Effect of Aldosterone on Mammalian Eccrine Sweat Glands

In Eutherian mammals, two main regional and functional types of eccrine (atrichial) sweat glands have been described[1], those on the frictional surfaces of the hairless pad, palm and sole which respond to psychogenic stimuli, and the thermoregulatory glands on the general body surface of man. It is claimed that mammalian (eccrine) sweat glands can be included in those extra-renal structures on which there is a definite action of mineralocorticoids[2,3]. This is demonstrably true of the thermal glands in man which have a specialized function for the conservation of Na[4] but there have been no similar studies on the effect of aldosterone on human palmar glands or those on the foot pads of other mammals.

Investigations have, therefore, been made of the action of exogenous aldosterone on palmar, forearm and cat-pad sweat electrolytes using a ventilated capsule method[5,6]. Secretion rate was measured by infra-red water-vapour analysis and solutes collected by washing from the dry epidermal surface. Sweating was induced on the palmar surface of the finger and flexor surface of the forearm in 6 subjects by intradermal injection of 10 µg acetylcholine in 0.1 ml isotonic saline through an indwelling needle inserted by infiltrating 0.2% procaine solution. D-aldosterone (Aldactone, CIBA) was injected s.c. (10 µg/kg) in a forearm site not used for sweat collection, and simultaneous measurements of palm and forearm sweat [Na] and [K] made before, and at intervals up to 3 days after the injection. Previous experiments on the sweat-gland response to aldosterone[4] had shown it necessary to adopt this long time course.

[Na] was found to be significantly lower and [K] significantly higher in the palm than in forearm sweat ($p < 0.001$), the sum of the 2 cation concentrations in palmar and forearm sweat being almost identical (Table I). Since secretion rates did not vary during the tests (approx. 0.003 µl/gland/min in both regions) our results show that D-aldosterone lowered [Na] but had little effect on [K] in both palmar and forearm sweat (Figure, Table I). In control studies it was found that prior injection of 0.2% procaine did not affect the electrolyte concentrations.

In the cat's pad, sweating was induced by stimulation of the internal plantar nerve using a 10 v stimulus, 0.3 msec pulse duration and a frequency of 1–3 cycles/sec[5]. The cats were anaesthetized with nembutal and as it was not possible to use each animal as its own control, groups of cats were studied at 12, 24 and 30–50 h after injection of aldosterone. Intramuscular injection of 100 µg/kg Aldocorten produced no change in sweat [Na] and [K] in serial samples collected in individual cats up to 10 h, nor in groups of cats studied up to 50 h after injection (Table II). In 5 cats a catheter was passed into a ureter for serial urine collections. Urinary [Na] decreased and [K] increased within 1 h of injecting Aldocorten, with a maximum lowering of the Na/K ratio at 6 h. Further experiments were then made on 5 cats to determine whether increased levels of endogenous aldosterone would affect the composition of the sweat. Acute haemorrhage is known to be a potent stimulus to aldosterone secretion[7,8]. Removal of 15% of the cat's blood volume

[1] J. S. Weiner and K. Hellmann, Biol. Rev. *35*, 141 (1960).
[2] I. E. Bush, Pharmac. Rev. *14*, 317 (1962).
[3] K. J. Collins and J. S. Weiner, Physiol. Rev. *48*, 785 (1968).
[4] K. J. Collins, Clin. Sci. *30*, 207 (1966).
[5] K. G. Foster, J. Physiol. *184*, 106 (1966).
[6] K. G. Foster and J. S. Weiner, J. Physiol. *191*, 1P (1967).
[7] J. O. Davis, C. C. J. Carpenter, C. R. Ayers, J. E. Holman and R. C. Bahn, J. clin. Invest. *40*, 684 (1961).
[8] P. J. Mulrow and W. F. Ganong, Circulation *25*, 213 (1962).

