identical with the elongation factors of the supernatant, are present in the ribosomal fraction. These factor(s), presumably, are involved in initiation: however, further investigation is required to elucidate this point. Moreover, preliminary results indicate that a fraction with stimulatory activity similar to that described above can be removed from ribosomes with 0.25 M-ammonium chloride. The nature of this fraction is also currently under investigation.

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Glycogen Synthesis in the Perfused Liver of Starved Rats

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In order to investigate the factors that control the reaccumulation of hepatic glycogen after starvation we have measured net rates of glycogen deposition in perfused livers from starved rats. Livers from 170–190 g male rats, starved for 48 h, were perfused with bicarbonate–albumin–saline, essentially as described by R. Hems, Ross, Berry & Krebs (1966), except that washed erythrocytes from fresh defibrinated rat blood (Baron & Roberts, 1963) were employed, so that haemoglobin was 4% (w/v). Liver samples (0.5 g) were taken at 20 min (ventral notched lobe) and 50 min (large main lobe).

When livers from 48 h-starved rats were perfused with glucose alone (30 mM) glycogen deposition was 0–0.4 $\mu\text{mol/min per g wet wt. of liver}$, but if pyruvate, glycerol and serine (each 5 mM) were added after 15 min perfusion with 30 mM-glucose the glucose concentration in the medium was stabilized and the rate of glycogen accumulation was 0.68 ± 0.05 (10) $\mu\text{mol/min per g wet wt. of liver}$. Unless the glucose concentration in the medium was 25–30 mM, glycogen accumulation was less than

0.5 $\mu\text{mol/min per g wet wt. of liver}$ and glucose was released into the medium.

When liver was perfused under the present conditions with glycerol, pyruvate and serine (each 5 mM) in the absence of glucose gluconeogenesis was 1.4 $\mu\text{mol/min per g wet wt. of liver}$ for 20–35 min, after which the rate decreased. In this experiment average glucose formation between 20–50 min was 0.76 $\mu\text{mol/min per g wet liver}$. Rapid glucose synthesis from substrate combinations including glycerol has been reported previously (Ross, R. Hems, Freedland & Krebs, 1967).

The present experiments suggest that the initial phase of post-starvation hepatic glycogen deposition after ingestion of glucose may largely be a consequence of gluconeogenesis; such a possibility was suggested from the results of experiments *in vivo* carried out by Olavarria, Godeken, Sandruss & Flawia (1968). This would resolve the difficulty that hepatic glucokinase activity in the post-starvation situation is not sufficient to support conversion of administered glucose into glycogen at the observed rate of glycogen deposition (for references and discussion see Friedmann, Goodman & Weinhouse, 1967; Olavarria *et al.* 1968). Initiation of glycogen accumulation apparently may occur solely as a result of a rise in blood glucose concentration, perhaps due to activation of glycogen synthetase (see Hers, de Wulf, Stalmans & Van den Berghe, 1970).

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The Physiological Role of L-Tyrosine Methyl Ester Sulphotransferase

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A sulphotransferase has been obtained from rat livers that appears to be specific for simple derivatives of L-tyrosine (Mattock & Jones, 1970). The physiological role of this enzyme is unknown,