J. exp. Biol. **162**, 23–36 (1992)
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THE IONIC BASIS OF THE RECEPTOR POTENTIAL OF FROG TASTE CELLS INDUCED BY SUGAR STIMULI

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Accepted 17 September 1991

Summary

The ionic mechanism underlying the receptor potential in frog taste cells induced by sugar stimuli was studied with conventional microelectrodes by replacing the superficial and interstitial fluids of the tongue with modified solutions. The taste cell generated a depolarizing receptor potential accompanying a remarkable reduction of input resistance in response to stimulation with galactose and sucrose. The magnitude of the receptor potential in response to galactose solution increased linearly with decreasing pH in the pH range 6–8, but remained constant above pH 8. The reversal potential was increased by only 29 mV by a 10-fold increase in the H^+ concentration of the stimulus, suggesting that there are pH-dependent and pH-independent components in the mechanism generating the receptor potential. The use of Na^+-free, Ca^{2+}-free and K^+-free interstitial fluids did not affect the receptor potential, but the elimination of Cl^- from the interstitial fluid largely abolished it. Interstitial 0.1 mmol l^{-1} *N,N'-dicyclohexyl-carbodiimide* (DCCD) completely inhibited the receptor potential and interstitial 0.1 mmol l^{-1} *N*-ethylmaleimide (NEM) decreased the potential to 40 % of the control value. Lowering the pH of interstitial fluid from 7.2 to 6.3 decreased the receptor potential to 30 % of the control value. It is concluded that part of the receptor potential in frog taste cells induced by sugar stimuli may be produced by an inflow of H^+ through the taste-receptive membrane. The intracellular pH of the taste cell may be regulated by a Cl^--dependent H^+ pump in the basolateral membrane.

Introduction

Single taste cells of vertebrates respond to various taste stimuli with depolarizing receptor potentials (Sato, 1986; Avenet and Lindemann, 1989). Recent patch-clamp studies on isolated taste cells have revealed that there are a variety of ionic channels on the cell membranes (Avenet and Lindemann, 1987; Kinnamon and Roper, 1988; Miyamoto *et al.* 1988*a*; Spielman *et al.* 1989). When taste stimuli are

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Key words: frog taste cell, receptor potential, sugar stimuli, *Rana catesbeiana*.

applied to the bathing solution of isolated taste cells, tastants such as sugar and salt may cause rather nonspecific effects, such as cell shrinkage, and tastants such as acid and quinine directly block ionic channels in the basolateral membrane.

With conventional intracellular recordings, measurements of the input resistance of a taste cell in response to sugar stimuli have produced anomalous results. Some studies have shown it to be reduced in frog and rat (Akaike and Sato, 1976; Ozeki, 1971) and unchanged in mouse (Tonosaki and Funakoshi, 1984*a*). Other reports have shown an increase in input resistance in rat and mouse (Sato and Beidler, 1982; Tonosaki and Funakoshi, 1984*b*). The reversal potentials for the sugar-induced receptor potential have been calculated in the different membranes (Ozeki, 1971; Tonosaki and Funakoshi, 1984*a*). These different results suggest that the mechanism underlying the depolarization in a taste cell induced by sugar stimulation varies from animal to animal.

Studies using an Ussing chamber have indicated that a transepithelial sodium current is induced in canine lingual epithelium by sugar stimuli. The current is completely blocked by amiloride (Mierson *et al.* 1988; Simon *et al.* 1989). However, the gustatory neural response induced by sugar stimuli is not greatly inhibited by the drug (Nakamura and Kurihara, 1990). The frog glossopharyngeal nerve generates gustatory impulses in response to sugar stimuli (Akaike and Sato, 1976; Miyake *et al.* 1976), but frog lingual epithelium does not generate transepithelial currents in response to sugar (Soeda and Sakudo, 1985). This suggests that the transepithelial current induced by sugar cannot be directly related to the gustatory nerve impulses.

The present study was undertaken to investigate the ionic mechanism underlying generation of the receptor potential induced by sugar stimuli, by separately replacing the superficial and interstitial fluids of the tongue.

Part of this study has been published in abstract form (Okada *et al.* 1986, 1987).

Materials and methods

Preparation

Fifty-one bullfrogs (*Rana catesbeiana*) weighing 224–568 g were used for the experiments over the course of a year. The animals were anaesthetized with an intraperitoneal injection of 50 % urethane–Ringer's solution at $3\,g\,kg^{-1}$ body mass. To prevent spontaneous contraction of the tongue, the hypoglossal nerve and the hyoglossal muscle were cut bilaterally. The tongue was fully pulled from the mouth and the base of the tongue was fixed with steel pins on a cork plate in an experimental chamber.

