High intensity interval exercise and postprandial triacylglycerol

Review Article: High intensity interval exercise and postprandial triacylglycerol.

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Abstract

This review examined if high intensity interval exercise (HIIE) reduces postprandial triacylglycerol (TAG) concentrations. Fifteen studies were identified, in which the effect of interval exercise conducted at an intensity of >65% of maximal oxygen uptake was evaluated on postprandial TAG concentrations. Analysis was divided between studies which included supramaximal exercise and those which included submaximal interval exercise. Ten studies examined the effect of a single session of low-volume HIIE including supramaximal sprints on postprandial TAG. Seven of these studies noted reductions in postprandial total TAG area under the curve the morning after exercise of between ~10%-21% compared with rest but three investigations found no significant difference in TAG concentrations. Variations in the HIIE protocol used, inter-individual variation or insufficient time post-exercise for an increase in lipoprotein lipase activity are proposed reasons for the divergent results among studies. Five studies examined the effect of high-volume submaximal interval exercise on postprandial TAG. Four of these studies were characterised by high exercise energy expenditure and effectively attenuated total postprandial TAG concentrations by ~15%-30% but one study with a lower energy expenditure found no effect on TAG. The evidence suggests that supramaximal HIIE can induce large reductions in postprandial TAG concentrations but findings are inconsistent. Submaximal interval exercise offers no TAG metabolic or time advantage over continuous aerobic exercise but could be appealing in nature to some individuals. Future research should examine if submaximal interval exercise can reduce TAG concentrations in line with more realistic and achievable exercise durations of 30 minutes per day.

Keywords: cardiovascular diseases; lipid metabolism; physical activity; postprandial period; triglyceride

Key Points

- High intensity interval exercise (HIIE) has been proposed as a time efficient method of improving metabolic health. The present article reviews the evidence for an effect of HIIE on postprandial triacylglycerol (TAG) concentrations.
- Seven studies have found single sessions of low-volume, supramaximal HIIE can reduce postprandial TAG to a similar extent as continuous aerobic exercise but the evidence is inconsistent.
- Single sessions of high-volume submaximal interval exercise can reduce postprandial TAG to a similar extent as continuous aerobic exercise but offer no time or metabolic advantage.

1. Introduction

Postprandial triacylglycerol (TAG) concentrations were first proposed as a risk factor for atherosclerosis by Zilversmit in 1979 [1]. Since Zilversmit's original hypothesis, experimental evidence has implicated elevated postprandial TAG in atherogenesis whilst prospective epidemiology studies have shown high non-fasting TAG to be an independent risk factor for cardiovascular disease in men and women [2-4]. Given that most individuals consume several meals throughout the day the postprandial state represents the usual metabolic state. This is opposed to the fasted state which usually only occurs in the first few hours of the early morning [2, 4, 5]. The macronutrient composition of meals including the total amount and type of dietary fat, amount of carbohydrate – particularly fructose – and possibly protein are also important contributors which can lead to exaggerated and extended elevations in postprandial TAG [6]. The postprandial period, therefore, represents a period of exaggerated TAG concentrations which can promote atherosclerosis by encouraging: a) an accumulation of TAG-rich lipoprotein remnants in the plasma; b) the catabolism of high-density lipoprotein and; c) formation of small, dense low-density lipoproteins which have increased susceptibility to oxidation [2, 7]. Given the importance of the postprandial period several strategies have been proposed to reduce TAG after meals. Exercise is one important strategy that has been shown to consistently induce a moderate reduction in postprandial TAG across various different populations [6, 8-11].

2. Aerobic exercise and postprandial TAG

International public health guidelines recommend that adults complete a minimum of 150 minutes of moderate intensity aerobic activity, accumulated in bouts of 10 minutes or more, each week, or alternatively, 75 minutes of more vigorous intensity aerobic activity each week [12]. Experimental studies demonstrate that performing continuous aerobic exercise can reduce postprandial TAG concentrations. However, many of these studies, though not all, have used acute aerobic exercise bouts where the duration of the exercise performed is well beyond that suggested in physical activity recommendations [6, 8-11, 13, 14]. The size of the exercise-induced energy deficit has been suggested to be the prime exercise variable determining the extent of any TAG reduction [6, 8-11, 13,

14]. The importance of the energy deficit was shown in one study where walking at different intensities reduced postprandial TAG concentrations to the same extent when the duration of exercise was manipulated to expend the same overall energy [13]. In a similar manner, walking at the same intensity but for twice the duration leads to an approximate doubling of the reduction in postprandial TAG [14]. However, the effect of aerobic exercise on postprandial TAG goes beyond producing a simple energy deficit as inducing dietary energy restriction equal to that of an exercise-induced energy deficit does not produce a similar reduction in postprandial TAG [15]. Thus, exercise appears to stimulate some factor(s) which influence either the rate of appearance or clearance of TAG-rich lipoprotein particles in the postprandial period. Moreover, it is important to note that the effects seen with aerobic exercise on postprandial TAG concentrations are substantially diminished when the energy used during exercise is replaced afterward [16, 17]. Nevertheless, meta-analyses [10, 11] and several systematic reviews [6, 8, 9] support the reduction in postprandial TAG with continuous aerobic exercise with the most recent meta-analysis reporting a significant correlation existing between the exercise energy expenditure and the effect size [11].

3. High intensity interval exercise (HIIE)

Whilst continuous aerobic exercise has a positive effect on many aspects of health, including postprandial TAG concentrations, many individuals still fail to achieve the minimal levels of activity set out in guidelines with ‘lack of time’ cited as the most common barrier for regular exercise participation [18, 19]. For example, in the U.K. ~60% of men and ~70% of women did not meet physical activity recommendations with the most common barriers identified as ‘work commitment’ (45% men and 34% women) and ‘a lack of leisure time’ (38% men and 37% women) [19]. Thus, to promote health in a shorter time HIIE has been proposed as a viable alternative to continuous aerobic activity [20]. This type of exercise has existed for some time [21,22] but gained more prominence in 2005 as a potential replacement for endurance exercise training when 2 weeks of HIIE training was shown to increase muscle oxidative capacity and double endurance capacity in recreationally active young individuals [23]. Accumulating evidence has since shown that HIIE induces multiple physiological adaptations similar to traditional endurance training [24-34]. The lower total exercise

volume and training time involved has led to the suggestion that HIIE training represents a valuable alternative to the current aerobic exercise guidelines which could encourage physical activity participation and reduce the risk of chronic diseases [20].

