Biochimica et Biophysica Acta 939 (1988) 207–213
Elsevier

BBA 73947

Potential difference responses to nutrient K^+, Cl^- and Na^+ changes in secreting and resting states of frog stomach

Manuel Schwartz [a], Gaspar Carrasquer [a], Warren S. Rehm [a] and Mumtaz A. Dinno [b]

[a] *University of Louisville, Departments of Physics and Medicine, Louisville, KY and* [b] *University of Mississippi, Department of Physics, University, MS (U.S.A.)*

(Received 31 August 1987)

Key words: Membrane potential; Nutrient effect; Thiocyanate inhibition; Omeprazole inhibition; Proton secretion; (*R. pipiens* fundus)

The effects of changes in nutrient concentrations of K^+, Cl^- and Na^+ on the transmucosal potential difference (PD) and the resistance were compared for secreting fundus and resting fundus of *Rana pipiens*. Increase of K^+ from 4 to 40 mM, decrease of Cl^- from 81 to 8.1 mM and decrease of Na^+ from 102 to 10 mM gave, 10 min after the change in the secreting fundus, ΔPD values of -28.2, -19.8 and -7.5 mV, respectively. In the resting fundus with SCN^- inhibition, the same changes in nutrient ion concentration gave ΔPD values of -20.1, -17.0 and -10.2 mV, respectively. Changes in Na^+ concentration were considered in a set of experiments of high acid secreting stomachs (4 to 6 μequiv.$\cdot h^{-1} \cdot cm^{-2}$). Here, ΔPD gave for 10-fold decreases in Na^+ concentration in secreting fundus -4.8 mV and in resting fundus with SCN^- inhibition -22.6 mV. Omeprazole inhibition gave results quite similar to those with SCN^- inhibition. From these results in going from secretion to inhibition, it follows that the increment of K^+ conductance if it increased was lower than the increase in NaCl symport conductance since the change in ΔPD for K^+ decreased and that for Na^+ increased. Also HCO_3^- conductance increased with inhibition. After SCN^- inhibition the transmucosal resistance initially increased and later decreased. The decrease can be accounted for by the increase in conductance of the NaCl symport pathway and of the HCO_3^- pathway.

Introduction

The effect on potential difference (PD) due to ion substitution, in which an ion is replaced by a relatively impermeant ion, has been used extensively to determine the relative ionic conductances of membranes. In such studies with K^+, Cl^-, and Na^+ changes in concentration in the nutrient solution of the secreting fundus and antrum of frog stomach, two types of PD responses were found [1–6]. First, a PD response was obtained in which an increase in concentration of a cation such as K^+ or a decrease in an anion such as Cl^- in the nutrient solution gave a decrease in the positivity of the nutrient relative to the secretory side of the gastric mucosa. This response was designated as a normal PD response and was attributed to the existence of a simple conductance pathway [1]. Second, a PD response was obtained under different conditions than that for the normal response. In this case, an increase in concentration of a cation such as K^+ or Na^+ gave an increase in the positivity of the nutrient. This response was designated as an anomalous response and was attributed to the existence of an electrogenic (Na^+ +

Correspondence: M. Schwartz, Department of Physics, University of Louisville, Louisville, KY 40292, U.S.A.

K^+)-ATPase antiport [2–4] and an electrogenic NaCl symport [5,6]. From these studies, simple K^+ and Cl^- conductance pathways [1], an electrogenic ($Na^+ + K^+$)-ATPase pump pathway [2–4] and an electrogenic NaCl symport pathway [5,6] have been postulated as present in the nutrient membrane.

In the present paper, the primary purpose was to determine the effect of inhibition of acid secretion on the transmucosal PD responses due to changes in nutrient K^+, Cl^- and Na^+ concentrations. In these studies we examined the normal response of K^+ and Cl^- and the anomalous response of Na^+ in the secreting and resting (inhibited) states. SCN^+, omeprazole and on occasion cimetidine were used to inhibit acid secretion.

