made on frog stomach acid secretion.[2] Increase of sodium chloride content increases the efficacy of the preparation, but magnesium is still needed for maximum performance.

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Department of Applied Pharmacology,
School of Pharmacy,
Hebrew University,
Jerusalem,
Israel

S. DIKSTEIN
F. G. SULMAN

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Influence of thyroid on free fatty acid release from *in vitro* electrically stimulated epididymal fat

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ADIPOSE tissue, both white and brown, is richly innervated, as shown in 1934 by Hausberger, with anatomical evidence.[1] In brown tissue, the innervation is symmetrical; unilateral denervation results in increased glycogen and lipid content and blockade of lipid utilization.[2, 3]

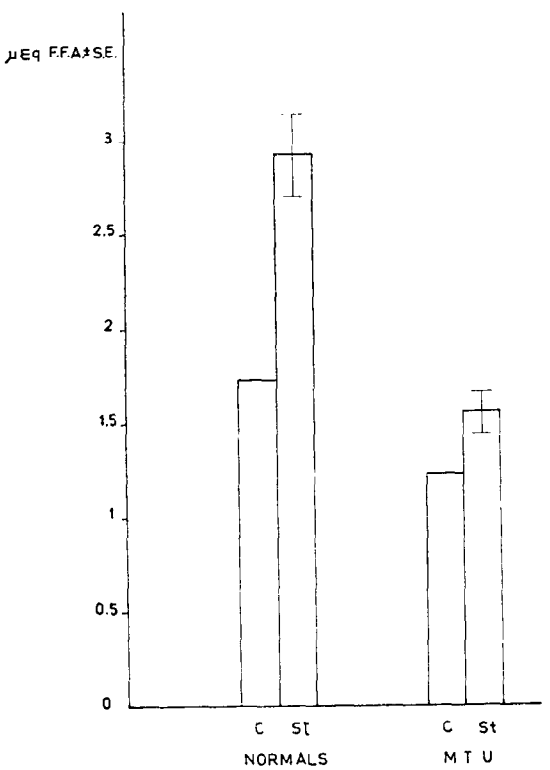

FIG. 1. Effect of methyl thiouracil (MTU) on FFA release from stimulated rat epididymal pad.

More recent investigations have shown that adipose tissue is rich in catecholamines, represented almost exclusively by noradrenaline,[1-6] indicating that adipose tissue innervation is sympathetic in nature.

Debons and Schwartz[7] and Deykin and Vaughan[8] have shown that *in vitro* incubation of adipose tissue, in presence of exogenous noradrenaline, does not induce any important variation in free fatty acid (FFA) release, in comparison with controls incubated without noradrenaline, when the animals have been fed with a propylthiouracil diet. We have observed that pretreatment with methylthiouracil (MTU) and triiodothyronine (T_3) have a profound effect on the *in vitro* direct electric stimulation of the postganglionic sympathetic fibers innervating adipose tissue.

