

LACTATE METABOLISM IN RUMINANT ADIPOSE TISSUE

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A paper by Hanson and Ballard (1967) is one of the most widely quoted papers on lipid metabolism in ruminant tissues. These authors concluded that acetate was the major substrate for fatty acid synthesis in ruminant adipose tissue and that glucose was poorly utilised for fatty acid synthesis. They attributed poor utilisation of glucose to low activities of ATP-citrate lyase and NADP-malate dehydrogenase. Since 1967, others have also concluded that glucose is not utilised for fatty acid synthesis in ruminant tissues because of negligible citrate cleavage pathway.

Recently Whitehurst et al. (1978) and Prior (1978) have indicated that ruminant adipose tissue can use lactate as a substrate for fatty acid synthesis. It is presumed that lactate is converted to fatty acid via the citrate cleavage pathway. This study was undertaken to identify the pathway for the synthesis of fatty acid from lactate in ruminant adipose tissue.

Thin slices of subcutaneous adipose tissue from four steers were incubated with either 25 mM 1-¹⁴C-acetate, 5 mM U-¹⁴C-glucose or 100 mM U-¹⁴C-lactate. In 2 h, lactate (1250 nmol/100mg was used for fatty acid) synthesis at a greater rate than acetate (800 nmol/100mg or glucose) (55 nmol/100mg). The activity of ATP-citrate lyase was sufficient to support lipogenesis from lactate.

The following inhibitors were added to the incubation media:

- I 2 mM α -cyano-4-OH-cinnamate
- II 10 mM n-butylmalonate
- III 60 μ M agaric acid
- IV 5 mM sodium oxamate

Inhibitor I decreased fatty acid synthesis from all substrates to 5% of control incubations, due to general disruption of cellular metabolism by this compound. Inhibitors II and III had no effect on fatty acid synthesis from any of the three substrates, suggesting a lack of uptake of these compounds into intact adipocytes. An inhibitor of lactate dehydrogenase (IV) decreased fatty acid synthesis from lactate by 40% and had no effect on synthesis from acetate or glucose. Oxamate also decreased synthesis of glyceride-glycerol and production of CO₂ from lactate.

Although the inhibitors were poorly absorbed into intact adipocytes, the results suggest that lactate is metabolised, by lactate dehydrogenase, to pyruvate, which then is converted to fatty acid via the citrate cleavage pathway.

HANSON, R.W. and BALLARD, F.J. (1967). Biochem. J. 105: 529.

PRIOR, R.L. (1978). J. Nutr. 108: 926.

WHITEHURST, G.B., BEITZ, D.C., POTHOVEN, M.A., ELLISON, W.R. and CRUMP, M.H. (1978). J Nutr. 108: 1806.

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