Recording

The intracellular potential in a taste cell was recorded by inserting a glass capillary microelectrode (20–50 MΩ) filled with $3\,mol\,l^{-1}$ KCl into the taste disc of the fungiform papillae scattered on the tongue. A glass capillary (100–150 μm in tip diameter) filled with $3\,mol\,l^{-1}$ KCl/3 % agar was used as an indifferent

electrode. The electrode was placed on the tongue surface in the experiments where the superficial fluid on the tongue was replaced with various solutions and was inserted into the lingual muscle in the experiments where the interstitial fluid of the tongue was replaced.

The microelectrode was connected to a preamplifier (DPZ-16A, Dia Medical System, Tokyo) and the membrane potential was recorded on a pen recorder. When the input resistance and reversal potential for a depolarizing response in a taste cell were measured, a bridge circuit was used for simultaneous current injection and membrane potential recording. The bridge was balanced completely before penetration of the cell to cancel the resistance of the microelectrode. The intensity of the current injected through the microelectrode was monitored by the potential drop across a 10 MΩ resistor.

The whole of the glossopharyngeal nerve on each side was dissected free from the surrounding connective tissue and cut near the hyoid bone. The nerve was placed over bipolar silver wires, for recording the impulses, and immersed in liquid paraffin. The neural impulses were amplified with an a.c. amplifier, integrated at a time constant of 0.3 s and recorded using a pen recorder.

Solutions

The ionic compositions of the superficial and interstitial fluids used in experiments on the tongue are listed in Table 1. *N*-ethylmaleimide (NEM, Sigma), Na_3VO_4 (Wako) and acetazolamide (Sigma) were dissolved in normal saline solution. *N*,*N'*-dicyclohexyl-carbodiimide (DCCD, Sigma) was dissolved in nor-

Table 1. *Ionic compositions of solutions*

Constituent ($mmol\,l^{-1}$)	Normal	Na^+-free	Ca^{2+}-free	K^+-free	Cl^--free (gluconate)	Cl^--free (Br^-)
NaCl	115	–	115	117.5	–	–
KCl	2.5	2.5	2.5	–	–	–
$CaCl_2$	1.8	1.8	–	1.8	–	–
Choline chloride	–	115	–	–	–	–
$MgCl_2$	–	–	1.8	–	–	–
Sodium gluconate	–	–	–	–	115	–
K_2SO_4	–	–	–	–	1.25	–
$CaSO_4$	–	–	–	–	1.8	–
NaBr	–	–	–	–	–	115
KBr	–	–	–	–	–	2.5
$CaBr_2$	–	–	–	–	–	1.8

All solutions were buffered with $5\,mmol\,l^{-1}$ Hepes.

The pH of all solutions was adjusted to 7.2 with NaOH or tetramethylammonium hydroxide (TMAOH).

In Ca^{2+}-free solution, $0.1\,mmol\,l^{-1}$ EGTA was added to chelate residual Ca^{2+}.

mal saline solution containing 0.5 % ethanol. The interstitial fluids contained 20 mmol l^{-1} glucose and 5 % (w/v) dextran (relative molecular mass about 70 000) (Pharmacia).

As sweet taste stimuli, 0.2–1.0 mol l^{-1} galactose and 1.0 mol l^{-1} sucrose (Wako and Nakarai) were used. Galactose was dissolved in deionized water (reagent grade water, Millipore), to which was added 10 mmol l^{-1} or 100 mmol l^{-1} NaCl, 10 mmol l^{-1} or 100 mmol l^{-1} $NaHCO_3$, and 10 mmol l^{-1} NaCl with 1 mmol l^{-1} amiloride (Merck Sharp and Dohme Research Laboratories), or 10 mmol l^{-1} NaCl with 1 mmol l^{-1} $CuCl_2$, or 10 mmol l^{-1} NaCl with 1 mmol l^{-1} $CdCl_2$, or 10 mmol l^{-1} NaCl with 1 mmol l^{-1} 4-acetamido-4′-isothiocyanostilbene-2,2′-disulphonic acid (SITS, Nakarai). Sucrose was dissolved in 100 mmol l^{-1} NaCl. The pH of galactose solution in 100 mmol l^{-1} NaCl was adjusted with 5 mmol l^{-1} Hepes–NaOH. To examine whether a high osmotic pressure of the solution stimulates the taste cell, 1 mol l^{-1} urea dissolved in 10 mmol l^{-1} NaCl was used as a taste stimulus.

Application of solutions

During intracellular recordings, either an adapting solution of normal saline or a stimulating sugar solution was perfused onto the tongue surface at a rate of 0.1 ml s^{-1}. A detailed description of the method for delivering solutions has already been given (Sato and Beidler, 1975). The exchange of normal interstitial fluid with modified solutions was made using the lingual arterial perfusion method described elsewhere (Okada *et al.* 1985). When measuring the gustatory neural responses elicited by galactose, the adapting and stimulating solutions were perfused onto the tongue surface at a rate of 0.5 ml s^{-1} through a 10 ml syringe.

