Table 1. GLUTATHIONE (μ MOL./GM., WET WEIGHT) IN BRAIN OF RATS WITH AND WITHOUT DIETARY PYRIDOXINE

Weeks Deficient	Cortex			Cerebellum			Medulla		
	Control	−B6	Difference	Control	−B6	Difference	Control	−B6	Difference
6	1·88	1·88	0·00	1·76	1·48	0·28	1·00	0·76	0·24
8	1·90	1·62	0·28	1·52	1·32	0·20	1·06	0·80	0·26
9	1·90	1·64	0·26	1·48	1·26	0·22	1·06	0·08	0·26
14	2·10	1·82	0·28	1·54	1·16	0·38	1·04	0·82	0·22

($P < 0·001$) after 6–14 weeks of pyridoxine deficiency. The fall in concentration was similar in all three parts of the brain but the greatest percentage fall (24 per cent) occurred in the medulla.

Hope's finding that cystathionine accumulates in the brain of pyridoxine-deficient rats[6] indicates that the formation of cystine from methionine is impaired. The present results could be explained by the assumption that availability of cystine for glutathione synthesis is limited. It would be of interest to investigate brain glutathione levels of pyridoxine-deficient rats fed on a diet containing no added cystine.

Table 2. MEAN CONCENTRATION OF GLUTATHIONE (μ MOLE./GM., WET WEIGHT) IN BRAIN OF RATS UNDER DIFFERENT DIETARY CONDITIONS

Brains	Mean Concentration of Glutathione (± S.E. of Mean)		6–14 Weeks Pyridoxine Defficient	
	Rat Cake Diet	Control Diet	Mean Fall Glutathione (± S.E. of Mean)	Percentage Fall Glutathione
Cortex	1·96 ± 0·064	1·95 ± 0·055	0·21 ± 0·048	10·8
Cerebellum	1·55 ± 0·052	1·58 ± 0·045	0·27 ± 0·048	17·1
Medulla	1·01 ± 0·012	1·04 ± 0·011	0·25 ± 0·048	24·0

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M. J. ASHWOOD-SMITH
A. D. SMITH

Medical Research Council
Radiobiological Research Unit,
Atomic Energy Research Establishment,
Harwell, Berks.

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Salicylate and Potassium Fluxes of Rat Diaphragm

SODIUM salicylate is known to cause a loss of potassium from rat diaphragms[1]. It is also known to uncouple oxidative phosphorylation[2], and it is suggested[1] that the loss of potassium is due to an interference with the energy supplies for active transport. If so, the inward movement of potassium should be decreased in the presence of salicylate and the outward movement unaltered or decreased as the intracellular concentration of potassium falls.

To examine this hypothesis the potassium fluxes of rat diaphragms have been measured with potassium-42 by conventional techniques[3–5] in the presence of 5m*M* sodium salicylate. In influx estimations diaphragms were divided in half, one half being soaked in potassium-42 Krebs saline containing salicylate, the other in potassium-42 Krebs saline containing no salicylate, both at *p*H 7·4. Left and right halves were allocated alternately to each treatment. After exchanging for 20 or 25 min. in the potassium-42 media the diaphragms were analysed[5] for potassium and potassium-42. Calculations of the fluxes were made, assuming the dimension of the tissue to be as described by Creese[3]. For estimations of efflux the diaphragms were mounted on a frame and soaked in potassium-42 Krebs saline for 1 hr. The mounted tissue was then placed over the window of a Geiger-Müller tube and the diminishing activity of the tissue followed as it was exposed to alternate streams of salicylate containing active and control inactive Krebs saline.

In all experiments salicylate caused a loss of potassium (mean control value 283 m.eq./kgm. dry tissue, mean value of salicylate treated tissues 224 m. eq./kgm. dry tissue, mean difference 59 ± 21 (S.E. of 7 pairs of estimates; $t = 2·895$, $0·05 > P > 0·02$). Contrary to expectation the principal effect of salicylate was consistently to accelerate the influx of potassium-42. The mean efflux rate constant in 6 paired observations was raised from 0·0178 min.$^{-1}$ to 0·0240 min.$^{-1}$ (mean increment 0·0062 ± 0·0016 (S.E.); $t = 4·00$, $P = 0·01$). In estimates of effiux, the salicylate-treated diaphragms always contained less potassium-42 at the end of the soak-in period than their paired controls. However, when allowance was made for the increased rate of concomitant efflux in the salicylate-treated diaphragms, using the estimated efflux rate constants shown above, the estimated potassium influx in salicylate-treated diaphragms was not less than, but often greater than in the control diaphragms. The average influx, uncorrected for effects of diffusion in the extracellular fluid, of salicylate-treated diaphragms (16·3 pmole/cm.^{2}/sec.) and of control diaphragms (15·5 pmole/cm.^{2}/sec.) did not differ significantly ($t = 0·814$; $0·5 < P < 0·4$; 7 pairs of estimates).

It therefore appears that the loss of potassium is due to an action on efflux and that in spite of possible interference with energy supplies, the uptake of potassium is not appreciably altered.

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J. A. HICKLIN

Department of Pharmacology,
London Hospital Medical College,
University of London,
Turner Street,
London, E.1.

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