mitochondria depleted of unsaturated fatty acids results in a recoupling of oxidative phosphorylation. Recoupling is insensitive to both chloramphenicol and cycloheximide, indicating that all the proteins necessary for oxidative phosphorylation are present in the depleted mitochondria and that the loss of oxidative phosphorylation is purely a lipid lesion.

The effects of depletion of unsaturated fatty acids on the energy-linked reactions of yeast mitochondria have been investigated. This approach is restricted by the absence from mitochondria of *S. cerevisiae* of many of the energy-dependent reactions that are present in mammalian mitochondria, including Site 1 phosphorylation, the reduction of NAD⁺ by succinate, the transhydrogenation of NADP⁺ by NADH and the accumulation of Ca²⁺. However, yeast mitochondria accumulate K⁺ in the presence of valinomycin and an oxidizable substrate; accompanying this active transport there is a stimulation of respiration. The ability of valinomycin to stimulate the energy-dependent uptake of K⁺ and to accelerate respiration is progressively lost as the mitochondrial content of unsaturated fatty acids is lowered, and is completely absent from mitochondria in which less than 20% of the fatty acids are unsaturated.

The effects of the lipid lesion on the terminal reactions of phosphorylation were investigated. The adenosine triphosphatase activity of mitochondria depleted of unsaturated fatty acids is normal, and is inhibited by oligomycin or by a protein inhibitor from ox heart mitochondria. P_i-ATP exchange activity is also unaffected by depletion of unsaturated fatty acids and is inhibited by oligomycin or uncouplers.

The mutant requiring unsaturated fatty acids appears to be a suitable organism for studying the role of lipids in mitochondrial membrane function and structure.

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Dietary Carbohydrate and Fatty Acid Synthetase Activity in Rat Liver and Adipose Tissue

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Substitution of sucrose for all or most of the starch in the diet increases the plasma triglyceride concentration in man (Macdonald & Braithwaite, 1964) and in the rat (Akinyanju & Yudkin, 1967).

Naismith (1971) demonstrated an increased activity in some of the enzymes involved in lipogenesis in rats fed on sucrose. We have now studied the effect of these and other carbohydrates on the activity of fatty acid synthetase in male rats. This was measured at 37°C in supernatants of tissue homogenates centrifuged at 30000g (Oesterhelt, 1967). Activity was expressed in units of μmol of malonyl-CoA incorporated into fatty acid-CoA/min/g of tissue.

Two groups each of five weanling rats were fed on a diet containing 68% carbohydrate as corn starch or sucrose. After 30 days the average plasma triglyceride concentration was 50mg/100ml on the starch diet and 82mg/100ml on the sucrose diet; liver synthetase activities were 0.86 and 2.5 units respectively.

In the second experiment several groups each of four rats aged 7 weeks were placed on diets with either starch or sucrose; after 3 weeks the rats were transferred from the one diet to the other. After 18h liver synthetase activity was two- to three-fold greater in the rats fed on sucrose than in those fed on starch, and this difference was maintained for the 3 weeks. The reversal of the diets at this time again led after 18h to two- to three-fold greater enzyme activity in the rats now on the sucrose diets. There were similar changes in plasma triglyceride concentration in the same direction.

In a third experiment groups of eight rats were fed on diets with 68% carbohydrate as starch, sucrose, fructose, glucose or maltose. After 30 days synthetase activity in the liver was 0.9, 2.1, 2.8, 0.9 and 1.4 units respectively; in the adipose tissue the activity was 0.12, 0.06, 0.03, 0.14 and 0.20 units respectively. Plasma triglyceride concentrations, in the same order, were 53, 79, 99, 57 and 47mg/100ml. Enlarged livers were found in the rats fed on sucrose, and especially in those fed on fructose.

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