

Clinical care and other categories posters: lipids and fatty liver

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Exercise reduces liver fat and improves body composition but not insulin sensitivity or inflammatory markers in non-alcoholic steatohepatitis

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Aims: Non-alcoholic steatohepatitis (NASH) represents steatosis and ballooning degeneration, inflammation and fibrosis, and is the hepatic manifestation of insulin resistance. Current treatments for NASH are limited, with no randomised controlled trials (RCTs) reporting the effects of exercise on insulin resistance in people with NASH. We aimed to investigate the effect of exercise on liver lipid, circulatory inflammation and insulin resistance in people with NASH, independent of weight loss.

Methods: 24 participants (mean age 52 ± 14 years, body mass index 33 ± 6) with histologically characterised NASH received resistance exercise ($n = 12$) or continued standard care ($n = 12$) over 12 weeks. Liver lipid content, body composition, inflammatory markers and insulin resistance were assessed at baseline and at 12 weeks.

Results: Relative to baseline exercise significantly reduced liver fat (-16 ± 24 vs $+9 \pm 15\%$, $p < 0.05$), visceral fat (-22 ± 33 vs $+14 \pm 48$ cm^2 , $p < 0.05$) and plasma triglycerides (-0.5 ± 1.0 vs $+0.3 \pm 0.4$ mmol/l , $p < 0.05$). Metabolically, there were reductions in fasting insulin, HOMA-IR, HbA1c and area under the glucose curve but these were not significant. There was no effect of exercise on liver enzymes or circulatory inflammation (IL6, TNF α , hsCRP and CK-18).

Conclusions: Exercise produced a significant reduction in liver fat and improved body composition, but had no effect on insulin resistance or inflammation in patients with NASH. This is the first RCT in patients with NASH and suggests that exercise alone may be insufficient to target its mediators and warrants further exploration.

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Poor glycaemic control is associated with higher serum triglyceride levels in clinical practice

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Aims: The relationship between glycaemic control and lipid metabolism in diabetes is complicated and yet to be fully elucidated. Here we aim to characterise the relationship between glycaemic control and serum triglyceride levels in a population with Type 2 diabetes.

Methods: We performed a retrospective cross-sectional analysis using the primary care records of a large number of people with Type 2 diabetes ($n = 34,278$). Data were collected as part of the University of Surrey – Lilly Real World Evidence (RWE) centre projects from over 100 primary care centres across England and Wales. All data used were collected as part of routine care. We performed a linear regression analysis to identify any independent relationship between serum triglyceride measurements and HbA1c adjusting for patient demographics and lipid lowering medications.

Results: A total of 22,702 people had complete data for all the variables of interest and were included in the final analysis. The mean HbA1c and triglyceride levels for the included cohort were 56.96 mmol/l and 1.84 mmol/l respectively. Age, gender, deprivation score, body mass index, use of insulin and use of lipid lowering medication were all correlated with triglyceride levels and were therefore included in the regression model. After adjusting for these confounders we identified a small but highly significant relationship between HbA1c and serum triglycerides with an increase of 5.2×10^{-4} mmol/l per mmol/l of HbA1c increase ($p < 0.0001$).

Conclusions: Poor glycaemic control is associated with higher serum triglycerides. The direction of causality for the association should be established.