Table I. [Na] and [K] in forearm and palmar sweat stimulated by 10 µg acetylcholine

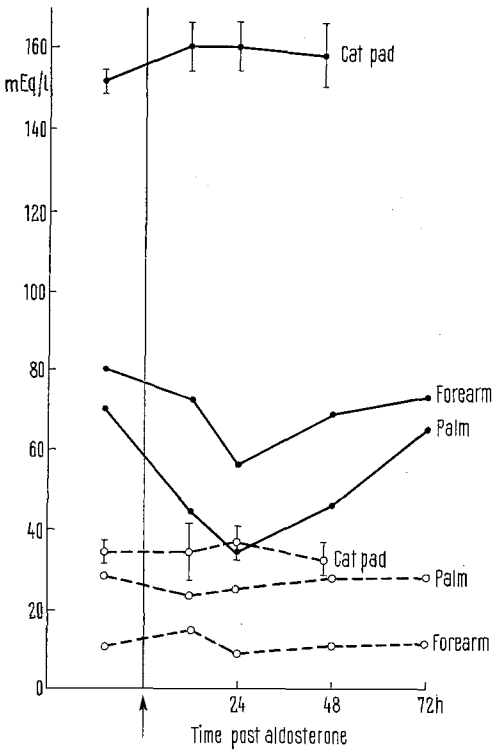
	Before aldosterone	+ 12 h	+ 24 h	+ 72 h
Palmar				
Na (mEq/l)	49.6 ± 8.6	43.0 ± 7.3	35.9 ± 4.2	43.5 ± 8.2
K (mEq/l)	26.9 ± 1.6	23.9 ± 1.4	29.2 ± 4.2	29.4 ± 2.9
Sweat rate (µl/cm²/min)	0.90 ± 0.08	0.83 ± 0.12	0.78 ± 0.08	0.70 ± 0.07
Forearm				
Na (mEq/l)	64.5 ± 7.0	48.3 ± 6.3	49.4 ± 4.5	56.6 ± 6.5
K (mEq/l)	12.0 ± 1.7	11.3 ± 1.4	14.7 ± 3.1	9.8 ± 1.0
Sweat rate (µl/cm²/min)	0.32 ± 0.04	0.31 ± 0.04	0.35 ± 0.05	0.31 ± 0.05

6 subjects ± S.E.

Table II. [Na] and [K] in cat-pad sweat produced by plantar nerve stimulation

	No. of cats	Sweat rate (µl/cm²/min)	Na (mEq/l)	K (mEq/l)
1–3 cycles/sec frequency				
Control	22	0.98 ± 0.12	151 ± 3	34 ± 3
Aldosterone (+ 12 h)	8	1.17 ± 0.25	161 ± 6	34 ± 7
Aldosterone (+ 24 h)	10	0.74 ± 0.07	161 ± 6	36 ± 4
Aldosterone (+ 30 to 50 h)	6	0.74 ± 0.10	158 ± 8	31 ± 4
Hemorrhage (+ 12 to 24 h)	5	0.62 ± 0.09	161 ± 7	28 ± 5
< 1 cycles/sec frequency				
Control	16	0.40 ± 0.04	125 ± 4	54 ± 4
Aldosterone (+ 12 to 24 h)	8	0.48 ± 0.12	122 ± 9	72 ± 8

produced an effect on urine electrolytes similar to that with Aldocorten injections but analysis of sweat collected up to 24 h after haemorrhage failed to demonstrate any significant change in [Na] and [K] (Table II). Another possibility to be considered is that aldosterone may influence sweat electrolyte concentration only at low sweat rates. With stimulation frequencies less than 1 cycle/sec, [Na] was significantly reduced and [K] raised ($p < 0.001$); the sum of these cations being similar to that with higher frequency stimulation. It has previously been reported that the [K] in cat-pad sweat varies inversely with stimulation frequency below 2 cycles/sec[5,9] and the present observations are in agreement with this. Again, aldosterone had no effect on [Na] and [K] in cat-pad sweat produced with low frequency stimulation (Table II).



Effect of D-aldosterone on sweat sodium (●—●) and potassium (○---○) concentrations in cat's pad sweat (± S.E.) produced by nerve stimulation (1–3 cycles/sec) and by acetylcholine injection in the forearm and palm of one subject.