Methods

Experiments were performed on fundi of stomachs of *Rana pipiens* by an in vitro method in which the stomachs were mounted between a pair of cylindrical chambers [7]. All experiments began with standard Cl^- solutions on both sides of the mucosa. The Cl^- nutrient (serosal) solution contained (in mM): Na^+, 102; K^+, 4; Ca^{2+}, 1; Mg^{2+}, 0.8; Cl^-, 81; SO_4^{2-}, 0.8; HCO_3^-, 25; phosphate, 1; and glucose, 10; and the Cl^- standard secretory (mucosal) solution which is hypertonic [8] contained: Na^+, 156; K^+, 4; Cl^-, 160. For increases in K^+ concentration on the nutrient side, K^+ replaced Na^+ and, for decreases in Na^+ concentration on the nutrient side, choline replaced Na^+. In the case of a decrease in Cl^- concentration, SO_4^{2-} replaced Cl^- and sucrose was added to make up any osmotic deficit.

In these studies, the transmucosal resistance, the transmucosal potential difference (PD) and the H^+ secretory rate were measured. Two pairs of electrodes were used, one for sending current across the mucosa and the other for measuring the PD. The PD is considered positive when the nutrient side is positive relative to the secretory side of the stomach. The resistance was determined as the change in PD per unit of applied current. Current (20 μA per 1.3 cm^2 of tissue area) was applied for 1 or 2 s, first in one direction and 2 or 3 s later, in the other direction. The H^+ secretory rate was determined by the pH stat method of Durbin and Heinz [9]. The pH of the secretory solution was generally maintained between 4.7 and 5.0. Moreover, both sides of the gastric mucosa were gassed with 95% O_2/5% CO_2 and 0.1 mM histamine in the nutrient solution was used to stimulate secretion.

For inhibition, 20 mM SCN^- in the secretory solution or 0.5 mM omeprazole in the nutrient solution decreased the H^+ rate to zero. Unlike SCN^- inhibition, omeprazole inhibition is not reversible by simply washing it out. Hersey et al. [10] reported partial reversibility by the use of mercaptoethanol. However, since the reversibility with omeprazole was at best only partially recovered, we generally used SCN^- first and then removed it and added omeprazole. For studies with 1 mM cimetidine, the latter inhibitor was added to a histamine-free nutrient solution.

In previous studies on the nutrient side, due to the existence of a diffusion barrier between the nutrient solution and the nutrient membrane, it took about 10 min (approx. five time constants) for the concentration of the ion at the cell membrane to attain the new concentration in the nutrient solution [1]. Hence the PD and resistance were read at the 10 min mark following the change to the new solution.

Results

PD responses and resistance changes of the secreting and resting fundus due to changes in K^+ concentration in nutrient solutions

Fig. 1 is a representative plot of resistance, PD and H^+ secretory rate versus time for the change from 4 to 40 mM K^+ and back to 4 mM K^+ in secreting and resting fundus. The increase in K^+ gave in secreting fundus a marked decrease in PD and a small decrease in resistance and the return to 4 mM K^+ brought the PD and resistance back towards control levels. The H^+ secretory rate showed a small increase in going from 4 to 40 mM K^+. SCN^- (20 mM) in the secretory solution initially increased the PD and resistance and later decreased the resistance from the maximal level but the resistance remained above the control level. This effect has been previously reported [11,12]. In resting fundus, the increase in K^+ gave a somewhat smaller decrease in PD and about the

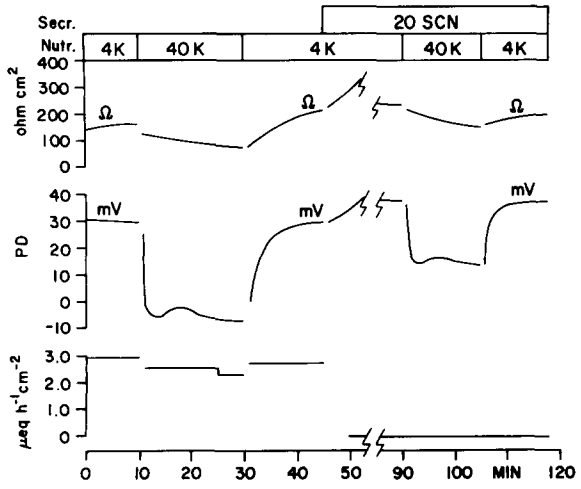

Fig. 1. Effect of changes in K^+ concentration on the nutrient side from 4 to 40 mM K^+ and back to 4 mM K^+ without and with 20 mM SCN^- in the secretory side. Resistance, PD and H^+ secretory rate are plotted vs. time. Concentrations are in mM.

same decrease in resistance compared to secreting fundus. The return to 4 mM K^+ brought the PD and resistance back towards control levels.

