menadione-treated rats show a slower initial rate of disappearance of DIT from the liver; this effect of menadione very likely results from inhibition of the deiodinating enzyme; however, direct proof for this mechanism is still lacking and a toxic effect of menadione on the liver cannot be excluded.

Our results show a relationship between the rate of DIT deiodination and the effect of this amino acid on the catecholamine tissue levels. When the rate of disappearance of the injected DIT is decreased by menadione, a marked fall in tissue NA levels occurs; this result suggests that the concentration of DIT is high enough to compete with the normal substrate of tyrosine hydroxylase and to cause enzyme inhibition.

The question arises whether MIT and DIT, the physiological precursors of thyroxine, could play a physiological role in the regulation of catecholamine biosynthesis. This appears to be unlikely because these iodinated amino acids, synthesized in the thyroid gland, are not usually released into the circulation. However, in a few pathological conditions, MIT and DIT can appear in the peripheral blood. A defect in deiodination of MIT and DIT has been reported in a few cases of familial cretinism[11], in which a continued leakage of these amino acids from the thyroid gland occurs. Identification of MIT and DIT in the serum from patients with thyroiditis or thyroid carcinoma has also been described[12]. Studies are now in progress in our laboratory to see whether such patients could show a diminution of their catecholamine production rate[13].

Résumé. La diiodotyrosine est un puissant inhibiteur in vitro de l'enzyme limitant la synthèse des catécholamines, la tyrosine hydroxylase. Or, administrée in vivo à des rats, la diiodotyrosine n'inhibe que très peu la production endogène de catécholamines. Cependant, en ralentissant la rapide désioduration de la diiodotyrosine injectée par le ménadione, on observe une inhibition marquée de la synthèse des catécholamines in vivo.

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The Effect of X-Irradiation on the Citrate Content in Mouse Liver

Changes in the citrate content in liver during different physiological states are of interest for several reasons. It is known that citrate inhibits hepatic phosphofructokinase[1-4], and, as this enzyme may be regulatory for glycolysis, an elevated or decreased citrate content might play an important role in the regulation of this enzyme. On the other hand, citrate is an activator for acetyl-CoA carboxylase, a regulatory enzyme in fatty acid synthesis[5], and, moreover, citrate is considered to be the source of extramitochondrial acetyl-CoA for fatty acid synthesis via the citrate cleavage enzyme reaction[6].

Radiation effects of citrate metabolism in mammals are reported only by DuBois et al.[7], who found a decreased citrate content in fluoracetate treated rats after 800 R. Data on the effects of irradiation on citrate metabolism in mice are lacking.

It was therefore considered interesting to measure the content of citrate in mouse liver after whole-body X-irradiation with 690 R (LD 80/30 days).

Materials and methods. Male white mice of the institute were used. Water and standard food (Altromin) were provided ad libitum, 'starved mice' were fasted 24 h before killing, water was given ad libitum.

Mice were irradiated with 690 R (148 R/min), at 150 KV, 20 mA, filtered with 0.43 mm copper, at a target distance of 30 cm. The dose corresponds to a LD 80/30 days. The livers of 8 individual mice were removed by the freeze-stop technique under light ether anaesthesia. Each liver was placed in a mortar and ground after the addition of a fourfold volume of 5% trichloroacetic acid (TCA). After homogenization in a Potter-homogenizer for 2 min, the homogenate was centrifuged at 10,000 *g* and the supernatant kept.

From an aliquot of the supernatant corresponding to 0.8 g liver, the TCA was extracted 3 times with 7 ml ether each time, and the aqueous layer was brought to dryness in vacuo. In the residue the citrate content was estimated by the method of Spencer and Lowenstein[8].

Each point in the Figure represents the mean value of 8 livers, with standard deviation of the mean.

Results. The citrate content in the liver of normally fed mice was 0.333 ± 0.011 μmoles/g wet liver tissue (48 mice), and in 24 h starved mice was 0.259 μmoles/g wet wt. (48 mice). These amounts of citrate are of the same order as in rats, although the data reported on citrate contents in the livers of fed and starved rats are conflicting[8,9].

