

Table III. Effect of α -Tocopherol on cerium induced changes in the lipid metabolism of the rat

	Plasma TG (μ g/ml)	Liver TG (mg/g)	Plasma FFA (μ eq/ml)
Group I Control	63.0 \pm 26.0	5.1 \pm 0.5	0.571 \pm 0.067
Group II Cerium	70.0 \pm 48.0 n.s.	15.0 \pm 6.4 $p < 0.01$	0.890 \pm 0.216 $p < 0.01$
Percent of control	111.1	294.1	155.9
Group III α -TP	61.0 \pm 10.0	5.7 \pm 2.5	0.727 \pm 0.254
Group IV α -TP + Ce	67.0 \pm 40.0 n.s.	7.3 \pm 3.7 ^a n.s.	0.691 \pm 0.168 n.s.
Percent of group III	109.8	128.1	95.1

^a Group IV is significantly different from group II, $p < 0.05$.

The levels of Cyt P-450, NADPH-Cyt-C-red and G-6-P DHG in different groups are presented in Table II. The TP treatment abolished almost completely the impairing effect of Ce on the activities of Cyt P-450 and NADPH-Cyt-C-red. Both of these enzymes are involved in the microsomal electron transport chain which function also in the oxidation of fatty acids¹⁸. This function may somehow leave these enzymes more susceptible to the destructive effects of lipoperoxidation, emphasizing thus the protective activity of the antioxidant. Although G-6-P DHG activity is enhanced by Ce treatment as reported earlier¹⁶, the TP treatment tends to decrease this effect slightly ($p < 0.05$). The activation remains still highly

significant after TP treatment ($p < 0.001$). The lack of a more pronounced antioxidant effect may be due to the soluble character of this enzyme.

The changes in lipid metabolism are collected in Table III. Ce had no significant effect on plasma TG level, but in liver the TG concentration increased three-fold after Ce injection. TP alone did not alter the TG level of plasma or liver but it inhibited almost totally the Ce induced accumulation of liver TG. The same normalizing effect of TP could be seen in the increase in the plasma FFA.

Administration of CCl_4 blocks the secretion of hepatic TG into the plasma accompanied by a decrease in plasma level¹⁹. Our study indicates that Ce may act on some different mechanism resembling that of ethanol which leaves the plasma TG at normal or elevated level⁷. The rise in the plasma FFA concentration may be related to the catecholamine depletion of the adrenal glands caused by Ce¹⁶, but why this elevation is inhibited by TP remains unknown²⁰.

Zusammenfassung. Durch Voraussgabe von α -Tocopherol wird bei der Ratte die durch Cerium bewirkte Verminderung der Glucose-6-Phosphatase-Aktivität und des Gehaltes an Cytochrom P-450, NADPH-Cytochrom C-Reduktase und Triglycerid in der Leber verhindert.

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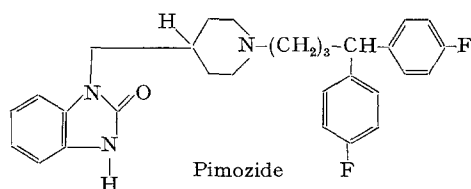
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²⁰ Acknowledgments. The work was supported by a grant from The Finnish Culture-Foundation, Finland.

Diuretic Effects of Intraventricularly Injected Noradrenaline and Dopamine in Rats

Since biogenic amines do not penetrate blood-brain barrier, we have studied behavioural effects of these amines after their application into the lateral ventricle of the rat brain^{1,2}. During these studies we have incidentally observed that noradrenaline (NA) affects the diuresis in rats. The aim of this paper was to study the effects of NA and dopamine (DA) injected intraventricularly on the diuresis in rats.

Methods. Experiments were carried out on female Wistar rats weighing of 190–240 g, from Central Animal Farm of Silesian School of Medicine. Animals were divided into groups and treated as follows. I. Artificial cerebro-spinal fluid (ACSF³ injected intraventricularly (i.vt.)



in volume of 10 μ l. II. DA, 100 μ g i.vt. III. Pimozide, 5 mg/kg i.p., 2 h later 10 μ l of ACSF. IV. Pimozide, 5 mg/kg i.p., 2 h later DA, 100 μ g i.vt. V. NA, 100 μ g i.vt. VI

ACSF i.vt., 10 min later phentolamine, 100 μ g i.vt. VII. Phentolamine, 100 μ g i.vt., 10 min later NA, 100 μ g i.vt. VIII. Normetanephrine, 100 μ g i.vt.

The following substances were used: dopamine hydrochloride (Sigma); pimozide (Janssen); 1-arterenol bitartrate monohydrate (NA) (Sigma); phentolamine hydrochloride (Regitine – Ciba); normetanephrine hydrochloride (Calbiochem).