Early studies using HIIE training sessions were characterised by low work volume. The initial protocol involved four to six 30 second all-out sprint efforts on a cycle ergometer (Wingate tests) per session with recovery periods of 4 minutes between each sprint [23]. However, Wingate tests require specialised cycle ergometers and the nature of the exercise sessions means that participants have to be highly motivated casting doubt on the applicability of this type of training in unfit populations [20]. Subsequently, variations on the original protocol emerged. These include eight to twelve 1 minute intervals at an intensity corresponding to ~100% of maximal oxygen uptake [30], extremely short duration sprints of between 6 and 15 seconds [35-38], a single maximal extended sprint [39], or the use of sprint running [40,41] as an alternative to cycling. Another approach has been to investigate the effect of interval exercise sessions conducted at submaximal intensities [42-47]. It is debatable whether interval exercise conducted at <100% of maximal oxygen uptake should be compared with the original supramaximal protocol [23] as submaximal interval exercise sessions involve a much higher work volume and longer duration exercise sessions. Nevertheless, current US Physical Activity Guidelines classify activity of 65-85% of maximal oxygen uptake as hard and >85% as very hard [48] suggesting that interval sessions conducted at these loads should be considered high intensity. Moreover, physiological adaptations benefitting health still occur with submaximal interval sessions [42-47]. Importantly, both supramaximal and submaximal protocols have been used as successful interventions for improving health outcomes or indicators in moderately overweight and obese individuals [31, 32], older adults [35], paediatric populations [32, 38, 41, 49], individuals with metabolic syndrome [50] and individuals with established coronary artery disease [42].

4. High intensity interval exercise and postprandial TAG

Given the substantial effect of continuous aerobic exercise on postprandial TAG concentrations and the extent of the physiological adaptations associated with HIIE, it is not surprising that investigations

have now examined how HIIE effects postprandial TAG concentrations. The aims of the present review were: (i) to discuss the evidence for an effect of HIIE on postprandial TAG concentrations, (ii) to evaluate the effectiveness of HIIE versus continuous aerobic exercise for lowering postprandial TAG concentrations and (iii) to discuss the mechanisms responsible for HIIE induced reductions in postprandial TAG. A search was made in PubMed using the following key words in combination: "postprandial triacylglycerol AND interval exercise", "postprandial triacylglycerol AND high intensity exercise" or "postprandial triacylglycerol AND sprint exercise". The same search was made using "postprandial triglyceride", "postprandial lipemia", or "postprandial lipaemia" as alternatives to "postprandial triacylglycerol". The search incorporated any article published in English and was cross-checked and supplemented using the authors' personal libraries. Criteria for inclusion in this review were: 1) the dependent variable was postprandial TAG concentration in humans, 2) studies were designed to evaluate the effect of interval exercise at an intensity >65% of maximal oxygen uptake. Criteria for exclusion of a study were: 1) continuous aerobic exercise only, 2) resistance exercise, or 3) protocols which examined the issue of accumulating exercise on postprandial lipaemia which has been reviewed previously [51]. Definition of a protocol as one examining accumulation was where the rest period between bouts of exercise was ≥10 minutes. None of the studies in the present review involved rest periods between exercise bouts of >5 minutes, although the recovery periods differed in nature with some using only passive recovery whilst others included low-intensity active recovery.

Fifteen studies met the inclusion criteria from fifty-nine studies retrieved. For our analysis we chose to divide studies into two types: (i) those involving low-volume HIIE sessions at supramaximal intensities (≥100% of maximal oxygen uptake) and (ii) high-volume interval exercise protocols conducted at submaximal intensities (65-<100% of maximal oxygen uptake). This is because the latter typically involve greater exercise volumes of longer duration at a submaximal level which detracts from the original time saving premise and supramaximal intensity associated with early HIIE training.

5. Effects of low-volume supramaximal HIIE on postprandial TAG

Ten studies were identified which examined the effect of low-volume HIIE using protocols >100% of maximal oxygen uptake on postprandial TAG [36-38, 41, 50, 52-56]. Table 1 provides a summary of the study designs and findings. The first published work was conducted by Freese and colleagues [52] and followed the original HIIE protocol previously described of four Wingate tests interspersed with 4 minutes of recovery [23]. Total postprandial TAG responses to a single high fat test meal were 21% lower the morning after HIIE compared with a rest day. Interestingly, also included was a condition where the energy expended in HIIE was replaced by a post-exercise meal, with the premise being that any exercise-induced reduction in TAG is due to the energy deficit of exercise. As noted previously, energy expenditure is a key factor in postprandial TAG reductions following aerobic exercise [6, 8-11, 13, 14] and energy replacement significantly mitigates reductions in TAG concentrations with aerobic exercise [16, 17]. Nevertheless, Freese and colleagues reported that a 10% reduction in total TAG concentrations persisted after HIIE despite energy replacement compared with the resting condition [52]. However, the extent of the mitigation meant that TAG concentrations after energy deficit were still significantly lower than when in energy replacement reinforcing the importance of this variable on exercise-induced reductions of postprandial TAG.

