

Biochemical mechanisms related to the homeostatic regulation of lipogenesis in animals

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Since most animals are intermittent eaters, they are confronted with the problem of temporarily storing much of the chemical energy ingested as food. Fat takes up less volume and weighs less per calorie of stored chemical energy than either carbohydrate or protein. Thus, in animals whose major foodstuff is carbohydrate, the use of lipogenesis as the primary means by which the energy of dietary carbohydrate is stored affords maximum efficiency. The major homeostatic function of lipogenesis is to store as fat the chemical energy of carbohydrate ingested in excess of the immediate energy requirements of the organism.

Clearly, a process as vital to caloric balance as fat synthesis must be precisely regulated to meet the ever-changing energy needs of the organism. While the experimental data on the regulation of lipogenesis are vast, they present a far from complete picture. The purpose of this review is to analyze these data and, when possible, formulate some general concepts concerning the biochemical mechanisms involved in this homeostatic regulation.

EFFECT OF FASTING ON LIPOGENESIS

That the rate of fatty acid synthesis can be markedly altered by changes in nutritional state was first demonstrated in 1944 by Boxer and Stetten (1) and Bernhard and Steinhauser (2). These investigators labeled the body water of rats with D_2O and found that either under-nutrition or a period of fasting greatly reduced the amount of deuterium incorporated into the body fat. The authors concluded that lipogenesis is greatly curtailed during conditions of restricted caloric intake.

In vitro studies by Bloch (3) and by Masoro *et al.* (4) using liver slices localized the impairment in lipogenesis to some point between acetyl-CoA¹ and fatty acids. Bloch found that liver slices from fasted rats had a markedly reduced ability to convert acetate- C^{14} to fatty acid. Masoro *et al.* (4) showed that, when

liver slices prepared from rats fasted for 24 hours were incubated in the presence of 400 mg per 100 ml of glucose- C^{14} , almost no C^{14} was incorporated into the fatty acids. However, the conversion of the glucose- C^{14} to $C^{14}O_2$ was not altered by the 24-hour fast, a fact that indicates a normal ability to form labeled acetyl-CoA from glucose. It is evident that under these conditions the reduced rate of lipogenesis did not result from a lack of substrate (acetyl-CoA) derivable from glucose but rather from an inability to utilize acetyl-CoA in fatty acid synthesis.

The biological importance of the restricted utilization of acetyl-CoA for fatty acid synthesis becomes clear when one considers the metabolic problems facing the fasting animal. The following abbreviated metabolic scheme assists in this consideration:



During fasting, very little glucose is available to the tissues of the body. Therefore, little acetyl-CoA is formed from glucose by way of reaction sequence I, and the homeostatic requirement for storing glucose carbon as fatty acid is abolished. However, acetyl-CoA is generated by way of reaction sequence II at a much higher rate than in fed animals (5). Therefore, if reaction sequence III were not greatly inhibited during fasting, a great amount of the fatty acid undergoing β -oxidation would be needlessly recycled.

¹ The following abbreviations are used in the text: CoA, coenzyme A; TPN, oxidized triphosphopyridine nucleotide; TPNH, reduced triphosphopyridine nucleotide; DPNH, reduced diphosphopyridine nucleotide; ATP, adenosine triphosphate; ADP, adenosine diphosphate; P_i, inorganic orthophosphate; ATPase, adenosine triphosphatase; FFA, free fatty acid or acids.

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