Experimental procedure

When the effects of test substances added to the galactose solutions were examined, the control and test responses were obtained from the same taste cell or nerve. Since it was technically difficult to hold a stable penetration of a microelectrode in a taste cell for the time it took to replace the interstitial fluid of the tongue completely, the effects of modified interstitial fluids upon the receptor potential induced by galactose were evaluated by comparing the control and test responses obtained from different taste cells in the same tongue.

All the experiments were carried out at room temperature (20–25 °C).

Results

Depolarization induced by sugar stimulation

Fig. 1A shows typical examples of the receptor potentials of a frog taste cell induced by galactose dissolved in deionized water after the tongue surface had been adapted to the normal saline solution and with the normal interstitial fluid surrounding the taste cells. The receptor potentials were recorded from taste cells whose resting potentials were in the range −12 to −41 mV. The low value of the resting potential in frog taste cell has been demonstrated by patch-clamping

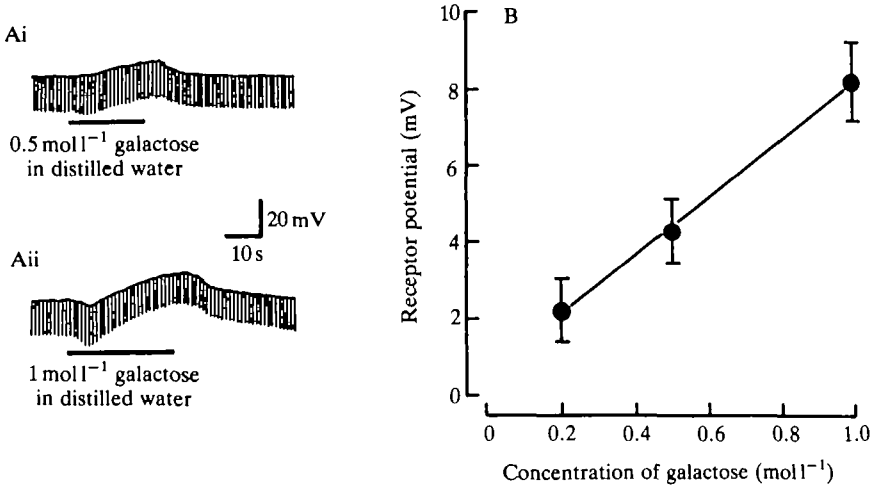

Fig. 1. (A) Receptor potentials in a frog taste cell induced by galactose dissolved in deionized water after adaptation to normal saline. Records i and ii were obtained from the same taste cell. To monitor the change in input resistance, a train of hyperpolarizing electrotonic potentials was superimposed on the membrane potential. The resting potential was −17 mV. (B) Relationship between the concentration of galactose and the amplitude of the receptor potential after adaptation to normal saline. The values are means±S.E.M. obtained from nine cells.

techniques (Avenet and Lindemann, 1988). As the galactose concentration was raised, the magnitude of the depolarization increased in proportion to the concentration (Fig. 1B). The magnitude of the receptor potential induced by 1 mol l^{-1} galactose dissolved in 100 mmol l^{-1} NaCl was nearly the same as that induced by 1 mol l^{-1} galactose dissolved in deionized water. It has been reported that NaCl at concentrations lower than 200 mmol l^{-1} does not inhibit the gustatory neural response to sugar (Funakoshi and Zotterman, 1963; Kumazawa and Kurihara, 1990).

When a taste cell was stimulated by galactose or sucrose dissolved in 100 mmol l^{-1} NaCl, the input resistance of the cell decreased markedly (Fig. 2A). The resting input resistance (57±7 MΩ, mean±S.E., N=4) of taste cells in the unstimulated state was reduced by 34 % by galactose and by 37 % by sucrose (Fig. 2B).

Effect of drugs on sugar-induced depolarization

It has been reported that amiloride inhibits sweet taste receptors in humans (Schiffman *et al.* 1983). However, in this study the amplitude of the depolarizing response in frog taste cells induced by a galactose solution containing 1 mmol l^{-1} amiloride was nearly the same as the control (data not shown). Iwasaki and Sato (1984) have shown that divalent cations, such as Cu^{2+}, inhibit the gustatory neural response to sugar in rats. However, the present study shows that, in frogs, adding

