
- 10 mM Q-HCl
- 1 mM acetic acid

control

test
Fig. 8