In Table I, in going from 4 to 40 mM K^+, ΔPD at 10 min was -28.3 mV for secreting fundus, -20.1 mV for resting fundus with SCN^- inhibition and -20.5 mV for resting fundus with omeprazole inhibition. The difference in ΔPD between secreting fundus and resting fundus with either inhibitor was significant ($P < 0.01$). Upon return to 4 mM K^+, the changes were reversible.

The decrease in resistance in going from 4 to 40 mM K^+ was for secreting fundus -64 ohm $\cdot$ cm^2; for resting fundus with SCN^-, -87 ohm $\cdot$ cm^2; and for resting fundus with omeprazole, -138 ohm $\cdot$ cm^2. Upon return to 4 mM K^+, the resistance increased but now there was no significant difference between the two inhibitors.

PD responses and resistance changes of the secreting and resting fundus due to changes in Cl^- concentration in nutrient solutions

Fig. 2 is a plot of the resistance, PD and H^+ secretory rate versus time for concentration changes from 81 to 8.1 mM Cl^- and back to 81 mM Cl^-. For secreting fundus, the decrease in Cl^- concentration from 81 to 8.1 mM in the nutrient solution caused a sizeable decrease in PD, a small decrease in resistance and a small decrease in H^+ rate. The return to 81 mM Cl^- brought the PD back to control levels, the resistance partly back to control levels and the H^+ rate to the control level. After SCN^- inhibited H^+ secretion, the decrease in Cl^- from 81 to 8.1 mM in the

TABLE I

EFFECT ON PD AND RESISTANCE OF CHANGES IN K^+ CONCENTRATIONS ON THE NUTRIENT SIDE IN THE SECRETING AND RESTING FUNDUS

Values are means $\pm$ S.D. Student's t-test using paired observations was used to determine the level of significance. Columns labeled PD and R refer to the control values of transmucosal potential difference and corresponding resistance and columns labeled ΔPD and ΔR refer to changes in the two parameters following the change to the final concentration of the ion. Number of experiments, 9.

$[K^+]$ (mM)		PD (mV)	ΔPD (mV)	R (ohm $\cdot$ cm^2)	ΔR (ohm $\cdot$ cm^2)
orig. soln.	final soln.				
Secreting state					
4	40	22.9 $\pm$ 6.3	-28.3 ± 7.7 [a]	162 $\pm$ 43	-64 ± 34 [a]
40	4	-6.5 ± 4.9	25.8 $\pm$ 7.7 [a]	83 $\pm$ 23	51 $\pm$ 31 [a]
Resting state: SCN^- inhibition					
4	40	28.3 $\pm$ 7.5	-20.1 ± 5.1 [a]	225 $\pm$ 56	-87 ± 22 [a]
40	4	6.4 $\pm$ 3.7	19.1 $\pm$ 4.9 [a]	119 $\pm$ 25	26 $\pm$ 15 [a]
Resting state: omeprazole inhibition					
4	40	29.8 $\pm$ 8.5	-20.5 ± 6.4 [a]	283 $\pm$ 66	-138 ± 46 [a]
40	4	9.5 $\pm$ 8.7	18.0 $\pm$ 4.0 [a]	127 $\pm$ 32	55 $\pm$ 34 [a]

[a] $P < 0.01$.