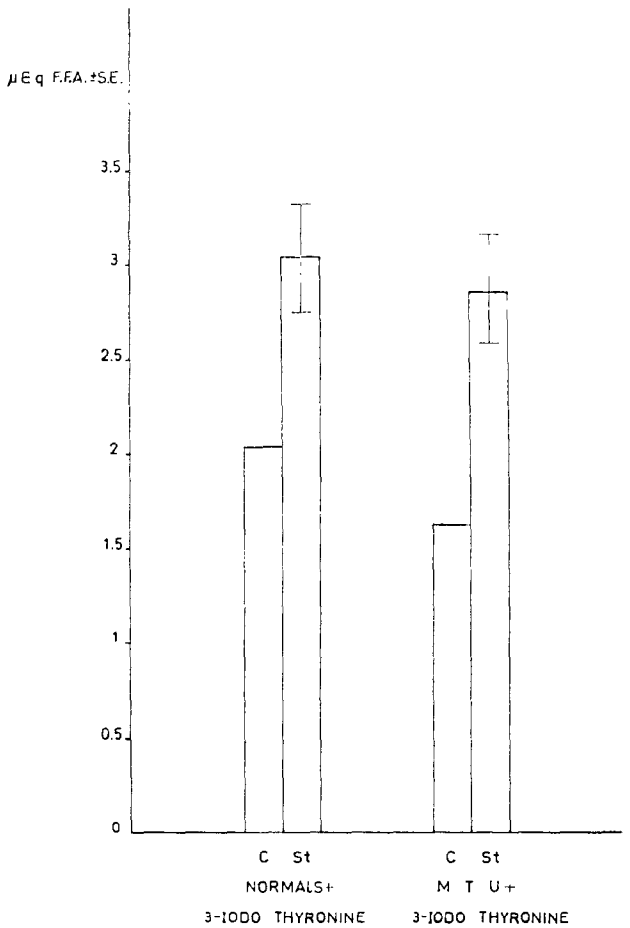

FIG. 2. Effect of 3-iodo thyronine on FFA release from rat epididymal fat after stimulation

Our investigation has been carried out, using male Sprague-Dawley rats weighing 270 ± 20 g. Groups of animals were fed for 25 days with a standard diet containing 0·5% of MTU and groups of normal and MTU-fed rats were treated with sodium triiodothyronine (T_3; Smith, Kline and French Laboratories, Philadelphia, U.S.A.) in three subsequent doses of 1 mg/kg i.p., at 8 hr intervals.

The animals were killed by decapitation and thyroid and epididymal adipose tissue were rapidly removed. In normal animals the weight of thyroid was 15 ± 0·31 mg (mean of 15 rats), in MTU-fed animals 88·0 ± 15 mg (mean of 15 rats). Epididymal fat pads were incubated and prepared for electrical stimulation according to a modification of the technique of Correl,[9] as previously described in detail.[10] The two epididymal fat bodies of the same animal are incubated in comparable conditions

one being stimulated, the other being kept as the control. The neurovascular pedicle was stimulated, using a Palmer stimulator, for 1 hr, frequency 25 impulses/sec, 5 volts, 1·0 msec square wave pulses, duration 1/1000 second. At the end of this incubation FFA in the medium have been titrated according to Dole.[11]

The results, summarized in Fig. 1, clearly show that epididymal fat from MTU-fed rats is insentitive to the direct stimulation of the sympathetic fibers, as far as FFA release is concerned. The basal FFA release is not significantly affected. When T_3 is administered to normal and MTU-fed rats, as shown in Fig. 2, an increased sensitization of adipose tissue to electrical stimulation appears evident. Both normal and MTU-fed rats response with a similar increase of FFA release, after stimulation, when compared with non-stimulated controls. T_3 treatment completely antagonizes the effect of MTU-feeding.

Our data clearly indicate that the metabolic activities of the sympathetic system at the level of adipose tissue are influenced and probably require normal thyroid activity.

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Institute of Pharmacology,
University of Milan,
Italy

F. BERTI
M. M. USARDI

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Evidence for the function of D-glucaric acid as an indicator for drug induced enhanced metabolism through the glucuronic acid pathway in man

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IN ANIMAL experiments the barbiturate induced stimulation of drug metabolism appears to be accompanied by a variety of phenomena:[1]

1. A profound influence of the drug on the smooth surfaced endoplasmatic reticulum in liver cells[2] accompanied by an enhanced microsomal capacity to oxidyze NADPH.[1–3]

2. An increased activity of androgen hydroxylases in rat-liver microsomes.[4] It has been found by Orrenius *et al.*[3, 5] and by Reichert and Remmer[6] that the level of the CO—cytochrome[7] connected with the oxidation of drugs in rat-liver microsomes is increased after pretreatment with phenobarbital. These facts suggest a relationship between the increase of the level of the CO—cytochrome and the increased activity of androgen hydroxylases, since the CO—cytochrome[8] in the adrenal cortex microsomes is connected with the hydroxylation of steroids.

3. A stimulation of the synthesis of free D-glucuronic, L-gulonic and L-ascorbic acid through the glucuronic acid pathway in rat-liver,[9] which is interpretable on the basis of the enhanced activity