fail to show any significant differences between readings from PCA-treated standards and those in water only. Assays of whole tissue homogenates from rats results in the following activities in μmoles/g/or brain, 6·5 to 7·0; liver, 35–41; and kidney, 2·5–2·7. Brain activities shown here are higher than those reported by Kraml, but are comparable to those reported by Weissbach *et al.*[2] when corrected for probable differences due to temperature and converted from a wet weight to a protein

TABLE 1. RELATIVE FLUORESCENCE OF 4-HYDROXYQUINOLINE IN THE PRESENCE OF PCA AND TCA*

4-HOQ (mμmoles)	Per cent transmission		
	H_2O	PCA	TCA
0	0·5	0·5	0·4
0	0·7	0·5	0·4
10	38·8	41·0	11·6
15	58·3	57·2	18·0
20	77·2	77·1	23·8
25	96·0	97·1	29·9

* Aqueous standards were prepared to a final volume of 5·0 ml and included 2·0 ml of 0·6 M PCA, 10% TCA or water. One ml was added to 3·0 ml of 1 N NaOH and fluorescence was determined at 380 mμ with activation at 315 mμ.

basis. No differences were observed in tissue MAO activities between samples precipitated with PCA and those precipitated with TCA, except for the final fluorescence levels and the somewhat better stability of the readings in the PCA samples.

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Biochemical Pharmacology, Vol. 17, pp. 2013–2015. Pergamon Press. 1968. Printed in Great Britain

Necessity of considering body temperature in drug–cold stress studies of catecholamines

(*Received* 15 *December* 1967; *accepted* 5 *March* 1968)

WORKERS in a number of laboratories, both in the United States and abroad, have recently reported experiments in which they have administered to rodents drugs that inhibit the biosynthesis of catecholamines and have then stressed the animals by placing them at low temperatures for periods of time

varying from 1 to 20 hr.[1–5] Inferences have been drawn concerning the normal effects of stress upon brain biogenic amines on the basis of the different effects of the drugs upon the level of these amines in the stressed and nonstressed animals. The drugs employed predictably cause hypothermia in experimental animals even when the animals are maintained at normal room temperatures for much shorter periods of time than some of the times reported for the cold stress. For instance, 80 mg/kg of *dl*-α-methyltyrosine may lower the body temperature of mice about 3° within 3 hr at a room temperature of 25–27°.[6] Moreover, according to Udenfriend,[7] when rats are given 150–200 mg/kg of α-methyltyrosine intraperitoneally, their body temperature falls rapidly even at normal room temperatures, and if they are placed in a cold room they uniformly die within 6–8 hr.

Yet, in a detailed study of published reports of cold-stress experiments in which drugs that inhibit catecholamine biosynthesis have been administered, I have been unable to find a single one which contains information on the body temperatures of the cold-stressed animals. (In two, 200 mg/kg of α-methyltyrosine, which is largely insoluble between pH 3 and pH 9, was administered i.v. at pH 2 to rats and they were stressed in individual cages in a cold room at 2–3° for 6 hr.[1,2]) This is unfortunate, for the effects of temperature upon chemical processes are well known.[8] When they are ignored, the effects of cold-stress are compounded with the effects of hypothermia and the results, however elegantly and expensively obtained, are not amenable to any straightforward interpretation. The following experiment emphasizes this point.

Male white Swiss mice with body weights in the 34–36 g range were employed. They were given an i.p. injection of pargyline (40 mg/kg in 0·9% saline at pH 7·0) or of vehicle alone, and they were placed individually at 4° for 2·5 hr. They were decapitated, their deep body temperatures were determined and their brains were analyzed for norepinephrine, dopamine or serotonin by methods previously reported.[9] Mice having body temperatures in the 29–31° range were selected for comparison with those that had maintained body temperatures in the range of 35–37°. The latter animals appeared quite normal, while the former were losing coordination and would soon have become ataxic had they not been sacrificed.

Brain amines in the saline-injected mice tended to increase slightly as the mice became hypothermic (Table 1). However, the amine accumulation induced by pargyline was distinctly less in the hypothermic mice than in those that maintained more normal body temperatures.

TABLE 1. HYPOTHERMIA AND BRAIN AMINES*

Treatment	Body temperature		Δ	P<
	35–37°	29–31		
Norepinephrine (ng/g)				
Saline	319 ± 10·5	375 ± 49·1	+56 ± 49·1	n.s.
Pargyline	698 ± 19·5	468 ± 22·8	−230 ± 30·0	0·001
Δ Treatment	+379 ± 22·1	+93 ± 53·8		0·001
Δ from 35–37°	+379 ± 22·1	+149 ± 54·8		0·001
Dopamine (ng/g)				
Saline	484 ± 31·4	586 ± 53·8	+102 ± 62·1	n.s.
Pargyline	1036 ± 65·0	767 ± 88·1	−269 ± 109·5	0·02
Δ Treatment	+552 ± 72·2	+181 ± 103·0		0·005
Δ from 35–37°	+552 ± 72·2	+283 ± 93·5		0·005
Serotonin (ng/g)				
Saline	970 ± 75·3	1347 ± 91·1	+377 ± 118·2	0·002
Pargyline	1606 ± 82·0	1348 ± 127	−258 ± 151·2	n.s.
Δ Treatment	+636 ± 111·3	+1 ± 156·0		0·001
Δ from 35–37°	+636 ± 111·3	+378 ± 147·6		n.s.
No. of mice				
Saline	14	6		
Pargyline	9	6		

* Male white Swiss mice were given a 0·2-ml i.p. injection of pargyline or of saline, and they were then placed in a cold box for 2·5 hr at +4°. Pargyline was 40 mg/kg in 0·9% saline at pH 7·0. Statistical comparisons were by Student's *t*-test. Values are means ±S.E.M.

One interpretation of these results would be that neurotransmission slowed more rapidly than did transmitter biosynthesis in the mice that became hypothermic, but that biosynthesis, nevertheless, was slower in their brains than in the brains of mice which had not become hypothermic.

Hypothermia is not a necessary concomitant of cold-stress when one is working with homeothermic animals; it is a complicating factor. The purpose of this report is to direct attention to this complication and to emphasize that valid inferences cannot be drawn concerning the effect of cold-stress upon neurochemistry unless complications introduced by differences in body temperature are recognized and taken into account.

Acknowledgement—Supported by grants from the U.S. Army Medical Research and Development Command and the Air Force Office of Scientific Research. Dr. E. A. Pritchett, Abbott Laboratories, generously provided pargyline (MO-911).

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Biochemical Pharmacology, Vol. 17, pp. 2015–2017. Pergamon Press. 1968. Printed in Great Britain

Urinary catecholamine excretion after mescaline in man*

(*Received* 12 *December* 1967; *accepted* 23 *February* 1968)

ONE MIGHT expect that increased sympathetic nervous activity associated with administration of psychotomimetic drugs to man would be accompanied by an increased urinary excretion of catecholamines. In a previous study, however, we found that these were little changed after LSD.[1] In view of the general similarities between LSD and mescaline, it was anticipated that the latter drug might also, have little effect on urinary catecholamine excretion. Two experiments were conducted in the present study: one comparing the effects of mescaline against a trial with placebo, the other comparing the effects of mescaline against a nontreatment trial.

* Work supported in part by Grant MH-03030 from the National Institute of Mental Health, United States Public Health Service.