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Reply by Zafeiridis and Mougios

We thank Drs Burns and Stensel for their interest in our work. We agree that the published articles on the delayed effect of resistance exercise (RE) on postprandial lipaemia (PL) provide controversial results. Three studies[1–3] employed comparable methodologies in terms of exercise protocol and feeding plan of the subjects, that is, two to four sets of eight to eleven exercises at relatively similar intensities (about 10–12 repetitions maximum (RM)) with 1·5–2·0 min of rest between sets and a standardised meal on the night prior to the fat tolerance test. These studies reported a decrease[1,3] or no change[2] in the postprandial lipaemic response. A fourth study[4] employed a similar RE protocol but focused on maintaining the subjects in a state of energy balance by increasing food intake up to two-fold after RE *v.* control on the night prior to the fat tolerance test. This study found no significant effect of RE on PL.

Shannon *et al.*[4] point out that the increased food intake in the exercise trials 'may have shortened the duration of the mechanisms involved with replenishing energy stores. This is reflected by our data indicating no changes in lipemic [...] responses between treatments'. They also state: 'The lack of a direct comparison group that did not receive an energy-balanced postexercise meal limits the interpretation of these findings'. Burns and Stensel in their letter contest this reasoning by citing a study[5] in which exercise produced a reduction in PL that was greater than that attributable to a similar energy deficit through food restriction. This, however, does not mean that doubling the food intake the night prior to the fat tolerance test may not mask or dampen the effects of exercise on PL to the degree that no significant difference from control is detected. In fact, in the cited study[5] the authors include the possibility that the enhancing effect of exercise on TAG metabolic capacity is additive to the effects of a whole-body energy deficit. We believe that the faster and/or more adequate replenishment of energy stores through a larger meal may render the body less eager to take up TAG from the next meal through a decrease in muscle lipoprotein lipase (LPL). Indeed, it has been shown that the regulation of LPL in muscle and adipose tissue is affected by nutritional status, that muscle LPL is increased after fasting and decreased after feeding and that the increased LPL activity following exercise may be blunted by post-exercise feeding.[6–9] The importance to control for food intake, when studying the effects of exercise on PL, is manifested by the fact that all relevant studies continued to replicate the meal before the fat tolerance test in the control and exercise trials after the study by Gill *et al.*[5]

The study recently completed by Burns *et al.*[10] also suggests that RE has the potential to reduce PL. This study differs in design from the previous ones[1–4], in that it included a high volume (five bouts of four sets of five exercises), very low intensity (fifteen reps at 30–40 % of 1 RM), long rest intervals (15–105 min) and a lunch between bouts. Although we acknowledge that the energy expenditure of such exercise is high, the estimate of 5·1 MJ mentioned by the authors in their letter appears excessive. Nevertheless, it is possible that the cumulative effect of multiple RE bouts on PL was abolished by the long intervals and food intake, as opposed to the studies by Petitt *et al.*[1] and our group[3], in which the subjects exercised (near) to exhaustion at each set. Previous studies that used intermittent exercise (aerobic of 1 MJ or games of approximately 3 MJ) demonstrated its superiority over continuous exercise only when a low rest:exercise ratio and no feeding during the intermittent activity were employed.[11,12]

The study by Burns *et al.*[10] further supports the notion that energy expenditure is not the only factor determining the effect of exercise on PL, given that aerobic continuous exercise of only 2·1 MJ reduces PL. Certainly, there is much more to learn regarding the influence of energy expenditure, exercise programme (intensity, rest intervals) and feeding strategy on the postprandial lipaemic response to RE.

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