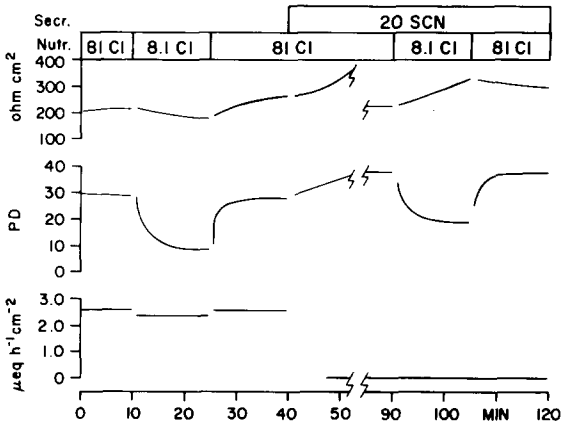

Fig. 2. Effect of changes in Cl^- concentration on the nutrient side from 81 to 8.1 mM Cl^- and back to 81 mM Cl^- without and with 20 mM SCN^- in the secretory side. Resistance, PD and H^+ secretory rate are plotted vs. time. Concentrations are in mM.

nutrient solution produced a somewhat smaller decrease in PD and a small increase in resistance. Upon return to 81 mM Cl^-, the PD returned to control levels and the resistance decreased to a small extent.

In Table II, in going from 81 to 8.1 mM Cl^-, ΔPD at 10 min was -19.8 mV in secreting fundus, -17.0 mV in resting fundus with SCN^- and -17.7 mV in resting fundus with omeprazole. The difference in ΔPD in secreting fundus and in resting fundus with SCN^- was small but significant ($P < 0.05$) while the difference in ΔPD in secreting fundus and resting fundus with omeprazole was not significant. The change in resistance was significant for SCN^- only with an increase of about 50 ohm $\cdot$ cm^2 in going from 81 to 8.1 mM Cl^- and with a decrease of about 25 ohm $\cdot$ cm^2 in the reverse change in Cl^- concentration.

PD responses and resistance changes of the secreting and resting fundus due to changes in Na^+ concentration in nutrient solutions

In these experiments, there is a marked difference in the results after inhibition depending upon whether the H^+ secretory rate is low or moderate on the one hand and high on the other hand. Initially, we consider the first category in which the secretory rate is less than 2.7 μequiv. $\cdot$ h^{-1} $\cdot$ cm^{-2}. For these lower rates, it is shown in Table III that, for the change from 102 to 10 mM Na^+ in the secreting fundus, ΔPD was -7.5 mV; in the resting fundus with SCN^-, ΔPD was -10.2 mV; and in the resting fundus with omeprazole, ΔPD was -14.6 mV. With Na^+, ΔPD is somewhat greater for omeprazole compared to ΔPD for SCN^- ($P < 0.01$). Upon return to 102 mM Na^+, there was again a significant difference between the secreting fundus and the resting fundus with either inhibitor, but here the difference between the inhibitors was not significant.

TABLE II

EFFECT ON PD AND RESISTANCE OF CHANGES IN Cl^- CONCENTRATIONS ON THE NUTRIENT SIDE IN THE SECRETING AND RESTING FUNDUS

See Table I for details. Number of experiments, 11.

[Cl^-] (mM)		PD	ΔPD	R	ΔR
orig. soln.	final soln.	(mV)	(mV)	(ohm $\cdot$ cm^2)	(ohm $\cdot$ cm^2)
Secreting state					
81	8.1	22.9 $\pm$ 7.6	-19.8 ± 5.8 [a]	140 $\pm$ 55	-11 ± 19
8.1	81	2.8 $\pm$ 4.1	17.4 $\pm$ 5.6 [a]	128 $\pm$ 46	1 $\pm$ 27
Resting state: SCN^- inhibition					
81	8.1	30.9 $\pm$ 6.6	-17.0 ± 3.4 [a]	175 $\pm$ 50	52 $\pm$ 47 [a]
8.1	81	14.4 $\pm$ 3.9	15.1 $\pm$ 4.5 [a]	232 $\pm$ 82	-22 ± 29 [b]
Resting state: omeprazole inhibition					
81	8.1	31.0 $\pm$ 7.6	-17.7 ± 4.2 [a]	200 $\pm$ 51	26 $\pm$ 47
8.1	81	13.0 $\pm$ 5.1	15.9 $\pm$ 4.2 [a]	229 $\pm$ 76	-34 ± 78

[a] $P < 0.01$; [b] $P < 0.05$.

