

## High intensity interval training and intramuscular lipid metabolism in healthy and obese humans

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Intramuscular lipids (IMCL) are primarily stored in small, discrete lipid droplets (LD) and 2-3-fold higher IMCL concentrations are found in type I muscle fibres. IMCL can be utilized as an energy source during exercise in type I muscle fibres and IMCL oxidation accounts for up to 50% of total fat oxidation during moderate intensity exercise. Regular exercise increases IMCL storage and a greater utilisation of IMCL accounts for the elevation in fat oxidation seen during exercise following a period of endurance-based exercise training. On the other hand, IMCL accumulation in the muscle of sedentary individuals is associated with impairments in insulin signal transduction via the accumulation of lipid metabolites, such as diacylglycerol and ceramides. As sedentary individuals exhibit low rates of IMCL utilisation, it is hypothesized that lipid metabolite accumulation is a consequence of a low turnover of the IMCL pool. As regular exercise stimulates frequent cycles of IMCL depletion and IMCL resynthesis in the post-exercise period, increasing the turnover of the IMCL pool may contribute to the insulin sensitisation of skeletal muscle following exercise training.

Previous studies have demonstrated that short term high intensity interval training (HIIT) using the 30 s 'all-out' Wingate-based approach increased the maximal activity of  $\beta$ -HAD in skeletal muscle and elevated rates of whole body fat oxidation to a similar extent as endurance-based exercise training (ET) (Burgomaster *et al.*, 2008). As such, changes in IMCL metabolism and whole body insulin action in response to HIIT and endurance-type exercise training have been investigated. Using the same 30 s 'all-out' protocol, it was demonstrated that 6 weeks of HIIT or ET in young lean males is sufficient to improve insulin sensitivity, elevate IMCL content and increase the expression of the LD-associated proteins perilipin 2 and perilipin 5 in both type I and type II muscle fibres (Shepherd *et al.*, 2013). Furthermore, IMCL breakdown in type I muscle fibres during 1 h of continuous, moderate intensity exercise was increased following both training modes. Subsequently, an adapted HIIT protocol using repeated 30 s bouts of exercise performed at 200% of peak power output for 4 weeks was employed in young obese males, alongside an ET control group. These training modes improved aerobic capacity and insulin sensitivity to a similar extent. The expression of perilipin 2 and perilipin 5 in type I muscle fibres was also increased in response to both training interventions whereas IMCL content was unchanged. In addition, an increase in LD-mitochondria interactions was observed post-training which may be important for greater IMCL utilization during exercise.

The observed improvements in the capacity to store and utilize IMCL may be mediated by the increased expression of the LD-associated perilipin proteins and likely contributes to the improvement in insulin sensitivity following exercise training. These findings add new information on how exercise training improves IMCL metabolism and add to the growing body evidence to support HIIT as an effective means to improve muscle insulin sensitivity.

Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, MacDonald MJ, McGee SL & Gibala MJ. (2008) *Journal of Physiology*, **586**, 151-60.

Shepherd SO, Cocks M, Tipton KD, Ranasinghe AM, Barker TA, Burniston JG, Wagenmakers AJ & Shaw CS. (2013) *Journal of Physiology*, **591**, 657-75.