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ACID-MALTASE ACTIVITY AND HYPOTHYROIDISM

SIR,—Earlier this year we reported the finding of low acid-maltase (α-glucosidase) activity in skeletal muscle of a patient with a myopathy associated with hypothyroidism.[1] Dr. Koster (Dec. 5, p. 1187) now reports that acid-maltase activity was unchanged in various tissues from rats made hypothyroid with methylthiouracil. We have obtained similar results.

Sixteen Wistar rats were given a single intraperitoneal injection of 1 mCi of ^{131}I, after which they were maintained on standard diet for various periods. Ten animals were killed after 2 months and the remaining five were killed after a further 4 months (one animal died during the course of the experiment). Nine normal rats were used as controls. Acid-maltase activity of the gastrocnemius was measured,[2] and, in addition, cathepsin D [3] and ribonuclease [4] were estimated. All three enzymes are lysosomal acid hydrolases. (The histopathology and serum enzymology were also investigated, and the results will be presented elsewhere.)

The accompanying table shows that the enzyme activities were unchanged 2 months and 6 months after thyroid

MUSCLE ENZYME ACTIVITIES IN NORMAL RATS AND IN RATS WITH ABLATED THYROIDS

Enzyme	Normal controls	After 2 mo.	After 6 mo.
Acid-maltase (nmole glucose/g./min.)	86·03 ± 10·61	91·60 ± 6·61	81·62 ± 11·91
Cathepsin D (Δ-O.D./g./hr.)	5·80 ± 0·33	6·18 ± 0·27	6·43 ± 0·61
Ribonuclease (Δ-O.D./g./hr.)	1·97 ± 0·33	1·96 ± 0·16	2·41 ± 0·38

O.D. = Optical density.

ablation by ^{131}I. We therefore conclude that, in our patient with myopathy, the reduced acid-maltase activity was probably unrelated to the hypothyroidism, even though the muscle enzyme level did increase by almost 100% after L-thyroxine therapy. However, it does not necessarily follow that the reduced enzyme level made no contribution to his symptoms. Dr. Koster points out that heterozygotes for Pompe's disease are symptom-free; but it is also true that patients with acid-maltase deficiency restricted to skeletal muscle develop a severe myopathy in the presence of enzyme levels similar to those described in our patient.[2] Admittedly in these cases the myopathy may be due to the storage of glycogen, a feature which was absent in our case. Nevertheless, the fact that the muscle symptoms in this patient were more severe than is usual in myxœdema suggests the possibility that the muscle enzyme deficiency may have played some part in the development of the clinical features. Perhaps a certain degree of acid-maltase deficiency may be compatible with normal function but may render the muscle more vulnerable to other potentially myopathic conditions such as myxœdema.

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