Table 2. INHIBITION OF THE ENDOGENOUS RESPIRATION AFTER ADDITION OF CHLORPROMAZINE AND AMMONIUM CHLORIDE IN BRAIN HOMOGENATES. MEDIUM: KREBS-RINGER SOLUTION (NO SUBSTRATE ADDED). THE INHIBITION IS SIGNIFICANT

No. of experiment	Treatment	$\mu\text{l. O}_2/100 \text{ mgm. wet w./hr.}$		Inhibition (per cent)
		Mean	S.E.	
9	Control	82.72	± 1.98	
6	Chlorpromazine (200 $\mu\text{gm./ml.}$)	62.73	± 3.08	- 24.17
8	NH_4^+ (60 mM)	64.74	± 2.87	- 21.8
9	NH_4^+ (60 mM) + chlorpromazine (200 $\mu\text{gm./ml.}$)	51.87	± 3.9	- 37.3

weight (hr.) and after addition of 200 $\mu\text{gm.}$ chlorpromazine/ml. the oxygen uptake was 143.2 ± 3.5 .

We have reviewed the specificity of the action of chlorpromazine on cerebral tissue. It did not influence the oxygen uptake of rat's hemi-diaphragm slices metabolizing glucose as substrate. In control animals the mean value with standard deviation of 7 exp. was 95.5 ± 13.4 ($\mu\text{l. oxygen}$)/100 mgm. wet weight (hr.) and after 10 $\mu\text{gm.}$ chlorpromazine/ml.— 94.7 ± 10.3 . In 6 exp. after addition of 200 $\mu\text{gm.}$ chlorpromazine/ml. the oxygen uptake showed the value 94.4 ± 11.3 .

It can be assumed that chlorpromazine produces an impairment in energy metabolism of the brain tissue such as depletion of oxygen uptake and inhibition of oxidative phosphorylation⁷. On the other hand chlorpromazine exerts no significant changes in cholinesterase activity of the brain as reported by Decsi⁸ and it does not affect the synthesis of acetylcholine studied in brain homogenates of the rat⁹. These negative results favour the view that the increase of acetylcholine content following chlorpromazine is due to the depression of central activity without direct relation of chlorpromazine to the acetylcholine metabolism of the brain.

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Action of Thyroxine on the Rate of Oxygen Consumption of the Lung-Fish (*Protopterus*)

THE part that endocrines play in the control of metabolic rate of fishes has been investigated by numerous authors. The results concerning the action of thyroxine are contradictory¹⁻⁵. This is mainly due to two essential factors: (1) feeding; (2) activity during measurements⁶. The current investigation was initiated to determine the effect of

thyroxine upon the rate of oxygen consumption of *Protopterus*. These fish do not present the above-mentioned difficulties.

Two young individuals were captured from Tienaba (in the vicinity of Dakar), in February (during the aquatic phase), and were maintained in water until the beginning of the experiments in April. Each fish served as its own control, and thus individual variations were eliminated. The first presented external gills, the second only vestigial ones. The total wet weight of the fish was 27.5 and 46 gm. respectively. (The initial weight of the animals before the experiments was the only one taken into consideration.)

The rate of oxygen consumption was measured by means of Haldane's blood gas volumeter, slightly modified for this purpose. The reaction bulb was replaced by a respiratory chamber, inside which was introduced a small quantity of water, the animal and a small tube containing a carbon dioxide absorbent. This apparatus permitted us to measure the volume of oxygen absorbed by the animal to the nearest 0.01 c.c.

The normal rate of oxygen consumption of each individual was followed during the three days preceding the thyroxine injection and several readings taken. Each fish received a single intra-muscular injection of approximately 0.5 c.c. of thyroxine (Roche), in the dorsal muscles.

Fig. 1. Action of thyroxine on the rate of oxygen consumption of *Protopterus*

In both cases there was a remarkable increase in the rate of oxygen consumption, one day after injection (Fig. 1). The respiratory rate was maximal about the fourth day, then it dropped gradually during the following days in order to reach approximately its initial level.

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