9497 P

Effect of Thyroxine on Oxygen Consumption of the Toadfish.

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It has been commonly assumed that thyroid medication increases the metabolic rate of all vertebrates. For cold-blooded animals, however, the evidence is very scanty. Negative results on frogs and several other poikilotherms have been reported.[1, 2] Positive results on metamorphosing amphibia had been frequently reported but upon critical review were found unconvincing by one of the present authors who obtained negative results.[3] It was therefore thought desirable to investigate the question in a fish where the complications of metamorphosis could be avoided.

A continuous flow apparatus was set up in which oxygen determinations were made by the Winkler technique.[4] By this method repeated determinations can be made on individual animals over a period of days without the slightest disturbance of the animals. With proper precautions for constant temperature (24°C.), elimination of light, etc., it was found that 8-24 hours after introduction into the chambers the oxygen consumption of the fish fell to a fairly constant "standard" level.

The toadfish, *Opsanus tau* (Linnaeus), was used. The animals, weighing about 350 gm., were paired off, starved for at least 3 days before and throughout the course of an experiment. The experimental animals were injected with synthetic thyroxine (disodium salt "Roche", 1 mg. per ml.). Usually 2-3 mg. were injected per day for 4-5 days. The dose was varied, however, a single massive dose of 10 mg. being tried as well as daily 1 mg. doses. The total dose varied from 10 to 23.5 mg. The controls were injected with distilled water brought to pH 11 with KOH. Oxygen determinations were made for a preliminary period of 3 days and for successive similar periods 12 to 18 days after the first injections.

For a statistical evaluation of the results the O_2 consumption of the experimental animal was divided by that of the control for each reading of "standard" metabolism. The values so obtained were

1 Henschel, Hans, and Steuber, Maria, *Klin. Wschr.*, 1930, **2**, 1442.

2 Drexler, E., and Issekutz, B. v. jun., *Naunyn-Schmiedeberg's Archiv.*, 1934, **177**, 435.

3 Etkin, William, *Physiol. Zool.*, 1934, **7**, 129.

4 Keys, A. B., *Biol. Bull.*, 1930, **59**, 187.

statistically analyzed for the preliminary run and for each of the subsequent periods.

Results. Five pairs of animals were carried through successfully. In 4 pairs no significant effect of the thyroxine could be noted. In the fifth a rise of 28% was found at the 9th to 12th day after the first injection. This animal, however, suffered a severe tail lesion at the time of introduction into the respiratory chamber. Since the standard error of the mean of each period varied from 3 to 7% of the mean, it is apparent that a change of even 25% in the standard level would be clearly brought out in the present study. The absence of such a rise in all but one case (where its significance is perhaps doubtful) leads the authors to the opinion that, in the toadfish, thyroxine, even in heavy doses, does not materially increase the oxygen consumption of the normal animal. At the end of the experiment in 3 cases 2 to 2.5 mg. of 2-4 dinitrophenol were injected into the experimental animals. This resulted in a rise of almost 100% in the oxygen consumption, showing that the animals were capable of responding to an effective agent.

9498

Low-Iodine Goiter and the Resistance of the Rat to Thyreotropic Stimulation.

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The rat thyroid is peculiarly resistant to stimulation by the anterior lobe thyreotropic hormone.[1] Anderson and Collip[1] found that rats showing spontaneous goiters of unknown etiology developed a more severe hyperthyroidism after thyreotropic injections than did normal animals, which suggests that the stress of hyperplasia of the thyroid in some way alters this resistance. Severely hyperplastic thyroids can consistently be produced in the rat by means of a diet with inadequate iodine intake.[2] In this work, the modified low-

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1 See review in Anderson, E. M., and Collip, J. B., *J. Physiol.*, 1934, **82**, 11.

2 Levine, H., Remington, R. E., and Von Kolnitz, H., *J. Nutrition*, 1933, **6**, 325.