T cells in flow-based adhesion assays. Total adhesion of CD4 and CD8 T cells was significantly (p < 0.05) reduced when ICAM-1, VCAM-1, and CXCR3 molecules were blocked or if G protein coupled receptors were inhibited with pertussis toxin (PTX). CD8 T cell adhesion was also dependent on vascular adhesion protein-1.

Conclusion We report high IL-18 expression by Kupffer cells in inflammatory liver disease. The ability of IL-18 to enhance T cell recruitment via sinusoidal endothelium suggests it acts to promote lymphocyte recruitment during the development of chronic hepatitis and is thus a potential novel therapeutic target in inflammatory liver disease.

Competing interests None declared.

EICOSAPENTAENOIC ACID IS EFFECTIVE AT REDUCING HEPATOCYTE TRIGLYCERIDE CONTENT OF UNTREATED C3A CELLS BUT IS NOT EFFECTIVE IN TWO MODELS OF CELLULAR STEATOSIS

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Introduction Eicosapentaenoic acid (EPA), one of the major physiologically active constituents of Omega-3 fatty acids, has been suggested as a treatment for non-alcoholic fatty liver disease (NAFLD). The aim of these experiments was to assess the effects of EPA on intrahepatic triglyceride content in cell culture models of steatosis.

Methods Human C3a hepatocytes were incubated in MEME (standard media) and two models of cellular steatosis: olate (a model of isolated steatosis) and LPON (a model of steatosis and mitochondrial dysfunction containing the gluconeogenic substrates Lactate, Pyruvate, Octanoate and ammoNia). Test media was either un-supplemented, or supplemented with 50 μM or 250 μM EPA. Hepatocyte triglyceride accumulation was assessed both by microscopy (using oil red staining) and by quantifying the intra-cellular triglyceride concentration of cells incubated in culture media for 5 and 7 days. Each cell culture experiment was performed in triplicate.

Results MEME When quantified by oil red staining a 73.1% (95% CI 63% to 83%) reduction in cell triglyceride content with 250 μM EPA compared with untreated cells was seen (7659 vs 28 564 pixels; p < 0.001). This was confirmed in cell culture experiments as 250 μM EPA was associated with reduced intrahepatic triglyceride content after both 3 (74.1 vs 94.9 mmol/g; p < 0.05) and 7 days (62.2 vs 80.9 mmol/g; p < 0.05) incubation compared with untreated cells equating to a 21.9% (95% CI 9% to 35%) and 23.1% (95% CI 5% to 41%) reduction respectively. For both experiments a linear trend between increasing EPA concentration and reduced triglyceride content was confirmed. Olate Here reduced triglyceride content with both 50 µM EPA (p < 0.01) and 250 µM EPA (p < 0.05) was seen with oil red staining and equated to reductions of 27.6% (95% CI 16% to 39%) and 22.5% (95% CI 9% to 36%) compared with untreated cells. However these results were not reproduced in cell culture experiments although on post test analysis there was a significant linear trend between increasing EPA concentration and reduced triglyceride content (p = 0.04). LPON Although incubation with 250 μM EPA reduced triglyceride content in the LPON model when quantified with oil red staining (60 308 vs 79 219 pixels in 250 μM EPA vs untreated cells, p < 0.05) this was not confirmed in cell culture experiments. On post hoc analysis no trend was demonstrated between protected EPA concentration and triglyceride content.

Conclusion These results suggest that EPA is effective at reducing triglyceride accumulation in untreated hepatocytes but is not effective in either olate or LPON models of cellular steatosis. It is therefore possible that the presence of steatosis and mitochondrial dysfunction in NAFD may limit the efficacy of EPA as a treatment.

Competing interests None declared.