All substances injected intraventricularly were dissolved in artificial cerebro-spinal fluid described by PALATČ et al.³, and applied in a volume of 10 μ l. Injections were made into the right lateral ventricle of brain according to HERMAN¹. Pimozide was dissolved in physiological saline solution and injected i.p. in a volume of 1 ml/kg of body weight. Immediately after i.vt. injection of drugs, rats were placed for 24 h in metabolic cages and the volume of urine was measured 2, 4 and 24 h after drug injection into the ventricle. During this time the rats had free access to water. The volume of urine was calculated per

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The influence of catecholamines on the diuresis of rats

Group	Treatment	The volume of urine in ml/100 g body wt. after treatment \pm S.E.		
		2 h	4 h	24 h
I	Artificial cerebro-spinal fluid (ACSF) (10 μ g i.vt.)	0.01 \pm 0.00 <i>n</i> = 30	0.08 \pm 0.02 <i>n</i> = 30	1.73 \pm 0.21 <i>n</i> = 27
II	Dopamine (DA) (100 μ g i.vt.)	0.13 \pm 0.05 <i>n</i> = 15 <i>p</i> I/II < 0.025	0.17 \pm 0.06 <i>n</i> = 15	1.52 \pm 0.25 <i>n</i> = 15
III	Pimozide (P) (5 mg/kg i.p.), 2 h later 10 μ l of ACSF i.vt.	0 <i>n</i> = 8	0 <i>n</i> = 8 <i>p</i> I/III < 0.0025	0.81 \pm 0.29 <i>n</i> = 8 <i>p</i> I/III < 0.025
IV	P (5 mg/kg i.p.) 2 h later DA (100 μ g i.vt.)	0.01 \pm 0.01 <i>n</i> = 8 <i>p</i> II/IV < 0.05	0.01 \pm 0.01 <i>n</i> = 8 <i>p</i> II/IV < 0.025	1.24 \pm 0.26 <i>n</i> = 7
V	Noradrenaline (NA) (100 μ g i.vt.)	0.23 \pm 0.08 <i>n</i> = 19 <i>p</i> I/V < 0.01	0.45 \pm 0.08 <i>n</i> = 19 <i>p</i> I/V < 0.0005	1.28 \pm 0.18 <i>n</i> = 15 <i>p</i> I/V < 0.025
VI	ACSF i.vt., 10 min later phentolamine (Ph) (100 μ g i.vt.)	0 <i>n</i> = 8	0.08 \pm 0.05 <i>n</i> = 8	1.64 \pm 0.35 <i>n</i> = 7
VII	Ph (100 μ g i.vt.), 10 min later NA (100 μ g i.vt.)	0 <i>n</i> = 10 <i>p</i> V/II < 0.01	0.02 \pm 0.02 <i>n</i> = 10 <i>p</i> V/VII < 0.0005	1.20 \pm 0.10 <i>n</i> = 10
VIII	Normetanephrine (100 μ g i.vt.)	0.06 \pm 0.05 <i>n</i> = 9	0.13 \pm 0.07 <i>n</i> = 9	1.19 \pm 0.29 <i>n</i> = 9

n = number of rats.

100 g of body weight. Results were elaborated statistically using Students *t*-test.

Results. DA increased the volume of urine significantly for 2 h after i.vt. injection. Pimozide blocked this effect of DA. Pimozide alone inhibited completely the diuresis during the first 4 h of observation, and inhibited significantly diuresis for 24 h after its application. NA increased significantly the volume of urine excreted 2 and 4, but decreased the diuresis in the total 24 h after i.vt. injection as compared with animals injected i.vt. with ACSF. This effect of NA was blocked by phentolamine for 4 h after its application. Phentolamine and normetanephrine had no significant effect on the diuresis, compared with animals injected with ACSF (Table).

Discussion. Our finding that pimozide, which blocks specifically central dopamine receptors⁴, had an anti-diuretic action and inhibited diuresis elicited by centrally

administered DA, suggests that central dopaminergic receptor activation in our experimental model was responsible for diuretic action of DA. We have observed also a diuretic action of centrally administered NA and a blockade of this effect by phentolamine. Recently it has been suggested that phentolamine has central α -adrenergic blocking activity⁵⁻⁷. GUZEK and LEŚNIK⁸ have shown that reserpine, which diminishes the storage of brain NA and 5-hydroxytryptamine, decreases the vasopressin content in the hypothalamus and raises the vasopressin content in the blood. Our results suggest that central catecholaminergic receptors may be involved in diuresis regulation.

Résumé. Chez les rats on a observé l'augmentation de la diurèse provoquée par l'injection de la dopamine et de la noradrénaline dans les ventricules latéraux du cerveau. La pimozide, administré dans le péritoine, inhibe l'action de la dopamine. L'action diurétique de la noradrénaline est enrayée après injection intraventriculaire de phentolamine.

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Spontaneous and Testosterone-Induced Motility of Isolated Guinea-Pig Cauda Epididymis

Actually few investigators have directly observed and/or measured epididymal motility, but the employment of indirect techniques suggested 40 years ago the existence of spontaneous contractions in guinea pig

epididymis¹⁻². Furthermore, in vivo spontaneous motility of rat epididymis, as well as their response to neurotransmitters³ and to sex hormones⁴, have been observed. The first recordings of spontaneous activity of isolated