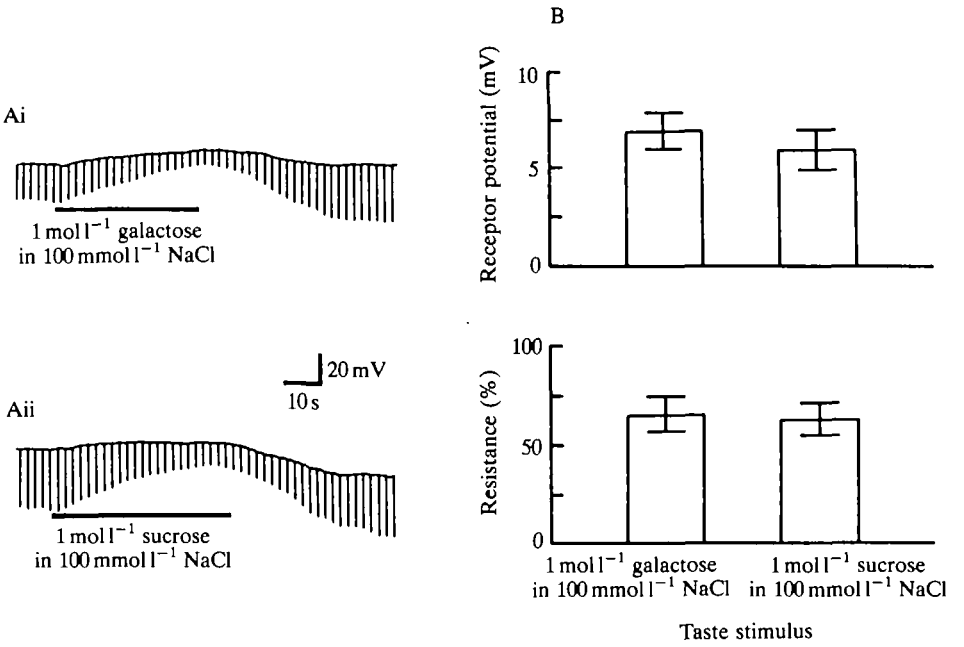

Fig. 2. (A) Receptor potentials induced by 1 mol l^{-1} galactose and 1 mol l^{-1} sucrose dissolved in 100 mmol l^{-1} NaCl after adaptation to normal saline. Records i and ii were obtained from the same taste cell. The resting potential and resting input resistance were −20 mV and 74 MΩ, respectively. (B) The mean amplitudes±S.E.M. of receptor potentials and input resistances in four taste cells induced by 1 mol l^{-1} galactose and 1 mol l^{-1} sucrose dissolved in 100 mmol l^{-1} NaCl. The tongue surface was adapted to normal saline solution. The resistance is expressed as a percentage of the control value in the resting state. The absolute value of the resting input resistance was 57±7 MΩ.

1 mmol l^{-1} $CuCl_2$ or 1 mmol l^{-1} $CdCl_2$ to 1 mol l^{-1} galactose had no effect on the receptor potential. Similarly, superficial addition of 1 mmol l^{-1} SITS, known to block anion channels, had no effect on the receptor potential (data not shown).

Effect of pH in galactose solution

The pH of the galactose solution was adjusted using 5 $mmol^{-1}$ Hepes–NaOH. The magnitude of the receptor potential induced by galactose increased linearly with decreasing pH between 6 and 8 (Fig. 3A). A similar effect on pH was shown for the gustatory neural responses (Fig. 3B). The amplitude of the responses induced by 1 mol l^{-1} galactose stimuli in 10 mmol l^{-1} $NaHCO_3$ (pH 8.4) and 100 mmol l^{-1} $NaHCO_3$ (pH 8.8) were 3.4±1.2 mV (N=7) and 3.1±1.0 mV (N=7), respectively (Fig. 3A). There was no significant difference between these two values. Thus, the depolarizing responses elicited by galactose solutions with a pH greater than 8 were hardly affected by the pH value and the HCO_3^- concentration.