Two other important aspects of this seminal work by Freese and colleagues [52] are noted. Firstly, postprandial TAG responses were measured over a 3 hour period. Most studies examining aerobic exercise have used longer postprandial protocols; typically 6 hours [9]. Justification for a shortened protocol as a valid assessment of the overall TAG response was provided by a study where the postprandial TAG response over 4 hours was shown to be highly predictive of the response over 8 hours in five lean and four obese subjects [57]. However, subsequent work by another group in a larger cohort reported that whilst the total and incremental TAG 4 hour area resulted in a moderate to high prediction of the 6 hour area, further reduction to 3 hours resulted in less predictability [58]. The second aspect of note was that, despite significantly reduced postprandial TAG concentrations, the study failed to address the question of whether HIIE was an equally effective or more effective strategy for reducing TAG concentrations than aerobic exercise. For HIIE to be considered for

224 inclusion in physical activity recommendations then changes in risk markers for health with this type
225 of exercise should be compared in relation to those produced by continuous aerobic exercise whilst
226 taking into consideration any time advantage. These criticisms of the original paper by Freese were
227 addressed by another group of researchers. Gabriel and colleagues examined 7 hour postprandial
228 TAG responses to two high fat test meals, given 3 hours apart, in 9 young healthy males the morning
229 after five Wingate tests, 30 minutes of brisk walking or a rest day [53]. An ~18% borderline
230 reduction in total TAG (P=0.056) and a significant ~34% reduction in the TAG incremental area
231 under the curve (HIIE: 6.42 (2.24) $mmol \cdot l^{-1} \cdot 7h^{-1}$ *vs.* Rest: 9.68 (4.77) $mmol \cdot l^{-1} \cdot 7h^{-1}$, $P<0.05$) occurred
232 only after HIIE compared with rest, demonstrating it as a viable alternative to aerobic exercise whilst
233 addressing the criticism of a shorter postprandial assessment period [53]. A follow-up study from the
234 same group using the same HIIE protocol showed similar findings, with a 21% reduction in the total
235 TAG area under the curve in response to two high fat test meals in comparison with rest the day after
236 exercise but the effect did not last for two days post-exercise [54].
237
238 Important successful modifications to the Wingate protocol were made in three studies. Compared
239 with rest, Thackray and colleagues found an ~10% decrease in postprandial capillary TAG in
240 response to a single high fat test meal the morning after ten 1 minute running intervals at maximal
241 aerobic speed in healthy boys aged 11-12 years [41]. The authors suggested that HIIE should be
242 investigated as an exercise strategy to improve children's health as interspersing moderate exercise
243 with high intensity work periods is associated with greater perceived exercise enjoyment in youth than
244 continuous moderate intensity exercise alone [59]. In two recent investigations, the influence of very
245 short duration sprints on postprandial TAG was evaluated [38, 56]. In the first of these, sixty 8
246 second sprints, interspersed with 12 seconds of moderate cycling, reduced TAG concentrations by
247 ~13% assessed over 4 hours the next morning compared with a resting control day in 12 sedentary
248 young women [56]. Importantly, the total exercise session lasted 20 minutes, similar to the time
249 needed for four to six Wingate tests with 4 minute recovery periods, and all women were reported to
250 complete the exercise protocol even though they were sedentary [56]. In the second study, forty 6
251 second maximal sprints were found to reduce postprandial TAG concentrations by ~13% the next day

252 in nine adolescent boys compared with a rest day prior to a postprandial TAG assessment [38]. One
253 noteworthy aspect of this study was the high dropout rate as 5 boys did not complete due to what was
254 described as, 'a failure to tolerate the exercise' [38]. Whilst other researchers have raised concerns of
255 issues of motivation and safety surrounding low-volume supramaximal HIIE [20], in the nine studies
256 described here, only this one [38] reported any dropouts from the HIIE protocol. Moreover, this
257 inability to complete the HIIE protocol contrasts starkly with the earlier Thackray study described in
258 adolescents [41] and the similar protocol used in sedentary women [56].
259
260 Most recently, the acute and chronic effects of HIIE were examined in 45 women with metabolic
261 syndrome [50]. The effect of a single bout of HIIE and 6 weeks of HIIE training was evaluated in 22
262 of the women whilst 23 women were assigned to a non-exercise control group. All HIIE sessions
263 involved 30 second maximal sprints with 4-8 sprints per session. Compared to their baseline
264 evaluation of postprandial TAG a single session of HIIE reduced the total TAG response by 13.1%
265 and after 6 weeks by 9.7%, whilst there was no significant change in postprandial TAG in the control
266 group over the same time. Given that all other studies evaluating the effect of low-volume
267 supramaximal HIIE on postprandial TAG have examined either healthy adolescents or young, healthy
268 adults this work represents an important step by addressing individuals with lipid and metabolic
269 disturbances who are at an increased risk of cardiovascular disease. Interestingly, the findings
270 suggests that, as with continuous aerobic exercise [60], much of the benefit of HIIE on TAG
271 concentrations is from a last bout effect as 6 weeks of training failed to magnify the effect of the
272 single session of HIIE [50].
273
274 Not all studies have noted significant mean reductions in postprandial TAG the morning after HIIE
275 sessions [36, 37, 55]. Tan and colleagues reported no difference in the TAG response to a single high
276 fat meal in 9 healthy young individuals the morning after four Wingate tests in comparison with a
277 control trial [55]. Of note, in the same study, 20 minutes of cycling at 70% of maximal oxygen
278 uptake also failed to mitigate postprandial TAG. The authors suggested wide inter-individual
279 responses to the interventions as a possible factor for the failure of either exercise session to reduce

TAG concentrations [55]. However, 30 minutes of brisk walking also failed to impact TAG responses in the study by Gabriel and colleagues whilst HIIE was able to induce a substantial reduction [53]. Two other studies have also failed to find changes in postprandial TAG with HIIE [36, 37]. The first found no effect of twenty 6 second maximal sprints on postprandial TAG metabolism when a single high fat test meal was consumed 18.5 hours later [36]. The second found no effect of either five 60 second sprints at 100% of maximal aerobic capacity or ten 15 second sprints at 200% of maximal aerobic capacity on postprandial TAG responses to a test meal given 1 hour later [37].

