DOM caused piloerection, behavioural stimulation and pronounced 'head twitching' in high doses (60 mg/kg). Lower doses had a less marked effect. DOM reduced the brain concentration of noradrenaline and elevated that of 5-hydroxytryptamine. Brain dopamine was initially elevated and then reduced by the drug.

Tritiated tyrosine and tryptophan were used to study the effect of DOM on the rate of incorporation of these amino acids into the biogenic amines. It was found that DOM increased the rate of incorporation of tyrosine into noradrenaline but reduced the incorporation of tryptophan into 5-hydroxytryptamine. No effect on the incorporation into dopamine could be detected. DOM also reduced the depletion of brain 5-hydroxytryptamine which followed the administration of p-chlorophenylalamine. These results suggest that DOM may increase the 'turnover' of noradrenaline and reduce that of 5-hydroxytryptamine. The changes in the brain concentration of tyrosine and tryptophan could not account for the effect of DOM on brain amines.

These results are qualitatively similar to those found in previous investigations for other hallucinogenic drugs (Leonard & Tonge, 1969 ; Tonge & Leonard, 1969 ; Tonge & Leonard, 1970).

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Monoamine metabolites in *Octopus vulgaris*

A. V. JUORIO

Departments of Anatomy and Pharmacology, University College London, London WC1E 6BT

Monoamine oxidase (MAO) is present in various tissues of cephalopods (Blaschko & Hawkins, 1952). The fact that dopamine (DM) and 5-hydroxytryptamine (5-HT) are present in neural tissues of cephalopods (Juorio, 1971) suggested that it may be metabolized by MAO to form 3,4-dihydroxyphenylacetic acid (DOPAC) and 5-hydroxyindolylacetic acid (5-HIAA) respectively. Table 1 shows that DOPAC and 5-HIAA are present in the optic lobes of *Octopus vulgaris*. The administration of an inhibitor of MAO (pargyline 100 mg/kg, 3 h) produced a marked fall in the concentration of both DOPAC and 5-HIAA (Table 1). The administration of L-DOPA (200 mg/kg, 3 h), the amino acid precursor of DM, led to a fifty-fold increase in the level of DOPAC.

These results suggest that in *Octopus*, the neural effects of DM and 5-HT are at least partly terminated by MAO.

TABLE 1. *Concentration of DOPAC and 5-HIAA in the optic lobes of* Octopus. *Values are in μg/g of fresh tissue and corrected for recoveries (mean ±S.E.M.).* $^{**}P<0.001$; $^{*}P<0.025$

	Dose mg/kg	Time h	DOPAC μg/g	5-HIAA μg/g
control	—	—	2·82±0·30 (17)	0·97±0·23 (10)
pargyline	100	3	0·29±0·03 (5)**	0·12±0·06 (5)*

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Further evidence for a central hypotensive action of α-methyldopa

L. FINCH* and G. HAEUSLER

Department of Experimental Medicine of F. Hoffmann-La Roche & Co Ltd., Basle, Switzerland

It was proposed that α-methyldopa exerts its hypotensive effect by the formation of α-methylnoradrenaline which was assumed to be a less potent false transmitter in the peripheral sympathetic nervous system (Day & Rand, 1963). However, it has been shown in the cat that adrenergic transmission is not or only slightly affected by α-methyldopa (Haefely, Hürlimann & Thoenen, 1967). More recently, Henning (1969) came to the conclusion that the decarboxylation of α-methyldopa within the central nervous system is essential for the hypotensive effect of the drug.

In conscious genetic hypertensive rats, the blood pressure started to decrease 1 h after the intraperitoneal injection of α-methyldopa (300 mg/kg) and the maximal fall of approximately 60 mmHg was attained 3 h later. This hypotensive effect of α-methyldopa was not prevented by the destruction of peripheral adrenergic nerve endings with 6-hydroxydopamine. Furthermore, in genetic hypertensive rats the responses to stimulation of the entire sympathetic outflow (Gillespie & Muir, 1967) were not influenced by pretreatment with α-methyldopa (300 mg/kg I.P.). Although α-methylnoradrenaline was 10 times less potent than noradrenaline in causing vasoconstriction in isolated perfused renal artery preparations of normotensive rats, the vasoconstrictor responses due to periarterial nerve stimulation were only slightly affected by pretreatment with α-methyldopa (300 mg/kg I.P.). These results rule out the possibility that the mechanism underlying the hypotensive effect of α-methyldopa is solely peripheral in nature.

A central hypotensive mechanism is supported by the finding, that destruction of adrenergic neurons in the brain by intraventricular injection of 6-hydroxydopamine markedly reduces the α-methyldopa-induced hypotension. Furthermore, α-methyldopa (100 mg/kg I.V.) significantly reduced the pressor responses which were produced by stimulation of the rat posterior hypothalamus. The onset of this inhibitory effect corresponded with the hypotensive effect of α-methyldopa. FLA-63, a selective dopamine-β-hydroxylase inhibitor, reversed the effect of α-methyldopa. Very similar results were obtained for the pressor responses produced by stimulation of the mid-brain reticular formation.

In the urethane anaesthetized cat, stimulation of the posterior hypothalamic area produced a rise in blood pressure which was strongly reduced by treatment with