Reversal potential

Fig. 4 shows two typical examples of receptor potentials induced by 1 mol l^{-1}

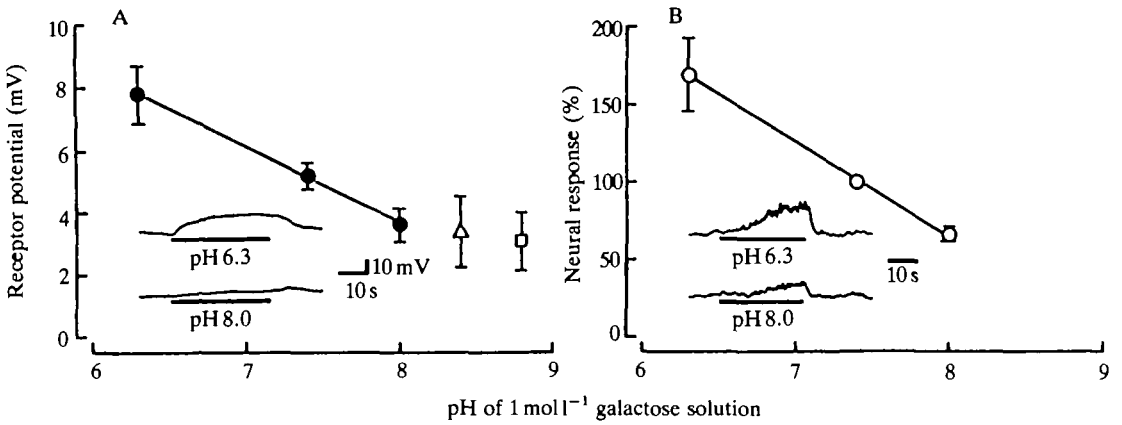

Fig. 3. The effect of varying the pH of galactose solutions on (A) the receptor potential of taste cells and (B) the neural response. 1 mol l^{-1} galactose was dissolved in 100 mmol l^{-1} NaCl containing 5 mmol l^{-1} Hepes–NaOH (●, ○), 10 mmol l^{-1} $NaHCO_3$ (△) or 100 mmol l^{-1} $NaHCO_3$ (□). The points are means obtained from 3–11 cells (A) or nerves (B) and the vertical bars are S.E.M. The insets are examples of gustatory receptor potentials (A) and neural responses (B). Neural responses are expressed as a percentage of the response elicited by a galactose solution at pH 7.2.

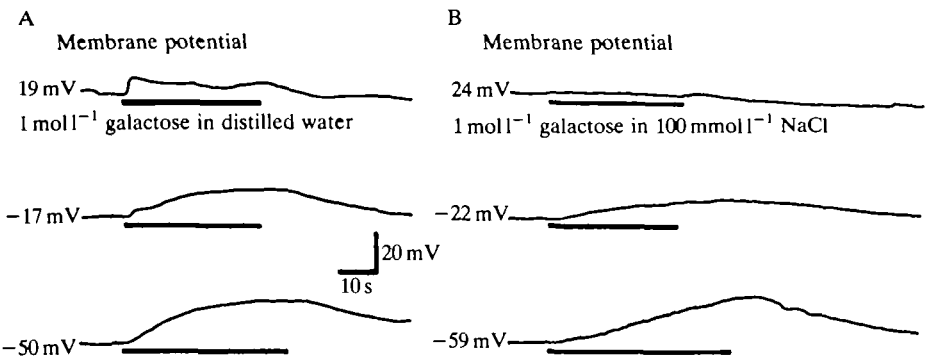

Fig. 4. Receptor potentials induced by 1 mol l^{-1} galactose dissolved in deionized water (A) and in 100 mmol l^{-1} NaCl (B) in two taste cells in which the membrane potentials were varied by current injection. The resting potentials were −17 mV in A and −22 mV in B.

galactose in deionized water (Fig. 4A) and in 100 mmol l^{-1} NaCl (Fig. 4B) in taste cells while the membrane potentials were varied using direct injections of current. When the membrane potentials of both cells were shifted positively, the magnitudes of the responses decreased. Plots of the responses of the two cells in Fig. 4A,B are given in Fig. 5A,B. The mean reversal potential, calculated by extrapolation, was 31±8 mV (mean±S.E., N=8). Since the reversal potentials for

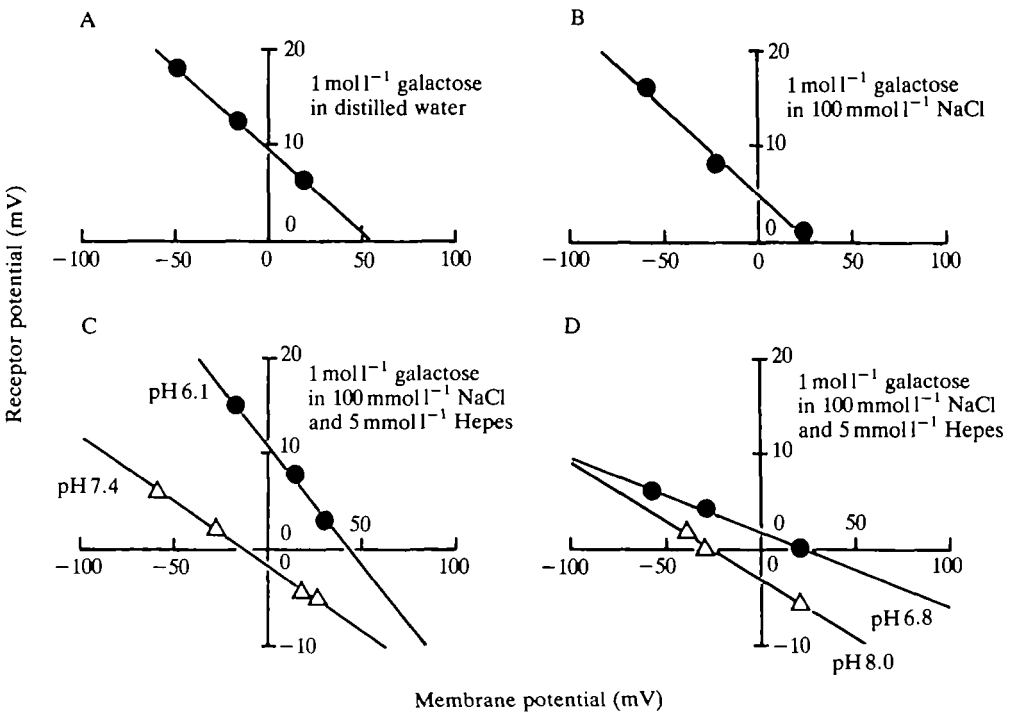

Fig. 5. Four examples of the relationship between the amplitude of receptor potentials induced by 1 mol l^{-1} galactose and the amplitude of membrane potentials shifted by current injected into the taste cell. The resting potentials were −17 (A), −22 (B), −28 (C) and −29 mV (D). In C and D, the pH of the galactose solution was adjusted with 5 mmol l^{-1} Hepes–NaOH. The graphs were obtained from four different taste cells. The lines through the experimental points were fitted using a least-squares linear regression.

