amount of norepinephrine or epinephrine formed in an incubation mixture which contained only the substrate (dopamine or epinine) and an incubation mixture which contained the compound to be tested and the substrate.

The accompanying table shows the effects of test compounds on the conversion of dopamine to norepinephrine and epinine to epinephrine. *p*-Hydroxyamphetamine is a more potent inhibitor than amphetamine. This is in agreement with the previous findings that tyramine is a more potent inhibitor

TABLE 1. INHIBITION OF NOREPINEPHRINE AND EPINEPHRINE SYNTHESIS *in vitro**

Inhibitor	Amount added in μmoles		Amount formed in μmoles		Percent inhibition	
	Substrate†	Inhibitor	Epinephrine from epinine	Norepinephrine from dopamine	Epinephrine from epinine	Norepinephrine from dopamine
None	1·05	none	0·075 ± 0·01	0·45 ± 0·05		
DL-Amphetamine	1·05	2·75	0·037 ± 0·004	0·315 ± 0·03	50	30
DL-Amphetamine	1·05	5·50	0·018 ± 0·002	0·180 ± 0·02	75	60
DL-*p*-Hydroxyamphetamine	1·05	2·37	0·022 ± 0·002	0·225 ± 0·02	70	50
DL-*p*-Hydroxyamphetamine	1·05	4·75	0·015 ± 0·002	0·135 ± 0·01	80	70

* Figures represent averages of 3 experiments in each series.
† Dopamine or epinine.

for dopamine to norepinephrine conversion than is phenylethylamine.[5]

Studies are now under way on the nature of the inhibition of epinephrine and norepinephrine synthesis by amphetamine and *p*-hydroxyamphetamine, as well as on the extent to which epinine is converted to epinephrine *in vivo*.

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Neurochemistry Laboratory,
Department of Psychiatry and Neurology,
New York University College of Medicine

M. GOLDSTEIN
J. F. CONTRERA

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Effect of colchicine on the intestinal xanthine oxidase

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COLCHICINE was shown to inhibit the enzyme xanthine oxidase (XO) of rat liver and to enhance the activity of this enzyme in blood serum.[1]

Since the small intestine of the rat is relatively rich in XO,[2] and since colchicine is eliminated through the bile and the bowel, it seemed worthwhile to investigate the effect of this drug on the intestinal XO.

Male Wistar white rats were used in this study. For intestinal analysis, a portion corresponding roughly to the jejunum of the rat was slit, washed thoroughly in running water and blotted dry. It was then analyzed for XO activity by the same general procedure used for liver.[3] The results are expressed in μl O_2/g dry weight/60 min.

Colchicine was administered by injection (0·1 mg/100 g of body weight) as an alcoholic solution (6·8 per cent) or given *ad libitum* orally by adding colchicine Houdé to the diet (5 mg/100 g of diet). Control animals injected with the alcoholic solution (6·8 per cent) without colchicine were simultaneously examined.

Three animals injected with a single dose of colchicine presented values of 1794 $\pm$ 162 μl O_2/g dry weight 22 hr after the injection. Control rats (5 animals) showed almost the same values as normal non-injected rats (1487 $\pm$ 249 μl O_2/g dry weight/60 min). When three doses (0·1 mg/100 g of body weight) were injected (6 rats) during subsequent days, values of 970 $\pm$ 191 were obtained against 1642 $\pm$ 381 for the controls (7 rats).

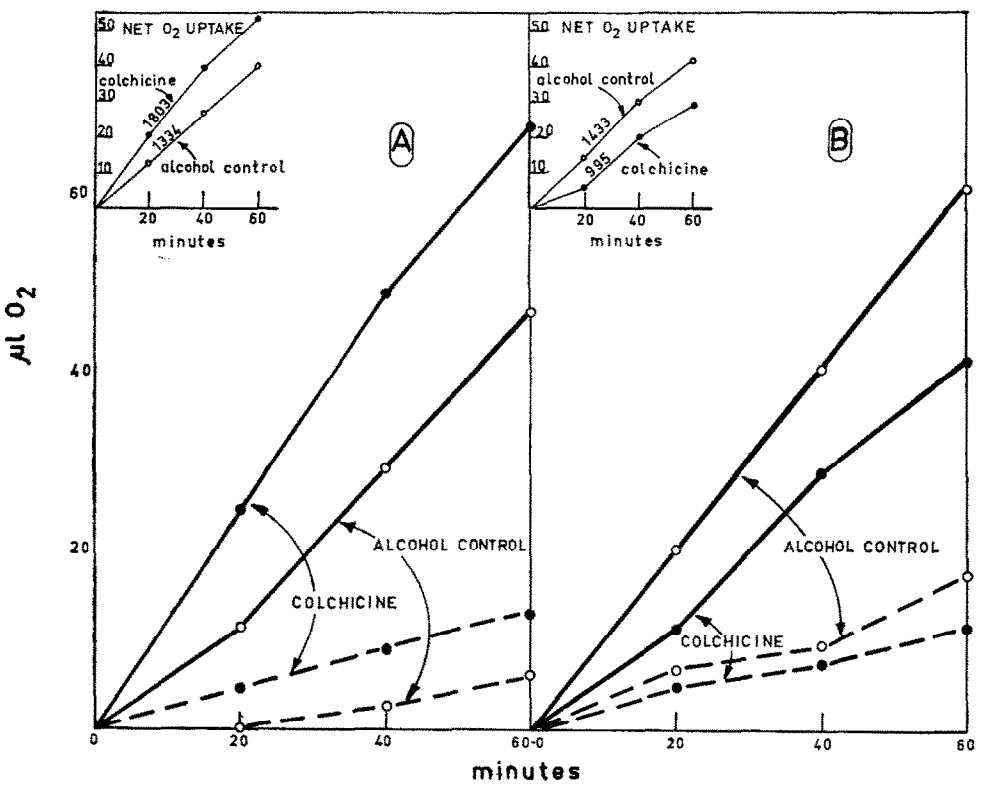

FIG. 1. Effect of colchicine on intestinal xanthine oxidase activity. (A) with a single dose; (B) with three doses. The numbers in graph in the upper part represent values of net O_2 uptake in μl O_2/g dry weight/60 min.

These results show that colchicine, injected as a single dose, slightly increases the activity of the intestinal XO ($t = 2{\cdot}41$, $0{\cdot}02 < P < 0{\cdot}05$). However, the enzyme is inhibited when the drug is administered orally (5 mg/100 g of diet) or when three consecutive doses are injected (0·1 mg/100 g of body weight), $t = 2{\cdot}87$, $0{\cdot}02 < P < 0{\cdot}05$, as shown in Fig. 1.

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Biochemical Laboratory,
Instituto Oswaldo Cruz,
Rio de Janeiro, Brazil

G. G. VILLELA
O. R. AFFONSO
E. MITIDIERI

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