A variety of factors should be considered in studies where no effect of HIIE on postprandial TAG was observed [36, 37, 55]. Sample sizes were relatively small, ranging from 9-15 participants per study. However, similar sample sizes were used in six of the studies where TAG concentrations were reduced after supramaximal HIIE [38, 41, 52-54, 56] and studies where aerobic exercise attenuated postprandial TAG concentrations have also used similar numbers [6, 8-11, 13-15]. Inter-individual variance could account for the negative findings and the issue was highlighted in the study by Thackray and colleagues where one-third of participants - 5 out of 15 children - had an increase or no change in TAG concentrations after HIIE compared with control [41]. Apolipoprotein E genotype may partly explain the inter-individual variance as one study found continuous moderate aerobic exercise was effective in attenuating postprandial TAG only in individuals who carried the ε2 or ε3 allele but had no effect on those with the ε4 allele [61]. The sprint protocol in two of the studies where no effect was seen on postprandial TAG [36, 37] was modified from the original Wingate protocol but the third study did employ Wingate tests without any effect on TAG concentrations [55]. Moreover, one investigation used both longer and shorter sprints and saw no effect of either on TAG concentrations [37] which contrasts with the significant ~13% reductions in TAG after sixty 8 second sprints [56] or forty 6 second sprints [38] described previously. The test meal is another potential source of variance. High fat test meal responses are reproducible [57, 62] but unlike glucose tolerance tests there is no standardised version despite a recent expert panel recommendation [63]. Nevertheless, all the studies reported gave at least >0.8g of fat/kg of body mass, which is regarded as a high rather than moderate fat load [6, 11, 63]. Time of meal consumption is another factor. One

systematic review noted that in more than 40 studies of aerobic or resistance exercise, where a 12-18 hour window was used between exercise and consumption of the fat meal, only 3 failed to find an exercise-induced decrease in postprandial TAG [6]. This compared with 6 out of 15 studies which found no effect on postprandial TAG when only a 3 hour window was used [6]. Only a 1 hour break between exercise and meal consumption was used in the study by Canale and colleagues [37] but a ~14 and ~18.5 hour window in the other two studies [36, 55] that failed to find a difference with HIIE. Another possibility relates to activity of the enzyme lipoprotein lipase (LPL) which is a suggested likely mechanism to explain decreased postprandial TAG after HIIE sessions [52-54]. Activity of LPL has been noted to peak ≥8 hours after a bout of aerobic exercise [64]. If the enzyme activity is increased in the same way after HIIE it could explain why a shorter interval between exercise and the test meal was insufficient to reduce TAG in the study by Canale and colleagues [37] but not the other two investigations [36, 55].

In summary, seven studies have found significant postprandial total TAG reductions of 10-21% after HIIE but comparable findings have not been seen in three recent publications questioning the consistency of HIIE as a mode of exercise for TAG reductions. No single explanation is currently satisfactory to explain the division among studies. Moreover, whilst total HIIE work time in all studies reported ranged from 2-10 minutes the actual total protocol length in most, including rest or low-intensity exercise time, was nearer 25 minutes, not including warm-up or cool-down. Thus, the time saving factor highlighted as a major benefit of this type of exercise is not visible if the exercise needs to be performed five or more times per week for regular benefit [50, 54] to postprandial TAG. The recent study in women with metabolic syndrome has provided evidence that HIIE effects postprandial TAG in individuals at an increased risk of cardiovascular disease. Future research needs to focus more on these individuals who benefit the most from reductions in postprandial TAG achieved with HIIE. The plausibility of using a single extended sprint - as has been done by others in overweight and obese men to examine fat oxidation and insulin sensitivity [39] - might provide a time efficient method which is attractive to these individuals with elevated postprandial TAG.

6. Effects of high-volume submaximal interval exercise on postprandial TAG

Five studies have examined how high volume submaximal interval exercise sessions influence postprandial TAG concentrations (Table 2) [43-47]. An investigation by Ferreira and colleagues examined expending 500 kcal in running in 3 minute intervals at 115% of the anaerobic threshold with 1.5 minutes recovery [43]. They found a 15% decrease in total postprandial TAG over 4 hours in response to a single high fat meal given 1 hour post-exercise compared with a resting control trial. The extent of the decrease was similar to that produced by continuous running at 85% of the anaerobic threshold (an 18% decrease). In a later study, cycling for 2 minute intervals at 90% of peak oxygen uptake, with recovery periods of 2 minutes at 25% of peak oxygen uptake, decreased postprandial total and incremental TAG concentrations to a large mixed meal by~30% and ~45%, respectively, compared with rest [44]. Moreover, cycling for 1 hour at 50% of peak oxygen uptake, with the same energy expenditure (~660 kcal) as the interval session, only produced an ~25% significant decrease in incremental TAG concentrations; significantly lower than the decrease found with intermittent cycling [44]. These two studies demonstrate that submaximal interval exercise sessions can diminish postprandial TAG to an extent similar to, and possibly greater than, aerobic exercise when energy expenditure is similar. However, one aspect of both studies to highlight is that the total exercise volume (40-42 minutes) is higher than the currently accepted minimum which approximates to 30 minutes over 5 days per week [12]. From this perspective, an early study found that four, 4 minute sprints at 85-95% of maximal heart rate or a work-matched continuous exercise protocol 60-70% of maximal heart rate had no effect on postprandial TAG the next day [45]. Thus, as with aerobic exercise, energy expenditure appears likely to be an important variable determining exercise-induced reductions in TAG during submaximal interval exercise. Rather than any TAG metabolic or time advantage this type of exercise might instead appeal to those individuals who enjoy intermittent work-outs of higher exercise volume.

Barrett and colleagues examined the effect of a protocol designed to imitate the demands of field sports on postprandial TAG. The protocol consisted of four 15 minute blocks, separated by 3 minutes rest, with each block divided into a continuous period of walking, jogging, cruise running, and

sprinting in order to simulate games activity in 12 young males [46]. The cruise section involved running at 70% of maximal oxygen uptake whilst the 15 metre sprint was maximal. In comparison with rest, total TAG concentrations were reduced over 6 hours by 25% and the extent of this reduction was similar to that of continuous walking (19% reduction) at 60% of maximal oxygen uptake with an average energy expenditure of 3.1 MJ in the same subjects [46]. Subsequent investigation in healthy adolescent boys using the same protocol showed similar findings with a 26% reduction after the simulated games activity protocol [47]. These two studies demonstrate that engaging in field and racquet sports or other activities characterised by intermittent periods of high and low intensity work can produce similar effects on postprandial TAG as more traditional continuous aerobic exercise. Such activities have been shown to appeal to certain groups of adults and youth [65, 66].

