presumed mercury ions to have also a reversible presynaptic action. In the light of the experiments illustrated in Figure 1, it must now be concluded that the transmitter release previously observed on readmission of Locke solution [12] represented in fact the long-lasting spontaneous release induced by the treatment with mercury and not the restoration of the response to preganglionic stimulation as was initially presumed.

On the basis of the information presented here the possibility cannot be discounted that both pre- and post-ganglionic effects of mercury ions are due to a generalized depolarizing action. However, in experiments in which the postganglionic nerve was left untied we have never observed a transient contraction of the nictitating membrane such as might be expected if the cell bodies of perfused ganglia underwent depolarization. Furthermore it proved possible to elicit contractions of the nictitating membrane by postganglionic stimulation in the presence of 0.1 m*M* mercury ions. CASTILLO and HUFSCHMIDT [13] found 250 μ*M* mercury to be the minimum concentration causing inexcitability of motor nerve fibres. The rapid reversibility of this effect by the application of thiols points to an increase in the threshold voltage rather than to a generalized depolarization as the cause of inexcitability. Nevertheless it is clearly desirable, in view of the toxicological importance of mercury, that their mode of action on synaptic transmission be investigated further by electrophysiological means [14].

Zusammenfassung. In den oberen zervikalen Ganglien der Katze verursacht eine Perfusion mit Locke-Lösung mit 0.1 m*M* Quecksilber Chlorid eine spontane Freisetzung von Acetylcholin. Dieses Phänomen wird vom Standpunkt der bekannten Wirkung anderer Metalionen bei der Freisetzung von Transmittern diskutiert.

K. KOSTIAL and M. LANDEKA

Institute for Medical Research and Occupational Health, 158 Moše Pijade, Y–41000 Zagreb, (Yugoslavia), 2 January 1975.

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Reserpine-Induced Changes in the Uptake and Distribution of Radiolabelled Calcium and Magnesium in the Brain and Pituitary Gland of the Rat

Little is known about the effects of various drugs affecting transmitter release on the in vivo movements of calcium and magnesium in brain tissue. Reserpine is well known to exert an influence on the uptake and release of transmitters and induces a long-lasting reduction of serotonin, norepinephrine and dopamine in the brain and peripheral stores [1, 2]. Amine depletion is thought to be due to changes in the permeability of neuronal membranes [3]. In the present report we present the effects of reserpine on the in vivo uptake of radiolabelled calcium and magnesium by certain areas of the rat brain and pituitary gland. The procedure of earlier investigations with some modifications was followed [4, 5]. Nonfasting white male rats (Sprague-Dawley) weighing 250–300 g and housed in individual cages were used in the study. The animals were injected for 3 days either with saline (0.2 ml, i.m.) for the control group, or with reserpine (2 mg/kg, i.m.) for the test group. The injection schedule on the last experimental day was the following: each animal received saline or reserpine respectively, 4 h prior to injection of the radiolabelled material. Each animal was anaesthetized with pentobarbital and received into the carotid artery a dose of 1 μCi of ^{45}Ca and ^{28}Mg (in 0.2 ml of Ringer's solution buffered to a pH of 7.56 with 4 m*M* HEPES buffer). Due to the short half-life of ^{28}Mg the injection solution was calibrated to be 1 μCi/0.2 ml of ^{45}Ca and ^{28}Mg at the beginning of the experiment. The injection was followed by decapitation in 15 sec. Following decapitation, the brain was quickly dissected free and the following tissues placed into tared scintillation vials and weighed: cortex, hippocampus, cerebellum, thalamus, superior colliculus, medulla and the pituitary gland. 1 ml aliquots of tissue solubilizer (Soluene-350, Packard) were added to each tissue vial and tissues digested within 2 h, after which 10 ml aliquots of a scintillation mixture (Dimilume, Packard) were added to each vial. The β-radiation of ^{28}Mg and ^{45}Ca was measured with a Beckman LS-200

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Table I. Uptake of ^{45}Ca by different brain areas and pituitary gland in rats following intracarotid injection of 1 μCi of ^{45}Ca

Tissue	*N*	Control	Reserpine	Statistical significance (*p*)
Cortex	82	3,311 ± 173	4,806 ± 507	< 0.004
Hippocampus	80	4,091 ± 342	7,907 ± 948	< 0.000
Thalamus	80	3,753 ± 511	6,086 ± 1,036	< 0.038
Superior colliculus	41	6,811 ± 990	9,127 ± 1,228	NS
Cerebellum	79	6,364 ± 528	7,644 ± 554	NS
Medulla	41	10,283 ± 1,848	8,319 ± 1,066	NS
Pituitary gland	40	179,508 ± 39,934	542,381 ± 159,304	< 0.018

The radioactivity is expressed as dpm/g of wet tissues. Values are expressed as means ± S.E.M.