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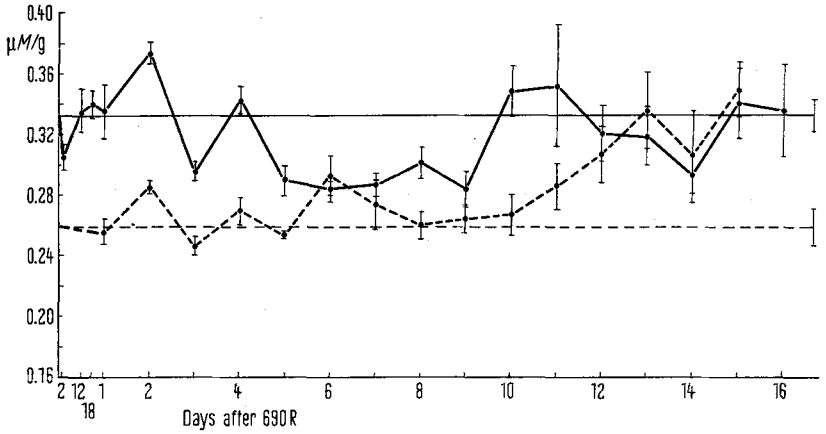

Citrate in mouse liver after 690 R. ·——· fed mice, ·– – – – · mice fasted for 24 h before killing. 8 livers for each point, standard deviations.

After whole body X-irradiation with 690 R, the content was decreased somewhat on certain days after irradiation in fed mice (see Figure). However, this change depends to a high degree on the irregular food intake after irradiation. To single out the effect of irradiation from the influence of irregular food intake, in a second series the citrate content was measured in the liver of mice fasted for 24 h before being killed. At the time of killing, all mice were therefore in the same nutritional state.

Under these circumstances, the content of citrate was almost unchanged for 11 days after irradiation, followed by an increase on 12th–15th day up to the value of normally fed mice (dotted line in the Figure).

Discussion. The results show that the citrate content is scarcely influenced in mouse liver by the irradiation and citrate synthesis via the citratesynthetase reaction seems to be normal even after lethal X-ray doses. Changes in fed mice depends mainly on starvation effects following irradiation, since the citrate content remains almost unchanged in fasted mice over a period of 11 days after the exposure (see Figure). Therefore, neither the inhibition of phosphofructokinase nor the activation of acetyl-CoA-carboxylase by citrate is considered to be altered during this period after irradiation. It is improbable that on the first to eleventh days after irradiation any irradiation induced modifications of glycolysis or fatty acid synthesis are effected by citrate. However, the elevated citrate level in starved mice on the 12th–15th day might influence somewhat the activities of these 2 enzymes in vivo. It is difficult to decide whether the extramitochondrial acetyl-CoA content is influenced by the change in the level of citrate, because acetyl-CoA can come from anaerobic glycolysis as well as from degradation of fatty acids. Hence, the changes in acetyl-CoA content after irradiation[10] are different from those of citrate[11].

Zusammenfassung. Nach einer Bestrahlung mit 690 R beruhen Veränderungen des Citratgehaltes in der Leber gefütterter Mäuse auf unregelmässiger Nahrungsaufnahme, während in 24 h hungernden Mäusen der Citratgehalt nach 690 R über 11 Tage fast unverändert bleibt und erst vom 12.–15. Tag nach der Bestrahlung ansteigt. Die Ergebnisse werden diskutiert im Hinblick auf Phosphofruktokinase – und Acetyl-CoA-Carboxylase-Aktivität.

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Possible Abiotic Origin of Precambrian Microfossils

Biochemical and electron microscope investigations of selected precambrian rocks have shown evidence for the existence of microfossils believed to be contemporary with the rocks[1,2]. Related findings have revealed indigenous amino acids and alkanes in the samples.

Over the past decade it has been shown that a large variety of biochemicals can be formed from simple gases and liquids under the action of high energy sources using hypothetical primitive earth conditions (for reviews see reference[3]). Recent results have also demonstrated that the transition from simple molecules to macromolecules is often accompanied by a separation of microstructures from the medium[4–6]. These findings and those to be presented here support the suggestion of a possible abiotic origin of the microfossil forms found in the precambrian rocks.

As a starting material for our experiments we used ammonium thiocyanate (NH_4SCN) which is a known product of juvenile volcanic gases[7] and has been produced under simulated primitive earth conditions[8]. In previous publications we showed that a small amount of methionine is synthesized by UV-irradiation of NH_4SCN[9] and that cell-like structures are produced in the presence of formaldehyde[10]. In the present communication we report evidence demonstrating a resemblance between these abiotically produced microspheres and the microfossils on the basis of (a) morphology, (b) chemical composition and (c) physical properties.