Increase with starvation in the pregnant rat of the liver lipoprotein lipase activity

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Lipoprotein lipase (EC 3.1.1.34) activity present in the capillary endothelia catalyses the hydrolysis of triacylglycerols in circulating triacylglycerol-rich lipoproteins, the products of which are taken up by the subjacent tissue (Nilsson-Ehle *et al.*, 1980; Lasunción & Herrera, 1983). Although the enzyme is generally found in extrahepatic tissues and not in liver (Nilsson-Ehle *et al.*, 1980), we have detected its transitory presence in the liver during the perinatal phase in the rat (Llobera *et al.*, 1979; Ramírez *et al.*, 1983). At late gestation, maternal circulating triacylglycerol-rich lipoproteins increase as result of enhanced production from endogenous triacylglycerols (Humphrey *et al.*, 1980) and decreased removal (Otway & Robinson, 1968). With starvation, the mother shows a further increase in circulating triacylglycerol concentration concomitant with enhanced ketosis (Scow *et al.*, 1964; Herrera *et al.*, 1969*a*). To relate these changes to possible modifications in lipoprotein lipase activity in adipose tissue or liver, in the present work we studied 21-day-pregnant Wistar rats and age- and sex-matched virgin controls. Animals were killed by guillotine in the fed state or after 24h starvation. Livers and lumbar fat-pads were rapidly frozen in liquid N_2 and kept at −80°C until processed. Lipoprotein lipase activity was measured in acetone/diethyl ether extracts by the method described by Llobera *et al.* (1979), and with a stable substrate emulsion specific for lipoprotein lipase assay (Nilsson-Ehle & Schotz, 1976; Corey & Zilversmit, 1977). Inhibitory characteristics in the presence of NaCl and protamine sulphate have previously been tested in adipose tissue and liver of fed adult rats, and were found to correspond to those of lipoprotein lipase activity in adipose tissue, but not in liver (Ramírez *et al.*, 1983). In the present study these characteristics were assayed in the liver of 24h-starved pregnant rats, and 86.9% inhibition was found when the enzyme was measured in the presence of 1M-NaCl, indicating that the activity found in this preparation corresponded to lipoprotein lipase.

As shown in Table 1, adipose-tissue lipoprotein lipase activity was much lower in fed 21-day-pregnant rats than in virgin animals, and in both groups 24h starvation decreased this activity. In the liver of fed animals, lipoprotein lipase activity was lower than in adipose tissue, and values in pregnant and virgin animals were similar. With 24h starvation there was a significant increment in lipoprotein lipase activity in the liver of the pregnant rats, whereas no change was found in starved compared with fed virgins (Table 1).

Present results in fed animals coincide with previous findings (Llobera *et al.*, 1979; Ramírez *et al.*, 1983) and indicate that maternal hypertriacylglycerolaemia is influenced by decreased removal of triacylglycerol-rich lipoproteins, owing to decreased lipoprotein lipase activity in adipose tissue. Maternal hypertriacylglycerolaemia is known to be further enhanced in the starved condition (Scow *et al.*, 1964), which may also be influenced by the diminished lipoprotein lipase activity in adipose tissue of the starved mother. The fate of those triacylglycerols and their physiological role in the starved pregnant rat are uncertain, but their increase coincides with marked ketosis in the mother (Scow *et al.*, 1964; Herrera *et al.*, 1969*a*). The source of these ketone bodies is the liver, which must support an enhanced ketogenesis at the expense of circulating lipids. Thus the augmented lipoprotein lipase activity in the mother's liver after food deprivation may represent a mechanism increasing fatty acid uptake from circulating triacylglycerol-rich lipoproteins (and/or their remnant particles). The factors modulating this change in liver lipoprotein lipase activity in the starved mother are unknown, but this effect coincides with other metabolic and hormonal changes, such as an exaggerated hypoglycaemia and catecholamine production (Herrera *et al.*, 1969*a*, *b*). It is possible that these two factors could modulate such change in the liver of the starved mother in a similar way as they do for the heart enzyme in the non-pregnant rat. Further studies are required to elucidate these points.

Table 1. *Effect of 24h starvation on adipose tissue and liver lipoprotein lipase activity in 21-day-pregnant and virgin rats*

Rats were killed by guillotine, and liver and lumbar fat-pads were immediately frozen in liquid N_2 and kept at −80°C until assayed. Enzyme activity was measured in acetone/diethyl ether extracts by a procedure described previously (Nilsson-Ehle & Schotz, 1976; Corey & Zilversmit, 1977; Llobera *et al.*, 1979), and is expressed as nkat/100g fresh wt. of tissue. Values are means ± S.E.M. for five to nine rats/group. *P* refers to the statistical comparison between starved and fed animals, whereas asterisks refer to the comparison between pregnant and virgin rats (**$P<0.01$; ***$P<0.001$); N.S., not significant.

	Adipose tissue	Liver
Pregnant rats		
Fed	55.3 ± 11.8	24.2 ± 3.9
24h Starved	24.5 ± 2.5	70.1 ± 6.9
P	<0.05	<0.001
Virgin rats		
Fed	241.0 ± 55.1***	20.9 ± 3.2
24h Starved	57.1 ± 8.0**	22.6 ± 2.1***
P	<0.01	N.S.

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