(Table I). The nucleotide composition of the RNA fraction of the tumors revealed no gross difference in base ratios (Table II). A comparison of the total ribonucleotide contents of these groups confirms the lower RNA concentration of the 2484L treated tumors. The effect of 2484L treatment on tumor RNA cannot be ascribed to hydration, since it was found that the total nitrogen per gram wet weight of similarly treated and control groups did not differ significantly. These results suggest that 2484L causes an inhibition of synthesis (or an acceleration of degradation) of RNA in C3H mammary carcinoma. Ledoux and Revell(9) have shown that the decrease in growth rate of an ascites tumor (Landschütz) is associated with a parallel decrease in RNA content under normal conditions of growth. Thus the inhibition of growth of this tumor by 2484L may be associated with the decrease in RNA content.

Summary. (1) The RNA content of 2484L treated C3H mammary adinocarcinomas was shown to be lower than that of untreated controls. (2) The nucleotide composition of both treated and control tumors appeared to be similar.

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Anaerobic Glycolysis of Brain Cortex Slices in Hyperthyroid and Control Rats.* (22765)

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The primary site of action of the thyroid hormone in the intermediary metabolic scheme cannot yet be considered as definitely established. An early theory first enunciated by Haffner shortly after the advent of the Warburg metabolic techniques, was that the thyroid hormone primarily affects a step involved in anaerobic glycolysis(1). This was disputed by McEachern who claimed that anaerobic glycolysis was not increased in the livers of rats made hyperthyroid, altho slices of such livers invariably showed increased oxygen consumption when compared with normal control liver-slice values(2). His data confirmed similar earlier work of Dresel(3). Furthermore Anselmino *et al.* had early claimed that spleen slices from hyperthyroid rats failed to show a rise in either oxygen consumption or anaerobic glycolysis in a comparison with normal controls(4). However, in view of a recent statement by Goldstein that anaerobic glycolysis is increased in hyperthyroid rats(5), it was decided to reinvestigate the problem of the relationship of thyroxin to glycolysis beginning with the anaerobic type and brain cortex slices for a reason to be discussed later.

Methods. L-thyroxine is dissolved in a

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TABLE I. Anaerobic Glycolysis of Brain Slices from Control and Hyperthyroid Rats.

Type of animal	No. of animals	No. of values	Anaerobic glycolysis expressed as $Q^{N_2}_{CO_2}$ *
Normal control rats	12	24	18.9 ± 2.34†
Hyperthyroid "	8	24	16.7 ± 3.66

* See text.
† Stand. dev. of mean.

minimal amount of 0.05N NaOH (1 mg in 0.1 cc of 0.05N NaOH). Two mg of such a thyroxine solution are injected subcutaneously into white Wistar rats weighing 100-200 g and this is followed by similar injections of 1 mg daily for 2-4 days, the thyroxine solution being made up fresh daily. The treated rats and untreated litter mates were fed *ad lib* with Purina rat checkers and water. All rats were decapitated for the experimental runs and cerebral cortex slices prepared from each hemisphere manually with a double-edged razor blade held by a Kelly Clamp. One or two slices, less than 0.5 mm thick, were placed into Warburg flasks each containing 3 cc of Krebs-Ringer-Bicarbonate-0.1% glucose solution(6). The vessels were gassed for 5 minutes with a 5% CO_2-95% N_2 gas mixture and placed in a Warburg bath at 37°C. After an 8-minute temperature equilibration period, CO_2 release from the bicarbonate buffer due to anaerobic glycolysis was measured every 15 minutes for 1 hour.

Results. The results are expressed in Table I as cmm CO_2 released per mg dry weight of brain cortex per hour ($Q^{N_2}_{CO_2}$), the slices having been dried for 24 hours at 100°C.

Discussion. Since anaerobic glycolysis in the liver is extremely low and possibly even rudimentary, the fact that it does not increase in liver slices(2-4) from hyperthyroid animals might be considered of doubtful significance. Brain cortex slices, like spleen, however, could be of value as a test for a primary thyroid hormone effect on glycolysis since they show no rise in oxygen consumption in the hyperthyroid state, but do possess a high rate of anaerobic glycolysis normally. Thus a rise in anaerobic glycolysis in hyperthyroid brain slices could not be considered as secondary to a rise in O_2 consumption, since the latter does not occur(7). The fact that such brain slices from hyperthyroid rats do *not* exhibit a higher rate of anaerobic glycolysis than similar slices from normal rats corroborates similar findings with spleen slices reported by Anselmino *et al.*(4). These results combined with those of Dresel(3) and McEachern(4) make the hypothesis of a primary effect of the thyroid hormone on anaerobic glycolysis very unlikely, considering especially that positive, physiological effects on the brain in different thyroid states have been reported(8) and are known clinically. The possibility must still be considered, however, that all the positive brain findings are secondary and due to influence of the thyroid gland upon other glands of internal secretion. If such were the case, then it is possible that the thyroid hormone has no direct effect on any phase of brain metabolism.

Conclusion. Anaerobic glycolysis is *not* increased in brain slices from rats injected with 4-8 mg of L-thyroxine, when compared to similar values from normal control rats.

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