PHYSIOLOGY

Action of Aldosterone on the Lachrymal Gland

THE lachrymal gland in mammals has acinar tissue leading to ducts lined with columnar epithelium showing no regional differentiation. The Na/K ratio of an established secretion shows no alteration in relation to variation in secretory rates, nor is there other evidence of sodium absorption from the acinar secretion associated with exchange for other ions[1]. There are no records of experiments on the effect of mineralocorticoids on the sodium–potassium relation of lachrymal secretion. In view of the fact that sheep were available with graded sodium depletion, and therefore proportional aldosterone secretion rate[2], saliva and tears were collected for sodium and potassium assay.

The sheep were Merino cross-bred. Four (two ewes and two wethers) had a parotid fistula from the start; a third ewe (Gretel) was used for two collections of tears over a fortnight and then a parotid fistula was made. Up to 450 m.equiv. sodium were lost daily from the fistulae and the degrees of sodium depletion were regulated by varying the amount of sodium bicarbonate solution given to drink[2]. Tears were collected by placing a 100-μl. capillary pipette at the lateral canthus of the eye and lachrymation was stimulated by blowing ammonia fumes at the conjunctiva. Precautions were not taken against evaporation because our interest was in the Na/K ratio. Parotid salivary samples were taken just before the tear collections. The sodium and potassium concentrations in each were estimated using a Beckman flame spectrophotometer.

Four animals were otherwise normal. The exception was Maxine, which had had bilateral Goldblatt clamps applied 12 months previously and had, at the time of the experiment, a systolic blood pressure of 150–180 mm mercury.

The results are shown in Table 1. This shows that the range of the Na/K ratio in tears is from 8·8 to 4·5, while that for saliva, which is known to reflect the degree of sodium depletion and aldosterone secretion[2], was from 41 to 0·58; in terms of concentration of potassium, there was variation by a factor of × 1·6 for tears and × 26 for saliva. The variation of lachrymal and salivary Na/K was not regularly concomitant, and it is likely that the variation of the potassium concentration of tears was due to the 'start-up' discharge of potassium at the onset of increased secretory rate which has been described for tears[3], for sweat[4,5] and for salivary glands[6–8].

As an extension of these results, it was decided to test the effect of aldosterone infusion in a sheep when sodium-replete and when sodium-deficient, in which latter instance the sensitivity of the parotid gland to aldosterone is increased[9]. Flora, a bilaterally adrenalectomized ewe, was normally maintained on 25 mg cortisone acetate and

Table 1. LACHRYMAL SODIUM AND POTASSIUM CONCENTRATIONS AND THE PAROTID SALIVARY Na/K OF FIVE SHEEP WITH SODIUM STATUS VARYING FROM REPLETE TO MODERATE TO SEVERE DEFICIENCY

Sheep	Salivary Na/K	Tears Sodium (m.equiv./l.)	Tears Potassium (m.equiv./l.)	Na/K
Gretel	(Before fistula)	162	19	8·5
		153	19	8·0
	41	140	17	8·2
	28	137	24	5·7
	6·1	141	27	5·2
	5·4	140	28	5·0
	1·06	142	20	7·1
Cecil	6·7	133	20	6·7
	4·0	144	23	6·3
	0·87	142	26	5·5
	0·58	127	28	4·5
Arianne	8·1	153	24	6·4
	3·3	168	19	8·8
	1·3	138	28	5·0
Finochio	13·2	146	24	6·1
	1·8	142	27	5·2
Maxine	8·5	138	20	6·9
	3·6	137	28	4·9
	0·85	152	27	5·6

Table 2. FLORA—THE LACHRYMAL SODIUM AND POTASSIUM CONCENTRATIONS AND THE PAROTID SALIVARY Na/K BEFORE INTRAVENOUS INFUSION OF *d*-ALDOSTERONE, AT 20 μg/H AND AFTER 150 MIN OF INFUSION, WHEN THE SHEEP WAS (1) SODIUM-REPLETE AND (2) SODIUM-DEFICIENT

Experiment	Salivary Na/K	Tears Sodium (m.equiv/l.)	Tears Potassium (m.equiv/l.)	Na/K
(1) Sodium-replete				
Before aldosterone	23·6	142	20	7·1
During aldosterone	5·9	132	27	4·9
(2) Sodium-deficient				
Before aldosterone	13·8	138	30	4·6
During aldosterone	1·6	130	23	5·6

5 mg DOCA daily. These hormones were withdrawn 36 h before experiments were performed. Tears and saliva were collected before and 150 min after the start of intravenous infusions of *d*-aldosterone at 20 μg/h.

The results in Table 2 are similar to the findings in sodium depletion (Table 1).

The lachrymal gland, in common with the pancreas and sublingual and palatine glands, does not show regional differentiation of ducts, and presumably does not have a sodium reabsorption and exchange mechanisms[1]. (Yoshimura and Hosokawa[10] state that lachrymal potassium increases proportionally to secretory rate and conclude that the gland actively secretes potassium without exchange for sodium or hydrogen. This behaviour would be different from that of the parotid, submaxillary and sweat glands in which, over a limited range, potassium decreases with increased secretory rate and sodium concentration. The conclusions of these authors should be accepted with caution because the high secretory rates occurred at the onset of increased secretion rates; constant secretory rates at several levels were not produced.) Because it shows trifling change in Na/K as compared with parotid saliva in sodium depletion and aldosterone infusion, and because such variation as does occur is not concomitant with the salivary variation and is explicable on grounds other than mineralocorticoid effect, it is reasonable to regard the gland as insensitive to aldosterone. A similar conclusion about the pancreas can be drawn from the observations that the potassium concentration of pancreatic juice did not rise in humans depleted of sodium by duodenal fistula[11]. In sheep the salivary Na/K of the palatine gland did not change in sodium depletion as did the Na/K of all the other salivary glands tested[12]. The insensitivity of these several glands to a level of aldosterone which has a large effect on the parotid and kidney[13] is consistent with the general proposition of Thaysen[6] that aldosterone may act at the sodium reabsorption and exchange mechanisms, but not at the acinar sodium secretory mechanisms.

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