Table II. Uptake of ^{28}Mg by different brain areas and pituitary gland in rats following intracarotid injection of 1 μCi of ^{28}Mg

Tissue	N	Control	Reserpine	Statistical significance (p)
Cortex	54	3,766 ± 649	4,215 ± 569	NS
Hippocampus	53	3,092 ± 837	5,323 ± 872	(< 0.06)
Thalamus	54	4,387 ± 2,200	3,546 ± 932	NS
Superior colliculus	27	3,835 ± 1,028	5,633 ± 1,333	NS
Cerebellum	51	5,059 ± 794	6,688 ± 872	NS
Medulla	27	9,028 ± 2,071	6,766 ± 1,554	NS
Pituitary gland	27	141,453 ± 39,065	239,794 ± 63,857	NS

The radioactivity is expressed as dpm/g of wet tissue. Values are expressed as *means* ± S.E.M.

scintillation counter within 10 h following the start of the experimental procedure.

In order to separate the ^{45}Ca and ^{28}Mg counts, it was necessary to recount the tissue vials after the ^{28}Mg radioactivity had, for all practical purposes, completely decayed. 8 half-lives (7 days) were considered adequate for ^{28}Mg decay (less than 0.4% of the original ^{28}Mg activity remained). Thus, the ^{45}Ca was counted 7 days after the first count. The ^{45}Ca counts (second count) were subtracted from the initial count (^{28}Mg + ^{45}Ca) to obtain the ^{28}Mg content of the samples. Appropriate corrections were made for the decay of both ^{28}Mg and ^{45}Ca. Standards containing equal amounts of ^{45}Ca and ^{28}Mg were used in the calculation of data and decay.

The amount of tissue radioactivity (dpm) of ^{45}Ca and ^{28}Mg for each tissue was divided by the tissue weight and expressed as dpm/g tissue. The data for each tissue was pooled for the animals within the control and test groups and subjected to statistical analysis using a two-tailed t-test.

Table I shows the ^{45}Ca uptake in control and reserpine-tested animals. Statistically significant increases in ^{45}Ca uptake due to reserpine were found in the cortex ($p < 0.004$), hippocampus ($p < 0.001$), thalamus ($p < 0.038$) and pituitary gland ($p < 0.018$). The relation between ^{45}Ca in the cortex and hippocampus was the same as we reported previously[4] for saline controls (cortex-hippocampus, $p < 0.040$) and the reserpine-tested animals (cortex-hippocampus, $p < 0.005$). The higher statistical significance in the reserpine-tested group could be due to the marked increase in hippocampal ^{45}Ca uptake.

Table II shows the ^{28}Mg uptake in the various brain regions of the control and reserpine-tested animals. There was a definite trend, but not statistically significant, in the hippocampus showing an increase in ^{28}Mg uptake due to reserpine. The ratio between the cortex and hippocampus uptake was greater than 1 in the control group (as previously reported[5]), but dropped to less than 1 in the reserpine-treated group.

In contrast to chronic administration, a single injection of reserpine (2 mg/kg, i.m.) did not modify the in vivo uptake of labelled calcium and magnesium.

The increase in the uptake of radiolabelled calcium is in agreement with the reports of a decrease in endogenous levels of calcium in the hippocampus and cortex of rats[6] and guinea-pig brain[7] following reserpine administration. RADOUCO-THOMAS[8] proposed that reserpine may stimulate catecholamine release by removing calcium from some functional site on the presynaptic membrane where its binding was inhibiting the catecholamine release. In our experiments the increased uptake of radiolabelled calcium by the pituitary gland, cortex, hippocampus and thalamus following administration of reserpine could indicate an attempt to replace this calcium. Further ultrastructural and biochemical investigations are needed to elucidate the mechanisms by which neurotransmitter releasing agents modify the blood-brain and blood-pituitary barriers[9].

Résumé. L'administration chronique de réserpine augmente l'incorporation de ^{45}Ca dans le cerveau et la glande pituitaire du rat.

IRENE SABBOT, ELIZABETH ROVNER and A. COSTIN

Brain Research Institute and Department of Anatomy, University of California at Los Angeles, Los Angeles (California 90024, USA), 14 January 1975.

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Effect of Subacute Poisoning by Cyolane[1] on Acetylcholine Esterase and Succinic Dehydrogenase in the Rat

It has been assumed that the toxicity of organophosphorus insecticides was not only due to their potency as anticholine esterase agents[2–6]. Thus the action of these insecticides on metabolic enzyme systems has drawn the attention of many investigators[7–10].

The present investigation was undertaken to obtain information on the effect of Cyolane on rat acetylcholine esterase activity in brain and blood, and liver succinic dehydrogenase activity. Cyolane, is widely used here in Egypt for control of cotton leaf worm, *Spodoptera littoralis*.

Materials and methods. Cyolane, 2-(diethoxy phosphenyl imino)- 1,3-dithiolane, was kindly supplied by American Cyanamide Company. Albino rats of both sexes, weighing 100–120 g and maintained on a stock diet, were used.

Cyolane was given orally in corn oil daily at doses