TABLE III

EFFECT ON PD AND RESISTANCE OF CHANGES IN Na^+ CONCENTRATIONS ON THE NUTRIENT SIDE IN THE SECRETING AND RESTING FUNDUS FOR LOW AND MODERATE H^+ SECRETORY RATES

See Table I for details. Number of experiments, 11. Average H^+ rate = 1.64 ± 0.57 (S.D.) μequiv. $\cdot h^{-1} \cdot cm^{-2}$, range = 0.57 to 2.68 μequiv. $\cdot h^{-1} \cdot cm^{-2}$.

[Na^+] (mM)		PD (mV)	ΔPD (mV)	R (ohm $\cdot cm^2$)	ΔR (ohm $\cdot cm^2$)
orig. soln.	final soln.				
Secreting state					
102	10	25.5 ± 9.1	−7.5 ± 2.4 [a]	129 ± 64	58 ± 57 [a]
10	102	18.1 ± 7.3	8.9 ± 2.7 [a]	186 ± 110	−39 ± 39 [a]
Resting state: SCN^- inhibition					
102	10	35.3 ± 9.1	−10.2 ± 2.9 [a]	180 ± 75	64 ± 33 [a]
10	102	24.4 ± 7.6	12.3 ± 4.4 [a]	240 ± 96	−62 ± 30 [a]
Resting state: omeprazole inhibition					
102	10	34.1 ± 11.4	−14.6 ± 5.1 [a]	204 ± 109	67 ± 48 [a]
10	102	21.8 ± 11.6	14.4 ± 4.4 [a]	268 ± 143	−70 ± 62 [a]

[a] $P < 0.01$.

In the change from 102 to 10 mM Na^+, the resistance increased in secreting and in resting fundus and, upon return to 102 mM Na^+, the resistance decreased. The changes in resistance were in all cases significant, but never more than 70 ohm $\cdot$ cm^2 in magnitude. Also for these changes in resistance, there were no significant differences between the secreting and resting states.

The PD response after SCN^- inhibition was greater for stomachs that had high secreting rates before inhibition than for those with lower secreting rates before inhibition. We see from Fig. 3 the dramatic decrease in PD after inhibition. We note that in secreting fundus, ΔPD decreased by about 2 mV whereas in the resting fundus ΔPD decreased by about 35 mV. The resistance increased markedly with a decrease in Na^+ concentration and returned to control level with an increase in Na^+ concentration. The resistance changes shown in Fig. 3 are considerably greater in magnitude than those shown in Table IV below.

Table IV shows data on PD and resistance for a change from 102 to 10 mM Na^+ and back to 102 mM Na^+ in the nutrient solution for high H^+ secretory rates. The decrease in Na^+ gave in secreting fundus $\Delta PD = -4.8$ mV compared to $\Delta PD = -22.6$ mV in resting fundus with SCN^-. Upon return to 102 mM Na^+, ΔPD in secreting fundus was 5.8 mV compared to 25.1 mV in resting fundus with SCN^-. For a decrease in Na^+ concentration from 102 to 10 mM Na^+, the increase in resistance with SCN^- was 210 ohm $\cdot$ cm^2 compared to 135 ohm $\cdot$ cm^2 for the secreting state. However, upon return to 102 mM Na^+, the decrease in resistance with SCN^- was not statistically significant.

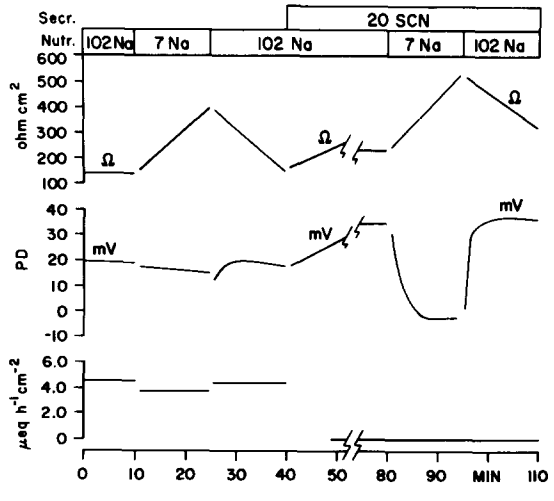

Fig. 3. Effect of changes in Na^+ concentration on the nutrient side from 102 to 10 mM Na^+ and back to 102 mM Na^+ without and with 20 mM SCN^- in the secretory side. Resistance, PD and H^+ secretory rate are plotted vs. time. Concentrations are in mM.

