between the activity of the deacylation and 3-hydroxy-3-methylglutaryl-CoA pathways. The changes in the capacity of the 'deacylase pathway' reported by Mulder *et al.* (1977) to occur in the cytosolic and mitochondrial fractions of cow and rat liver are therefore likely to be due to contamination of cytosolic fractions by mitochondrial damage during tissue extraction.

The activities of 3-oxo acid CoA-transferase found in the livers of ruminants (Table 1) are similar to those found in the livers of other mammals (Beis, 1977) and most other vertebrates (Zammit *et al.*, 1979). The activity of this enzyme, like that of 3-hydroxy-3-methylglutaryl-CoA lyase, was found to be associated entirely with the mitochondrial fraction (Table 1). Recent studies have also shown that (at least in the rat) the enzyme is present in the parenchymal liver cells (V. A. Zammit, unpublished work). It has been suggested (Zammit *et al.*, 1979) that 3-oxo acid CoA-transferase is involved in the feedback regulation of ketogenesis in liver by ketone bodies in liver mitochondria. The mitochondrial localization of this enzyme suggests that 3-oxo acid CoA-transferase may indeed have access to the same pool of acetoacetyl-CoA as the enzymes of the 3-hydroxy-3-methylglutaryl-CoA pathway, and may thus be involved in the regulation of acetoacetyl-CoA concentrations in liver mitochondria.

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The development of hepatic fatty acid oxidation at birth in the rat

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At birth the transplacental diet containing glucose as the major oxidizable substrate is replaced by the high-fat-content milk diet of suckling. During the suckling period in the rat the hepatic capacity for fatty acid oxidation (Foster & Bailey, 1976*a*) and ketogenesis (Foster & Bailey, 1976*b*; Shah & Bailey, 1977) is considerably higher than the low values in the foetus. However, the detailed changes in hepatic fatty acid oxidation and ketogenesis and the factors that control these changes in the newborn are not fully understood. The present report correlates changes in blood and hepatic metabolites with changes in the activities of enzymes of β-oxidation and ketogenesis in the immediate postnatal period and the effect of starvation on these processes.

It was observed that a period of transient hypoglycaemia occurs at about 1.5 h after birth in both fostered and starved newborns. However, in the starved animals a profound hypoglycaemia develops after 6 h. Similar observations have been reported previously (Snell & Walker, 1973; Girard *et al.*, 1973). Both blood acetoacetate and D-3-hydroxybutyrate concentrations increase markedly 6 h after birth in fostered animals but not in starved, as also found by Girard *et al.* (1973) and Ferré *et al.* (1978). In starved newborns the unesterified fatty acid content of the plasma changes little over the period studied, whereas in fostered animals an increase after 1.5 h to a plateau 6 h after birth is noted. Interestingly, a further increase is observed between 18 and 24 h after birth (Foster & Bailey, 1976*b*). In contrast, the plasma triacylglycerol concentration in starved animals falls after birth, whereas that in the fostered animals increases only after 12 h of life.

The liver glycogen concentration falls after birth in both fostered and starved animals, with depletion occurring by 12 h in starved and by 18 h in the fed animals. Similar results have been observed by Snell & Walker (1973) and Ferrè *et al.* (1978). The liver triacylglycerol concentration changed little after birth in starved newborns, but increased markedly after 12 h in the fed (a developmental profile similar to that of the plasma triacylglycerols).

Hepatic mitochondrial palmitoyl-CoA synthetase activity increases in suckled pups to a maximum within 3 h after birth. The rapidity of this rise suggests that activation rather than induction of enzyme synthesis may be involved. In contrast, in the absence of suckling, the enzyme activity remains relatively constant after birth. The activity of the other fatty acid-oxidation enzyme studied, carnitine palmitoyltransferase I, changes little over the first 18 h in both sets of experimental animals. However, further studies indicate a large increase in enzyme activity occurring between 18 and 24 h after birth. The activity of hepatic mitochondrial hydroxymethylglutaryl-CoA synthase decreases in both sets of experimental animals during the initial 6 h after birth. After this, a sustained increase in activity during the remainder of the period studied is only seen in the suckled pups.

The results indicate that the well-documented increase in hepatic fatty acid oxidation and ketogenesis that occurs after birth requires the stimulus of a high-fat-content diet and is dependent on increased activities of mitochondrial acyl-CoA synthetase and hydroxymethylglutaryl-CoA synthase.

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