version of acetate and mevalonate into cholesterol by rat liver homogenates. The activities of mevalonate kinase and phosphomevalonate kinase change either only slightly or not at all on starvation for 48 h and re-feeding for 144 h. The activities of all the other enzymes for which assays were performed changed drastically under the above conditions. It appears from determinations of the constants for the rates of synthesis and degradation of mevalonate pyrophosphate decarboxylase and squalene synthetase that these proteins vary in quantity in rat liver because of changes in the rate of their synthesis. In contrast, the changes in the activity of isopentenyl pyrophosphate isomerase appears to be due to a change in the rate of degradation of this protein. The rates of both synthesis and degradation of farnesyl pyrophosphate synthetase change on starvation and re-feeding.

The activity of 3-hydroxy-3-methylglutaryl-CoA reductase changes drastically on starvation and re-feeding. However, when an attempt is made to calculate rate constants for this enzyme by the rate of approach to a steady state, a curved rather than a straight line is obtained. Similarly, although one is able to calculate rate constants during starvation for acetyl-CoA carboxylase and the fatty acid synthetase, this method of analysis fails when applied to the results obtained on re-feeding the animals. Further investigations are being carried out on variations in the activities of these enzymes in liver by methods that utilize labelling of the enzyme *in vivo* or synthesis of the enzyme by isolated liver cells.

The results obtained strongly suggest that the genes coding for the enzymes of cholesterol synthesis are not co-ordinately controlled. In contrast, the activities of acetyl-CoA carboxylase and fatty acid synthetase change together, thereby suggesting that these enzymes are under a single control (and that the genes coding for these enzymes may be located on the same chromosome). There is also a relationship between the enzymes effecting the synthesis of cholesterol and those effecting the synthesis of fatty acids. When animals are transferred from a normal to a fat-free diet, the activities of acetyl-CoA carboxylase and fatty acid synthetase rise to values several times those of controls. At the same time the activities for 3-hydroxy-3-methylglutaryl-CoA reductase and for the conversion of acetate and mevalonate into cholesterol fall markedly. These results suggest that there is a cellular mechanism by which acetyl-CoA can be shunted into the pathway for fatty acid synthesis and away from the pathway for cholesterol synthesis when a fat-free diet is given.

Correlation of Chemical Structure and Biological Activity of Mycobacterial Cell Walls and Derived Lipids

By E. LEDERER (*Institut de Chimie des Substances Naturelles, Centre Nationale de Recherche Scientifique, 91-Gif-sur-Yvette, and Institut de Biochimie, Centre d'Orsay, Université de Paris-Sud, 91-Orsay, France*)

The mycobacterial cell wall contains a peptidoglycan-glycolipid complex and various more-or-less defined glycolipid fractions. The chemistry has been described in detail (Petit *et al.*, 1969; Adam *et al.*, 1969; Amar-Nacasch & Vilkas, 1970; Azuma *et al.*, 1970; Wietzerbin-Falszpan *et al.*, 1970; for a review see Lederer, 1971).

Mycobacterial cell walls have several interesting biological properties, such as immunological adjuvant activity (White, 1965) and specific and non-specific immunization (Anacker *et al.*, 1969; L. Chedid & A. Lamensans, unpublished work).

Some of these activities are also shown by some glycolipids and other compounds that are biogenetically or functionally more or less closely related to the mycobacterial cell wall, such as wax D (White *et al.*, 1964; Markovits *et al.*, 1971) and cord factor (Bekierkunst *et al.*, 1969).

It is intended to give an up-to-date and critical summary of recent work in this field where the frequent absence of criteria of chemical purity and the variety of biological test systems used are still preventing a general consensus among various workers in the field.

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