the responses to galactose in both water and 100 mmol l^{-1} NaCl were similar, it is unlikely that Na^+ and Cl^- in the stimulus solution affect the galactose-induced receptor potential. The reversal potential shifted positively as H^+ concentration in the sugar solutions was increased (Fig. 5C,D). The mean value of the reversal potential was shifted only 29 mV by a 10-fold change of H^+ concentration in the sugar solution (Fig. 6). This is smaller than the shift predicted from the Nernst potential.

Effects of interstitial ions

Fig. 7 shows the effects of exchanging the normal interstitial fluid for modified saline solutions on the resting potential and galactose-induced depolarization in taste cells. The receptor potentials were obtained by stimulating the taste cells with 1 mol l^{-1} galactose in deionized water while the tongue surface was adapted to the normal saline solution. When interstitial Na^+ was replaced with choline, the amplitude of the resting potential increased to 140 % of the control value, while the amplitude of the receptor potential did not change significantly. Exchange of

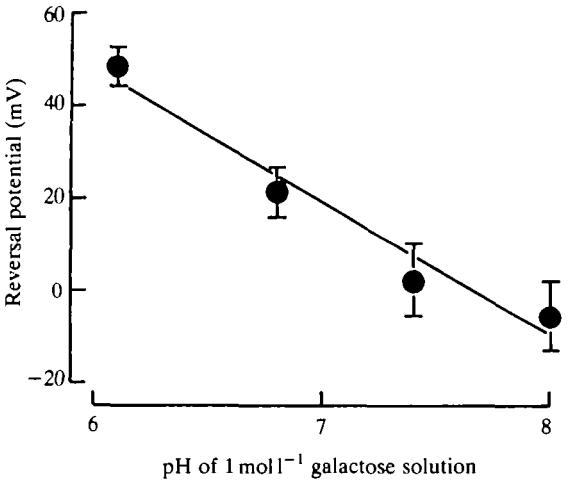

Fig. 6. Relationship between the pH of the 1 mol l^{-1} galactose solution and the reversal potential for the receptor potential. Galactose was dissolved in 100 mmol l^{-1} NaCl and its pH was adjusted using 5 mmol l^{-1} Hepes–NaOH. The points are means obtained from 3–4 cells and the vertical bars are S.E.M. The line was fitted using a least-squares linear regression.

Cl^- with gluconate did not affect the resting potential, but decreased the receptor potential to 2% of the control value. Br^- completely substituted for Cl^- in generating the receptor potential. The use of Ca^{2+}-free and K^+-free interstitial fluids did not affect the receptor potential (data not shown).

Interstitial 0.1 mmol l^{-1} DCCD (an inhibitor of the H^+ pump) irreversibly abolished the receptor potential, whereas interstitial 0.1 mmol l^{-1} NEM (a chemical modifying reagent for protein) reversibly decreased the response to 40% of the control value (Fig. 7). Neither interstitial 0.1 mmol l^{-1} Na_3VO_4 (an inhibitor of ATPase) nor interstitial 0.1 mmol l^{-1} acetazolamide (an inhibitor of carbonic anhydrase) affected the receptor potential (data not shown).

The magnitude of the receptor potential remained similar to that of the control (pH 7.2) when interstitial pH values were raised to pH 8, but when interstitial pH was lowered to 6.3, the receptor potential decreased to 30% of the control value (Fig. 8). However, the resting potential was constant over this range of interstitial pH.

Discussion

Many investigators have shown only poor gustatory responses to sugar stimuli in frogs (Sato, 1976). It has been reported that 1 mol l^{-1} sucrose elicits a depolarization of about 20 mV in mouse taste cells (Tonosaki and Funakoshi, 1984*b*), whereas sucrose induces only a small depolarization of about 6 mV in frog taste cells (Fig. 2B). It is, therefore, concluded that the sensitivity of frog taste cells to sugar is lower than that of mammalian taste cells. As has already been