TABLE IV

EFFECT ON PD AND RESISTANCE OF CHANGES IN Na^+ CONCENTRATIONS ON THE NUTRIENT SIDE IN THE SECRETING AND RESTING FUNDUS FOR HIGH H^+ SECRETORY RATES

See Table I for details. Number of experiments, 7. Average H^+ rate = 5.00 ± 0.67 (S.D.) μequiv.·h^{-1}·cm^{-2}, range = 4.05 to 5.82 μequiv.·h^{-1}·cm^{-2}.

[NA^+] (mM)		PD (mV)	ΔPD (mV)	R (ohm·cm^2)	ΔR (ohm·cm^2)
orig. soln.	final soln.				
Secreting state					
102	10	25.9 ± 7.9	−4.8 ± 2.5 [a]	135 ± 29	135 ± 135 [c]
10	102	21.9 ± 7.4	5.8 ± 3.7 [a]	259 ± 117	−114 ± 93 [b]
Resting state					
102	10	38.2 ± 10.7	−22.6 ± 8.3 [a]	226 ± 67	210 ± 196 [c]
10	102	12.2 ± 10.3	25.1 ± 8.6 [a]	411 ± 259	−186 ± 201

[a] $P < 0.01$; [b] $P < 0.02$; [c] $P < 0.05$.

We note further that an experiment was performed with omeprazole as the inhibitor of a high secreting stomach and another experiment with cimetidine. In the experiment with omeprazole, the H^+ secretory rate was, prior to addition of omeprazole, 4.8 μequiv.·h^{-1}·cm^{-2}. The change in PD was −9 mV in secreting fundus compared to −28 mV in resting fundus. In the experiment with cimetidine, the H^+ secretory rate was initially 4.5 μequiv.·h^{-1}·cm^{-2}. The change in PD was −4.2 mV in secreting fundus compared to −26.5 mV in resting founds. Thus these results are comparable to those with SCN^-.

We also note for changes in K^+ and Cl^- concentrations in which cimetidine was used in several experiments, the responses in PD and resistance were comparable to those in which SCN^- and omeprazole were used. Inhibition produced a change in the pattern of PD and resistance responses due to ion concentration changes.

Discussion

In each experiment ΔPD between the results for the secreting state and SCN^- inhibition were compared with ΔPD between the results for the secreting state and omeprazole inhibition. For these two inhibitors, there were no significant differences in the ΔPD values for changes in nutrient K^+ and Cl^- concentrations. For the decrease in nutrient Na^+ concentration, there was a small difference in the ΔPD values which was significant. For the most part there was little difference in the corresponding changes in resistance. In particular, for Na^+ the differences between inhibitors were not significant.

In the present study, we note that during the secreting state ΔPD values for a 10-fold increase in nutrient K^+, a 10-fold decrease in nutrient Cl^- and a 10-fold decrease in nutrient Na^+ were respectively −28, −20 and −7.5 mV. In past work in stomachs with high secretory rates, ΔPD was found to be zero for HCO_3^- [13]. After inhibition with SCN^-, ΔPD values for the same nutrient concentration changes of K^+, Cl^- and Na^+ were, respectively, −20, −17 and −10 mV. Past work gave for a 10-fold decrease in nutrient HCO_3^- in resting fundus ΔPD = −15 mV [14].

In general, we can get from the PD responses some idea of the changes in conductance in going from secretion to inhibition. The K^+ transport is associated with both a simple conductance pathway and a ($Na^+ + K^+$)-ATPase pump pathway [1–4]. Since the resistance of the latter pathway is high, changes in ΔPD generally reflect changes in the K^+ conductance limb of the simple conductance pathway (but there are exceptions [2–4]). The Na^+ transport is associated mainly with the NaCl symport pathway [5,6]. Then for Na^+, changes in PD reflect essentially changes in symport conductance. From the present results for K^+ and Na^+ it follows that, if there is an increase

in the conductance of the K^+ pathway in going from the secreting to the resting state, it is certainly lower than that of the NaCl symport pathway since the change in ΔPD for K^+ decreased and for Na^+ increased with inhibition. Moreover, for Na^+ in the case of high acid secreting stomachs, the change in ΔPD in going from secretion to inhibition is substantial and hence the increase in conductance is marked. For HCO_3^- [14], there is an increase in conductance from about zero to some moderate value is going from the secreting to the resting state. In the case of Cl^- [1,5,6], there are two pathways, a simple conductance pathway and the NaCl symport pathway. Neither pathway has negligible conductance. Since ΔPD changed very little from the secreting to the resting state, it is plausible that inhibition acts oppositely on the two pathways, i.e., an increase in symport conductance and a decrease in the simple Cl^- pathway conductance.