7. Mechanisms for TAG reduction with high intensity interval exercise

Most TAG is carried in intestinal derived chylomicrons and hepatically-derived very low density lipoproteins (VLDL) and the concentration of TAG in the circulation reflects a balance in the rate of appearance and clearance of these two particles [67]. The primary proposed mechanism for increased TAG clearance with aerobic exercise is an increase in the activity of the enzyme LPL which is expressed on the capillary endothelium of skeletal muscle and has been shown to correlate with changes in TAG [68]. Heavy or prolonged aerobic exercise bouts can substantially increase post-heparin plasma LPL activity - an indicator of whole body LPL activity from all tissues [69]. The time course for changes in LPL with exercise is delayed, however, and increases in LPL mRNA levels are reported to peak 4 hours post-exercise whilst LPL mass peaks ≥8 hours after exercise, with both returning to baseline within 24 hours [64]. These facts led to the hypothesis that increased LPL activity is the likely mechanism for reductions in postprandial TAG after HIIE [52, 53]. Indirect support for this proposal came from Gabriel and colleagues who found that HIIE had no effect on plasma levels of β-hydroxybutyrate, a marker of hepatic fatty acid oxidation indicating altered VLDL synthesis [53]. Subsequently, the same group found an increase in LPL dependent TAG breakdown the morning after HIIE, compared with a control trial, which was associated with the reduction in total plasma TAG [54]. If increased LPL activity is responsible for TAG reductions following HIIE it

would be surprising. The energy expenditure of low-volume HIIE is well below any threshold associated with increases in LPL activity after aerobic exercise [69]. However, one suggestion is that LPL activation is fibre specific with increases in activity occurring because HIIE recruits fast twitch fibres [54]. Some support for this comes from observations in rats where LPL mRNA levels and mass and LPL enzyme activity were all increased in white but not red hind-limb skeletal muscles after short-term run training [70]. Moreover, as noted early on, the effect of aerobic exercise on postprandial TAG has been shown to be greater than that of a simple energy deficit [15] suggesting that exercise stimulates some factor(s) which influences either the rate of appearance or clearance of TAG-rich lipoprotein particles in the postprandial period. If LPL is that factor, it is possible that HIIE has a greater effect on its activity than aerobic exercise of lower intensity and this would help explain why replacement of the post-exercise energy deficit did not completely mitigate postprandial TAG concentrations in the study by Freese and colleagues [52]. Future studies should investigate this mechanism further using a direct measurement of TAG clearance, such as arterio-venous TAG differences across previously exercised muscle.

Studies of moderate intensity aerobic activity suggest that reduced hepatic VLDL secretion may be a more important method in postprandial TAG reduction than increased LPL activity and/or mass [67]. However, evidence for a decrease in the appearance of hepatically-derived VLDL as a mechanism for TAG-reduction with HIIE is limited. An increased fasting and steeper postprandial rise in plasma β-hydroxybutyrate was seen after a single session of high volume submaximal interval exercise whilst at the same time total and incremental postprandial TAG were attenuated by ~30% and ~45%, respectively, compared with a no-exercise control trial [44]. However, as previously noted, another study found no change in β-hydroxybutyrate after low volume HIIE despite postprandial TAG attenuation [53]. Another investigation used stable isotopes to calculate changes in fasting, but not postprandial, VLDL-TAG secretion and clearance rates in 8 healthy sedentary young men after a single bout of HIIE at intensities of 60% and 90% of peak oxygen uptake taken for 32 minutes. They found that fasting VLDL-TAG was reduced 14 hours post-exercise due to an ~21% increase in clearance rate and no change in VLDL-secretion which would suggest increased skeletal muscle LPL

mass and/or activity post-exercise [71]. In summary, most evidence suggests HIIE elicits increased clearance of postprandial TAG via increased skeletal muscle LPL activity and/or mass at this time. However, support for this hypothesis comes from single sessions of HIIE and there has been little direct examination of VLDL secretion and clearance in the postprandial state.

8. Conclusions

In conclusion, seven studies have found that a single session of low-volume, supramaximal HIIE induced large reductions in postprandial TAG concentrations but three recent works have failed to consistently replicate this. Differences in exercise protocols, inter-individual participant variation, or insufficient time post-exercise for increases in LPL activity may be reasons for the divergent results. Thus, whilst the efficacy of low-volume HIIE to attenuate postprandial TAG has been shown, the variability suggests that a prudent approach should be taken when recommending this type of exercise as an alternative strategy to continuous aerobic exercise in individuals who need to reduce their TAG concentrations. Given there is only one study in individuals with high TAG concentrations [50], future research should examine the potential of supramaximal HIIE to mitigate postprandial TAG in individuals with both monogenic and polygenic hypertriglyceridemia. This would help to diversify and explain individual differences in the TAG lowering response to this type of exercise. High volume submaximal interval exercise is effective in reducing postprandial TAG but it appears to offer no benefit over continuous aerobic exercise in terms of TAG metabolic or time advantage. Future research should examine if submaximal interval exercise can reduce TAG concentrations in line with more realistic and socially acceptable durations of exercise of 30 minutes per day.