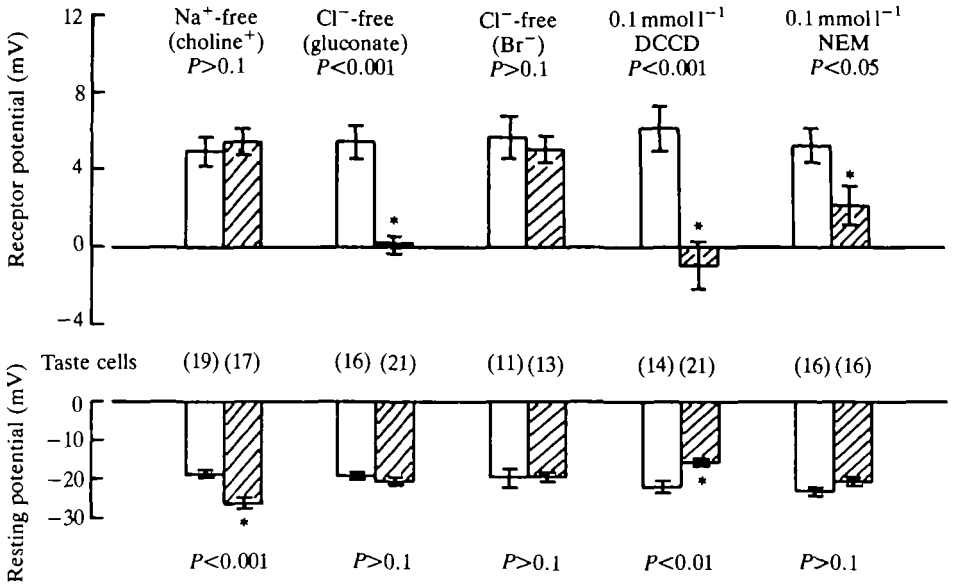

Fig. 7. Effects of changing interstitial ionic compositions and adding drugs on the resting potentials and the receptor potentials induced by 1 mol l^{-1} galactose dissolved in deionized water. The open columns are the control responses and the hatched columns are the test responses. The numerals within parentheses show the number of taste cells sampled and the vertical bars are S.E.M. * denotes a statistically significant difference between the control and test values. DCCD, *N*,*N*′-dicyclohexyl-carbodiimide; NEM, *N*-ethylmaleimide.

demonstrated by Akaike and Sato (1976), 1 mol l^{-1} galactose in 10 mmol l^{-1} NaCl elicited a mean depolarization of 7.4±0.7 mV (mean±S.E., N=23) in the taste cells, whereas 1 mol l^{-1} urea in 10 mmol l^{-1} NaCl induced only a small depolarization of 0.8±1.3 mV (N=7). This indicates that the receptor potential produced in response to the sugar solution was not induced by the osmotic pressure of the solution, but was elicited by the stimulating action of sugar molecules.

The amplitude and reversal potential of the depolarizing receptor potentials in a frog taste cell induced by 1 mol l^{-1} galactose increased linearly with decreasing pH of the galactose solution (Figs 3, 6). These finding suggest that some of the receptor potential is induced by an inflow of H^+ through the taste-receptive membrane. In rat kidney epithelia, an H^+ conductance has been found and this conductance is insensitive to Na^+ and SITS (Burckhardt and Frömter, 1987).

The receptor potential of frog taste cells in response to sugar consisted of both pH-dependent and pH-independent components (Fig. 3A). Since the pH-independent component was affected neither by superficial HCO_3^- concentration (Fig. 3A) nor by a reduction in intracellular HCO_3^- concentration using acetazolamide (an inhibitor of carbonic anhydrase), the pH-independent component may not be generated by the electrogenic Na^+/HCO_3^- cotransport system that has been found in rat kidney (Yoshitomi *et al.* 1985). Part of the driving force for

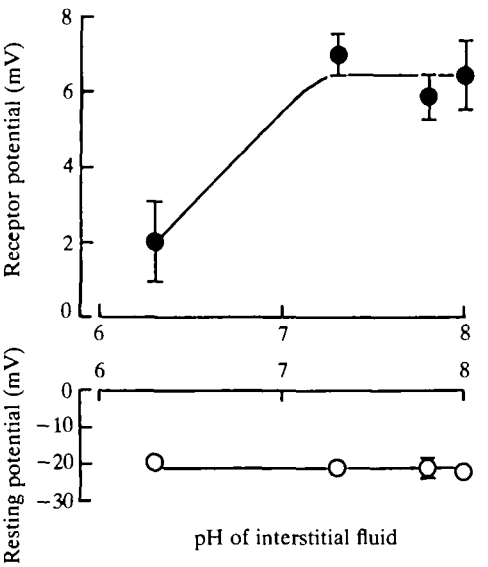

Fig. 8. Effect of interstitial pH on the resting potential and the receptor potential induced by 1 mol l^{-1} galactose dissolved in deionized water. Interstitial pH was adjusted with 5 mmol l^{-1} Hepes–NaOH. The points are means obtained from 9–30 cells and the vertical bars are S.E.M.

the pH-independent component may be attributed to the negative membrane potential. Even though the pH gradient across the apical membrane is not maintained, extracellular H^+ may enter the cell as a result of a negative intracellular potential. Another possible mechanism for the pH-independent component may be an acidic cell surface pH (microclimate pH) formed by H^+ secretion across the apical membrane. Microclimate pH has been demonstrated in rat small intestine (Shimada, 1987). Although this has not been demonstrated on the surface of the frog tongue, an acidic cell surface pH can be maintained by H^+ secretion even if the pH of the superficial fluid is neutral. Further investigation of the frog tongue using a pH electrode is necessary.