The present work helps to explain previously reported effects of inhibition on the transmucosal resistance, R_t. With SCN^-, an initial rapid increase in R_t is followed by a decrease, but R_t remains above the pre-SCN^- control level. Evidence indicates that the initial increase in R_t is due to an increase in resistance of the secretory membrane and that the subsequent decrease in R_t is due to a decrease in the resistance of the nutrient membrane. These changes are important in determining whether the proton pump is electrogenic or neutral (see Refs. 12, 15 for details). Further evidence that inhibition decreases the resistance of the nutrient membrane is that, with Ba^{2+} in the nutrient fluid, inhibition decreases the resistance of the nutrient membrane of the acid-secreting (tubular) cells, but R_t remains considerably above the control level before Ba^{2+} is added to the secreting fundus [11]. The decrease in R_t has been shown to result from a decrease in resistance of the nutrient membrane of the tubular cells [12,15]. The increases in conductance of the NaCl symport and the HCO_3^- pathway with inhibition can account for the decrease in resistance of the nutrient membrane.

Acknowledgements

This work was supported by NSF Grant No. DMB-8414983. The authors are deeply grateful to Elizabeth Ann Hagan for excellent technical assistance and for preparation of the manuscript.

References

1 Spangler, S.G. and Rehm, W.S. (1968) Biophys. J. 8, 1211–1227.
2 Schwartz, M., Chu, T.C., Carrasquer, G. and Rehm, W.S. (1981) Biochim. Biophys. Acta 649, 253–261.
3 Schwartz, M., Kissel, D.E., Carrasquer, G. and Rehm, W.S. (1983) Biochim. Biophys. Acta 727, 45–55.
4 Schwartz, M., Carrasquer, G. and Rehm, W.S. (1984) Biochim. Biophys. Acta 769, 105–116.
5 Carrasquer, G., Chu, T.C., Rehm, W.S. and Schwartz, M. (1982) Am. J. Physiol. 242 (Gastrointest. Liver Physiol. 5) G620–G627.
6 Carrasquer, G., Kissel, D.E., Rehm, W.S. and Schwartz, M. (1983) Am. J. Physiol. 245 (Gastrointest. Liver Physiol. 8), G554–G561.
7 Rehm, W.S. (1962) Am. J. Physiol. 203, 63–72.
8 Rehm, W.S., Chu, T.C., Schwartz, M. and Carrasquer, G. (1983) Am. J. Physiol. 245 (Gastrointest. Liver Physiol. 8), G143–G156.
9 Durbin, R.P. and Heinz, E. (1959) J. Gen. Physiol. 41, 1035–1047.
10 Hersey, S.J., Sachs, G. and Kasbekar, D.K. (1985) Am. J. Physiol. 248 (Gastrointest, Liver Physiol. 11), G246–G250.
11 Sanders, S.S., Shoemaker, R.L. and Rehm, W.S. (1977) Am. J. Physiol. 234, E298–E307.
12 Rehm, W.S., Carrasquer, G. and Schwartz, M. (1986) Am. J. Physiol. 250 Gastrointest. Liver Physiol. 13), G511–G517.
13 Sanders, S.S., O'Callaghan, J., Butler, C.F. and Rehm, W.S. (1972) Am. J. Physiol. 222, 1348–1354.
14 Schwartz, M., Carrasquer, G. and Rehm, W.S. (1985) Biochim. Biophys. Acta 819, 187–194.
15 Rehm, W.S., Carrasquer, G. and Schwartz, M. (1986) Am. J. Physiol. 250 (Gastrointest. Liver Physiol. 13), G639–G647.