The finding that aldosterone promotes Na-retention in human forearm and palmar glands but not in cat-pad glands may be explained on morphological grounds. The ducts of the glands in the cat-pad are rudimentary and the ductal cells contain few mitochondria as compared with human forearm glands[10]. Other mammalian exocrine glands such as the lachrymal, pancreatic, sublingual and palatine glands with no regional duct differentiation are similarly unresponsive to aldosterone[11]. The cat-pad glands are therefore experimentally useful as a preparation for studying the eccrine secretory process unmodified by ductal reabsorption. In our view, human palmar glands correspond to those of the cat's pad with regard to function (i.e. they respond only to psychogenic stimuli), but they differ morphologically[12] in possessing a duct segment similar to general body eccrine glands.

Zusammenfassung. Nachweis, dass Natriumretention bei der menschlichen Temperaturregulation und in den exokrinen Schweissdrüsen der Handflächen von Aldosteron verursacht wird; dies im Unterschied zu den Drüsen in Katzenpfoten, welche charakteristischerweise vom exokrinen Sekretionsprozess des duktalen Ionenwechsels nicht beeinflusst werden und weder auf Aldosteron noch auf Blutung reagieren.

K. J. COLLINS, K. G. FOSTER and J. L. HUBBARD

M.R.C. Environmental Physiology Unit, London School of Hygiene and Tropical Medicine, Keppel Street, London W.C.1 (England), 29 June 1970.

[9] J. F. G. SLEGERS, in *Cystic Fibrosis*, CIBA Found. Study Gp. No. 32 (Churchill, London 1968), p. 68.
[10] B. L. MUNGER and S. W. BRUSILOW, J. biophys. biochem. Cytol. *11*, 403 (1961).
[11] E. BOTT, J. R. BLAIR-WEST, J. P. COGHLAN, R. A. DENTON and R. D. WRIGHT, Nature *210*, 102 (1966).
[12] S. W. BRUSILOW and B. MUNGER, Proc. Soc. exp. Biol., N.Y. *110*, 317 (1962).

Thermoregulatory Heat Production by Periaortic Brown Adipose Tissue in the Non-Cold-Acclimatized Rat

Since SMITH[1,2] suggested that brown adipose tissue might be an important source of thermoregulatory heat production, this has been shown beyond doubt to be true in the cold-adapted rat[3], the non-cold-adapted rat[4], the warm-adapted rat[5] and for the new-borns and adults of a number of other species[6–12], including the human neonate[13–16]. Changes in brown adipose tissue temperature in the course of changes in thermoregulatory heat production were measured exclusively in the interscapular

[1] R. E. SMITH, Physiologist *4*, 113 (1961).
[2] R. E. SMITH, Fedn Proc. *21*, 221 (1962).
[3] R. E. SMITH and J. C. ROBERTS, Am. J. Physiol. *206*, 143 (1964).
[4] Sz. DONHOFFER, F. SÁRDI and GY. SZEGVÁRI, Nature, Lond. *203*, 765 (1964).
[5] Sz. DONHOFFER and Z. SZELÉNYI, Acta physiol. hung. *32*, 53 (1967).
[6] Sz. DONHOFFER and Z. SZELÉNYI, Acta physiol. hung. *28*, 349 (1965).
[7] D. HULL and M. M. SEGALL, J. Physiol., Lond. *181*, 449 (1965).
[8] T. HEIM and D. HULL, J. Physiol., Lond. *187*, 271 (1966).
[9] K. BRÜCK and B. WÜNNENBERG, Fedn Proc. *25*, 1332 (1966).
[10] T. HEIM and M. KELLERMAYER, Acta physiol. hung. *30*, 107 (1966).
[11] T. HEIM and M. KELLERMAYER, Acta physiol. hung. *31*, 339 (1967).
[12] H. TARKKONEN and H. JULKU, Experientia *24*, 798 (1968).
[13] M. J. R. DAWKINS and J. W. SCOPES, Nature, Lond. *206*, 201 (1965).
[14] W. AHERNE and D. HULL, Lancet *1*, 765 (1965).
[15] Sz. DONHOFFER, T. HEIM and Z. SZELÉNYI, Wien. klin. Wschr. *79*, 464 (1967).
[16] T. HEIM, M. KELLERMAYER and M. DANI, Acta paediat. hung. *9*, 109 (1968).