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References

1. Zilversmit DB. Atherogenesis: a postprandial phenomenon. Circulation. 1979; 60:473-485.
2. Nordestgaard BG, Varbo A. Triglycerides and cardiovascular disease. Lancet. 2014; 384:626-635.
3. Bansal S, Buring JE, Rifai N, et al. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA. 2007; 298:309-316.
4. Nordestgaard BG, Benn M, Schnohr P, et al. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA. 2007; 298:299-308.
5. Hegele RA, Ginsberg HN, Chapman MJ, et al.; European Atherosclerosis Society Consensus Panel. The polygenic nature of hypertriglyceridaemia: implications for definition, diagnosis, and management. Lancet Diabetes Endocrinol. 2014; 2:655-666.
6. Peddie MC, Rehrer NJ, Perry TL. Physical activity and postprandial lipidemia: are energy expenditure and lipoprotein lipase activity the real modulators of the positive effect? Prog Lipid Res. 2012; 51:11-22.
7. Cohn JS. Postprandial lipemia: emerging evidence for atherogenicity of remnant lipoproteins. Can J Cardiol. 1998; 14(Suppl B):18B-27B.
8. Katsanos CS. Prescribing aerobic exercise for the regulation of postprandial lipid metabolism: current research and recommendations. Sports Med. 2006; 36:547-560.
9. Maraki MI, Sidossis LS. The latest on the effect of prior exercise on postprandial lipaemia. Sports Med. 2013; 43:463-481.
10. Petitt DS, Cureton KJ. Effects of prior exercise on postprandial lipemia: a quantitative review. Metabolism. 2003; 52:418-424.
11. Freese EC, Gist NH, Cureton KJ. Effect of prior exercise on postprandial lipemia: an updated quantitative review. J Appl Physiol. 2014; 116:67-75.

12. World Health Organization. Global Recommendations on Physical Activity for Health. World Health Organization 2010. ISBN: 978 92 4 159 997 9.
13. Tsetsonis NV, Hardman AE. Reduction in postprandial lipemia after walking: influence of exercise intensity. Med Sci Sports Exerc. 1996; 28:1235-1242.
14. Gill JM, Herd SL, Hardman AE. Moderate exercise and post-prandial metabolism: issues of dose-response. J Sports Sci. 2002; 20:961-967.
15. Gill JM, Hardman AE. Postprandial lipemia: effects of exercise and restriction of energy intake compared. Am J Clin Nutr. 2000; 71:465-471.
16. Burton FL, Malkova D, Caslake MJ, et al. Energy replacement attenuates the effects of prior moderate exercise on postprandial metabolism in overweight/obese men. Int J Obes (Lond). 2008; 32:481-489.
17. Harrison M, O'Gorman DJ, McCaffrey N, et al. Influence of acute exercise with and without carbohydrate replacement on postprandial lipid metabolism. J Appl Physiol. 2009; 106:943-949.
18. Ministry of Health. National Health Survey 2010 Singapore. Epidemiology and Disease Control Division, Ministry of Health, Singapore 2010. ISBN:978-981-08-8540-3.
19. Townsend N, Bhatnagar P, Wickramasinghe K, et al. Physical activity statistics 2012. British Heart Foundation 2012: London.
20. Gibala MJ, Little JP. Just HIT it! A time-efficient exercise strategy to improve muscle insulin sensitivity. J Physiol. 2010; 588:3341-3342.
21. McKenna MJ, Schmidt TA, Hargreaves M, et al. Sprint training increases human skeletal muscle Na(+)-K(+)-ATPase concentration and improves K+ regulation. J Appl Physiol. 1993; 75:173-180.
22. MacDougall JD, Hicks AL, MacDonald JR, et al. Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol. 1998; 84:2138-2142.
23. Burgomaster KA, Hughes SC, Heigenhauser GJ, et al. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. J Appl Physiol. 2005; 98:1985-1990.

24. Gist NH, Fedewa MV, Dishman RK, et al. Sprint interval training effects on aerobic capacity: a systematic review and meta-analysis. Sports Med. 2014; 44:269-279.
25. Weston M, Taylor KL, Batterham AM, et al. Effects of low-volume high-intensity interval training (HIT) on fitness in adults: a meta-analysis of controlled and non-controlled trials. Sports Med. 2014; 44:1005-1017.
26. Gibala MJ, Little JP, van Essen M, et al. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. J Physiol. 2006; 575:901-911.
27. Burgomaster KA, Howarth KR, Phillips SM, et al. Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J Physiol. 2008; 586:151-160.
28. Rakobowchuk M, Tanguay S, Burgomaster KA, et al. Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. Am J Physiol Regul Integr Comp Physiol. 2008; 295:R236-R242.
29. Babraj JA, Vollaard NB, Keast C, et al. Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. BMC Endocr Disord. 2009; 9:3.
30. Little JP, Safdar A, Wilkin GP, et al. A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: potential mechanisms. J Physiol. 2010; 588:1011-1022.
31. Whyte LJ, Gill JM, Cathcart AJ. Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. Metabolism. 2010; 59:1421-1428.
32. Corte de Araujo AC, Roschel H, Picanço AR, et al. Similar health benefits of endurance and high-intensity interval training in obese children. PLoS One. 2012; 7(8):e42747.
33. Cocks M, Shaw CS, Shepherd SO, et al. Sprint interval and endurance training are equally effective in increasing muscle microvascular density and eNOS content in sedentary males. J Physiol. 2013; 591:641-656.

34. Shepherd SO, Cocks M, Tipton KD, et al. Sprint interval and traditional endurance training increase net intramuscular triglyceride breakdown and expression of perilipin 2 and 5. J Physiol. 2013; 591:657-675.
35. Adamson SB, Lorimer R, Cobley JN, et al. Extremely short-duration high-intensity training substantially improves the physical function and self-reported health status of elderly adults. J Am Geriatr Soc. 2014; 62:1380-1381.
36. Allen E, Gray P, Kollias-Pearson A, et al. The effect of short-duration sprint interval exercise on plasma postprandial triacylglycerol levels in young men. J Sports Sci. 2014; 32:911-916.
37. Canale RE, Farney TM, McCarthy CG, et al. Influence of acute exercise of varying intensity and duration on postprandial oxidative stress. Eur J Appl Physiol. 2014; 114:1913-1924.
38. Sedgwick MJ, Morris JG, Nevill ME, et al. Effect of repeated sprints on postprandial endothelial function and triacylglycerol concentrations in adolescent boys. J Sports Sci. 2014 Oct 30:1-11. [Epub ahead of print]
39. Whyte LJ, Ferguson C, Wilson J, et al. Effects of single bout of very high-intensity exercise on metabolic health biomarkers in overweight/obese sedentary men. Metabolism. 2013; 62:212-219.
40. Macpherson RE, Hazell TJ, Olver TD, et al. Run sprint interval training improves aerobic performance but not maximal cardiac output. Med Sci Sports Exerc. 2011; 43:115-122.
41. Thackray AE, Barrett LA, Tolfrey K. Acute high-intensity interval running reduces postprandial lipemia in boys. Med Sci Sports Exerc. 2013; 45:1277-1284.
42. Currie KD, Dubberley JB, McKelvie RS, et al. Low-volume, high-intensity interval training in patients with CAD. Med Sci Sports Exerc. 2013; 45:1436-1442.
43. Ferreira AP, Ferreira CB, Souza VC, et al. The influence of intense intermittent versus moderate continuous exercise on postprandial lipemia. Clinics (Sao Paulo). 2011; 66:535-541.
44. Trombold JR, Christmas KM, Machin DR, et al. Acute high-intensity endurance exercise is more effective than moderate-intensity exercise for attenuation of postprandial triglyceride elevation. J Appl Physiol. 2013; 114:792-800.

