Marked hyperlipidaemia in rats bearing the Yoshida AH-130 ascites hepatoma

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Malignant tumour burden is a pathological state associated with a considerable demand for nutrients by the growing tumour [1]. Although a great deal of work has been carried out, there are contradictory results about the effects of tumour burden on lipid metabolism both in experimental animals and man. Lipid oxidation has been reported either increased [2], unchanged [3] or decreased [4]. In addition, lipoprotein lipase activity decreases in adipose tissue, with a concomitant increase in plasma triacylglycerol levels [5]. We have examined the effects on in vivo lipid metabolism induced by the presence of the rapidly growing tumour Yoshida AH-130 ascites hepatoma.

Female Wistar rats fed ad libitum were injected intraperitoneally with a Yoshida AH-130 ascites hepatoma cell suspension (120 x 10^6 cells). The Yoshida AH-130 is a rapidly growing tumour with a volume doubling time of 1 day [6]. In these animals we examined the metabolic fate of an orally administered triolein [7], the lipoprotein lipase (LPL) activity [8], as well as the lipogenic rate [9]. We also determined the levels of both triacylglycerols (TAG) and non-esterified fatty acids (NEFA) in plasma. The experiments were performed either 4 or 7 days after tumour inoculation.

The implantation of the Yoshida AH-130 ascites hepatoma resulted in a decrease in body weight. In addition, the tumour induced a marked hyperlipidaemia, which was explained both by an increased hepatic lipogenic rate and a decreased circulating triacylglycerol clearance; however, the animals showed a decrease in the intestinal absorption of an oral triolein load. Although hypophagia can explain some of the observed changes in lipid metabolism in this experimental model, others factors must be involved in fat depletion and hyperlipidaemia. Cytokines, tumour necrosis factor-α in particular, could explain some of the changes in lipid metabolism related with cancer cachexia [10].