A low pH can exert many effects on taste cells. For example, H^+-induced cation currents have been found in chick dorsal root ganglion cells (Konnerth *et al.* 1987). Frog taste cells also have H^+-gated Ca^{2+} channels in the apical membrane, but these channels are activated by acid stimuli with a pH lower than 5 (Miyamoto *et al.* 1988*b*). The observation that the pH range affecting the sugar-induced depolarization was 6–8 suggests that the sugar responses cannot be attributed to H^+-gated Ca^{2+} channels. It has been demonstrated that H^+ concentration in the pH range 6–9 does not affect the binding of sugar to the receptor in bovine taste buds (Dastoli and Price, 1966). It is interesting to note that the action of miraculin, which causes a sweet taste in the presence of acids (Kurihara and Beidler, 1969), may be similar to the action of sugar in an acidic solution in the present study. It is thought that miraculin acts by affecting the binding of sugar to the receptor.

Voltage-sensitive H^+ currents have been demonstrated in snail neurones and these currents are inhibited by heavy metals such as Cu^{2+} (Thomas and Meech, 1982). Cu^{2+} and Cd^{2+} did not, however, affect the sugar-induced receptor potential in frog taste cells. It is important to find a specific blocker for the sugar-activated H^+ channels.

Since the input resistance of the taste cell was markedly reduced by galactose (Fig. 2), and since the reversal potential for the receptor potential did not coincide with the equilibrium potential for K^+ (Fig. 5), it is unlikely that the frog taste cell is depolarized by an inhibition of the resting potassium conductance. This mechanism has been suggested in a mouse taste cell (Tonosaki and Funakoshi, 1984*a*).

The observation that the receptor potential was not inhibited by adding amiloride to the galactose solution indicates that the receptive membrane of the frog taste cell may lack an amiloride-sensitive sodium transport system activated by sugar (Mierson *et al.* 1988; Simon *et al.* 1989). The sucrose-induced depolarization of the mouse taste cell has been reported to be only partly inhibited by amiloride (Tonosaki and Funakoshi, 1989). It may be necessary to perform further experiments using other amiloride analogues to rule out a sodium transport system activated by sugar.

If a sugar stimulus induces H^+ inflow through the taste-receptive membrane, the intracellular pH in the taste cell will decrease gradually during stimulation. However, the generation of stable responses to sugar by a taste cell suggests that the cell may have a regulatory system extruding excess intracellular H^+. The elimination of interstitial Na^+ and K^+ did not affect the receptor potential induced by galactose (Fig. 7), so that any H^+ extrusion from taste cells following sugar stimulation is unrelated to Na^+/H^+ and K^+/H^+ antiport at the basolateral membrane. The observation that the receptor potentials were inhibited by the modified interstitial fluids that lacked Cl^- and contained DCCD, NEM (blockers of H^+-ATPase) and a high concentration of H^+ (Figs 7, 8) suggests that a DCCD/NEM-sensitive, Cl^--dependent H^+ pump exists in the basolateral membrane of frog taste cells. Such a pump has been found in rat kidney (Ait-Mohamed *et al.* 1986; Andersen *et al.* 1985). If the pump is voltage-dependent, shifting the membrane potential of a taste cell positively would increase the driving force for H^+ extrusion and, thereby, raise intracellular pH. Since DCCD and NEM have a broad spectrum of activity, drugs with a more specific action should be explored in further work.

The decrease in the receptor potential for sugar following elimination of interstitial Cl^- cannot be attributed to an inhibition of HCO_3^-/Cl^- exchange at the basolateral membrane because intracellular HCO_3^- has no effect on the sugar response. If there is an exchange system in the basolateral membrane, the inhibition of the exchange will result in a rise in intracellular pH, so that the sugar-induced response will be enhanced by an increased H^+ gradient across the taste-receptive membrane. However, this was not the case in these experiments.

In conclusion, part of the depolarizing receptor potential in a frog taste cell in

response to sugar stimuli appears to be generated by H^+ inflow through the taste-receptive membrane. A Cl^--dependent H^+ pump, which exists in the basolateral membrane of the taste cell, regulates intracellular pH. The voltage-dependence of the H^+ pump couples the pump with the sugar-gated H^+ channels that exist in the receptive membrane.

This work was supported in part by Grants-in-Aid (nos 60480402, 61107001 and 62124031) for Scientific Research from the Ministry of Education, Science and Culture of Japan.

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