45. Tyldum GA, Schjerve IE, Tjønna AE, et al. Endothelial dysfunction induced by post-prandial lipemia: complete protection afforded by high-intensity aerobic interval exercise. J Am Coll Cardiol. 2009; 53:200-206.
46. Barrett LA, Morris JG, Stensel DJ, et al. Effects of intermittent games activity on postprandial lipemia in young adults. Med Sci Sports Exerc. 2006; 38:1282-1287.
47. Barrett LA, Morris JG, Stensel DJ, et al. Exercise and postprandial plasma triacylglycerol concentrations in healthy adolescent boys. Med Sci Sports Exerc. 2007; 39:116-122.
48. U.S. Department of Health and Human Services. Physical Activity Guidelines Advisory Committee Report, 2008. U.S. Department of Health and Human Services 2008. Washington DC, U.S.
49. Burns SF, Oo HH, Tran AT. Effect of sprint interval exercise on postexercise metabolism and blood pressure in adolescents. Int J Sport Nutr Exerc Metab. 2012; 22:47-54.
50. Freese EC, Gist NH, Acitelli RM, et al. Acute and chronic effects of sprint interval exercise on postprandial lipemia in women at-risk for the metabolic syndrome. J Appl Physiol. 2015 Jan 15:jap.00380.2014. doi: 10.1152/japplphysiol.00380.2014. [Epub ahead of print]
51. Miyashita M, Burns SF, Stensel DJ. An update on accumulating exercise and postprandial lipaemia: translating theory into practice. J Prev Med Public Health. 2013; 46 Suppl 1:S3-S11.
52. Freese EC, Levine AS, Chapman DP, et al. Effects of acute sprint interval cycling and energy replacement on postprandial lipemia. J Appl Physiol. 2011; 111:1584-1589.
53. Gabriel B, Ratkevicius A, Gray P, et al. High-intensity exercise attenuates postprandial lipaemia and markers of oxidative stress. Clin Sci (Lond). 2012; 123:313-321.
54. Gabriel BM, Pugh J, Pruneta-Deloche V, et al. The effect of high intensity interval exercise on postprandial triacylglycerol and leukocyte activation--monitored for 48 h post exercise. PLoS One. 2013; 8:e82669.
55. Tan MS, Mok A, Yap MC, et al. Effect of sprint interval versus continuous cycling on postprandial lipaemia. J Sports Sci. 2013; 31:989-995.

584 56. Tan M, Chan Moy Fat R, Boutcher YN, et al. Effect of high-intensity intermittent exercise on
585 postprandial plasma triacylglycerol in sedentary young women. Int J Sport Nutr Exerc Metab.
586 2014; 24:110-118.
587 57. Weiss EP, Fields DA, Mittendorfer B, et al. Reproducibility of postprandial lipemia tests and
588 validity of an abbreviated 4-hour test. Metabolism. 2008; 57:1479-1485.
589 58. Maraki M, Aggelopoulou N, Christodoulou N, et al. Validity of abbreviated oral fat tolerance
590 tests for assessing postprandial lipemia. Clin Nutr. 2011; 30:852-857.
591 59. Crisp NA, Fournier PA, Licari MK, et al. Optimising sprint interval exercise to maximise
592 energy expenditure and enjoyment in overweight boys. Appl Physiol Nutr Metab. 2012;
593 37:1222-1231.
594 60. Herd SL, Hardman AE, Boobis LH, et al. The effect of 13 weeks of running training followed
595 by 9 d of detraining on postprandial lipaemia. Br J Nutr. 1998; 80:57-66.
596 61. Ferreira AP, Ferreira CB, Brito CJ, et al. The effect of aerobic exercise intensity on
597 attenuation of postprandial lipemia is dependent on apolipoprotein E genotype.
598 Atherosclerosis. 2013; 229:139-144.
599 62. Gill JM, Malkova D, Hardman AE. Reproducibility of an oral fat tolerance test is influenced
600 by phase of menstrual cycle. Horm Metab Res. 2005; 37:336-341.
601 63. Kolovou GD, Mikhailidis DP, Kovar J, et al. Assessment and clinical relevance of non-fasting
602 and postprandial triglycerides: an expert panel statement. Curr Vasc Pharmacol. 2011; 9:258-
603 270.
604 64. Seip RL, Mair K, Cole TG, et al. Induction of human skeletal muscle lipoprotein lipase gene
605 expression by short-term exercise is transient. Am J Physiol. 1997; 272:E255-E261.
606 65. Hunt K, Wyke S, Gray CM, et al. A gender-sensitised weight loss and healthy living
607 programme for overweight and obese men delivered by Scottish Premier League football
608 clubs (FFIT): a pragmatic randomised controlled trial. Lancet. 2014; 383:1211-1221.
609 66. Ratel S, Lazaar N, Dore E, et al. High-intensity intermittent activities at school: controversies
610 and facts. J Sports Med Phys Fitness. 2004; 44:272-280.

