cluding definitely that this isomer is converted into *l*-indolelactic acid, as the data suggest. A test of *d*-tryptophane alone is contemplated to clear up this point.

Only traces of mycelium were obtained in the acetyl-*l*- and acetyl-*dl*-tryptophane culture flasks. N analyses of the mycelium-free cultures showed that their total N content (275.0 mg.) had not decreased during the period of incubation. Apparently, *Oidium lactis* is unable to hydrolyze either acetyl-*d*- or acetyl-*l*-tryptophane. This is in striking contrast to the utilization of acetyl-*l*-tryptophane by the rat.[4]

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Effect of Thyroparathyroidectomy and Thyroxin on Rate of Atrophy of Skeletal Muscle.

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Previous investigations have indicated that the thyroid secretion exerts an influence on skeletal muscle. Simpson[1] suggested that the lack of development of skeletal muscle in cretin sheep might be due to inactivity rather than a specific action of thyroid secretion on muscle growth. Gudernatsch[2] showed that the feeding of thyroid material caused an accelerated rate of atrophy of the tadpole's tail during metamorphosis. This effect has usually been attributed to the mechanical influences of increased growth changes rather than a specific effect of thyroid substance on autolysis.[3]

This report is concerned with the influence of thyroparathyroidectomy and thyroxin administration on the rate of atrophy of the denervated gastrocnemius muscle of the rat. We have previously reported[4] some effects of denervation on rat's skeletal muscle and described the technique employed. The weight of the denervated muscle has been compared to that of the opposite control muscle in a series of adult animals 7, 14, 21 and 28 days after section of the

[4] Berg, C. P., Rose, W. C., and Marvel, C. S., *J. Biol. Chem.*, 1929, **85**, 207; du Vigneaud, V., Sealock, R. R., and Van Etten, C., *J. Biol. Chem.*, 1932, **98**, 565; Berg, C. P., *J. Biol. Chem.*, 1934, **104**, 373.

[1] Simpson, E. D., *Am. J. Physiol.*, 1927, **80**, 735.

[2] Gudernatsch, J., *Am. J. Anat.*, 1914, **15**, 431.

[3] Bradley, H. C., *Physiol. Rev.*, 1922, **2**, 415.

[4] Hines, H. M., and Knowlton, G. C., *Am. J. Physiol.*, 1933, **104**, 379.

sciatic nerve. A series of 26 animals were subjected to thyroparathyroidectomy a few weeks prior to the time of nerve section. A group of 36 animals were given subcutaneous injections of *dl*-thyroxin. The amounts given totaled approximately 1 mg. per rat per week, and the injections were made at intervals of 3 to 4 days. The initial dose was given 3 to 4 days prior to denervation of the muscle. The effectiveness of the thyroxin administration was determined by frequent measurements of the oxygen consumption of the animals. The amount of thyroxin given was adequate to cause an elevation of the metabolic rate but less than that required to cause an appreciable loss of body weight. In order to measure the rate of atrophy it is essential that the control muscle should neither gain nor loose weight subsequent to the time of denervation of the opposite muscle. A group of animals from the same stock served as controls for the above experiments.

The average values for the loss of weight by the denervated muscle in the 3 groups of animals are given in Table I. Studies made on

TABLE I.

Summary of % of Weight Lost by the Denervated Muscle.			
Days after Denervation	Control Animals	Thyroparathyroidectomized Animals	Animals receiving Thyroxin
7	22.3	21.5	33.1
14	48.3	43.1	58.7
21	61.9	53.8	71.8
28	72.3	66.8	76.6

the water content of the muscles indicate that no significant difference existed between the water content of muscles from animals in the various groups. These results indicate that denervated muscle in animals subjected to thyroparathyroidectomy loses weight at a slower rate than does denervated muscle in control animals selected from the same stock and kept on the same diet. The experiments demonstrate that thyroxin administration accelerates the rate of atrophy of denervated muscle. It is believed that thyroxin exerts this effect by acting as an accelerator to the katabolic processes involved in the disintegration of the cytoplasm of the denervated muscle. These results also indicate that thyroxin is active in skeletal muscle deprived of a nerve supply and offer additional evidence of a direct influence of this hormone on muscle cells.