67. Malkova D, Gill JM. Effects of exercise on postprandial lipoprotein metabolism. Future Lipidol. 2006; 1:743-755.
68. Seip RL, Angelopoulos TJ, Semenkovich CF. Exercise induces human lipoprotein lipase gene expression in skeletal muscle but not adipose tissue. Am J Physiol. 1995; 268:E229-E236.
69. Ferguson MA, Alderson NL, Trost SG, et al. Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. J Appl Physiol. 1998; 85:1169-1174.
70. Hamilton MT, Etienne J, McClure WC, et al. Role of local contractile activity and muscle fiber type on LPL regulation during exercise. Am J Physiol. 1998; 275:E1016-E1022.
71. Bellou E, Magkos F, Kouka T, et al. Effect of high-intensity interval exercise on basal triglyceride metabolism in non-obese men. Appl Physiol Nutr Metab. 2013; 38:823-829.

Table 1. Studies examining the effect of low-volume supramaximal high intensity interval exercise on postprandial triacylglycerol.

Reference	n Sex	Age (y)	Study design	Test meal energy and fat content	Time from exercise cessation to test meal consumption (h)	Main findings
Allen et al 2014 [36]	15M	25 (4)	i. Twenty 6-s maximal cycle sprints ii. Rest (control)	Standardised: 5.3 MJ 64% energy fat	18.5	TAG AUC: i. Sprints: 7.26 (2.49) $mmol \cdot l^{-1} \cdot 4h^{-1}$ ii. Rest: 7.67 (2.37) $mmol \cdot l^{-1} \cdot 4h^{-1}$ P>0.05 between trials
Canale et al 2014 [37]	12M	23.7 (1.1)	i. Five 60-s cycle sprints at 100% of maximal capacity ii. Ten 15-s cycle sprints at 200% of maximal capacity iii. 60-min continuous cycling at 70% of HRR	51 kJ/kg bm 0.8 g fat/kg bm	1.0	No interaction or condition effect noted among trials, both P>0.05

			iv. Rest (control)			
Sedgwick et al 2014 [38]	9M	13.1 (0.6)	i. Forty 6-s maximal cycle sprints ii. Rest (control)	Breakfast: 93 kJ/kg bm 1.5 g fat/ kg bm Lunch: 85 kJ/kg bm 1.1 g fat/ kg bm	16	TAG AUC: Sprints: 8.65 (0.97) $mmol \cdot l^{-1} \cdot 6.5h^{-1}$ Rest: 9.92 (1.16) $mmol \cdot l^{-1} \cdot 6.5h^{-1}$ P=0.023 between trials Effect size = 0.40
Thackray et al 2013 [41]	15M	11.8 (0.4)	i. Ten 1-min running intervals at 100% MAS ii. Rest (control)	Breakfast: 93 kJ/kg bm 1.5 g fat/ kg bm Lunch: 86 kJ/kg bm 1.1 g fat/ kg bm	15.5	TAG AUC: Sprints: 5.2 (1.1) $mmol \cdot l^{-1} \cdot 6.5h^{-1}$ Rest: 5.8 (1.5) $mmol \cdot l^{-1} \cdot 6.5h^{-1}$ Effect size = 0.50
Freese et al 2011 [50]	22F	52.0 (10.6)	i. Single session of four 30-s maximal cycle sprints ii. 6 weeks of four to eight 30-s maximal	84 kJ/kg ffm 1.6 g fat/ kg ffm	14	i. 13.1% reduction in TAG AUC after single sprint session, $P<0.05$ *vs.* rest Effect size = 0.32 ii. 9.7% reduction in TAG AUC after 6 weeks of sprint training, $P<0.05$ *vs.*

			cycle sprints for 3 bouts/week iii. Rest (control)			rest Effect size = 0.23
Freese et al 2011 [52]	6M 6F	22.0 (3.2) 20.8 (0.8)	i. Four 30-s maximal cycle sprints ii. Four 30-s maximal cycle sprints with energy replacement post-exercise iii. Rest (control)	68 kJ/kg bm 1.2 g fat/ kg bm	14	i. 21% reduction in TAG AUC after sprints in energy deficit, P=0.006 *vs.* rest ii. 10% reduction in TAG AUC after sprints in energy balance, P=0.044 *vs.* rest iii. 12% reduction in TAG AUC after sprints in energy deficit vs energy balance, P=0.032
Gabriel et al 2012 [53]	9M	24 (3)	i. Five 30-s maximal cycle sprints ii. 30 min continuous walking at 6.7 (0.2) km/h iii. Rest (control)	Two identical meals 3 hours apart: 46 kJ/kg bm 0.7 g fat/ kg bm	18-21	TAG AUC: i. Sprints: 14.13 (2.83) $mmol \cdot l^{-1} \cdot 7h^{-1}$ ii. Walking: 16.33 (3.51) $mmol \cdot l^{-1} \cdot 7h^{-1}$ iii. Rest: 17.18 (3.92) $mmol \cdot l^{-1} \cdot 7h^{-1}$ P=0.056 sprint *vs.* rest P>0.05 walking *vs.* rest

						P>0.05 sprints *vs.* walking
Gabriel et al 2013 [54]	8M	25(4)	i. Five 30-s maximal cycle sprints ii. Rest (control)	Two identical meals 3 hours apart: 46 kJ/kg bm 0.7 g fat/ kg bm	i. 19-22 ii. 43-46	TAG AUC Day 2 (19-22 h): i. Sprints: 7.46 (1.53) mmol·l^{-1}·7h^{-1} ii. Rest: 9.47 (3.04) mmol·l^{-1}·7h^{-1} P<0.05 between trials TG AUC Day 3 (43-46 h): i. Sprints: 9.05 (0.92) mmol·l^{-1}·7h^{-1} ii. Rest: 9.36 (1.07) mmol·l^{-1}·7h^{-1} P>0.05 between trials
Tan et al 2013 [55]	5M 4F	22.9 (2.2)	i. Four 30-s maximal cycle sprints ii. 20-min continuous cycling at 70% of maximal oxygen uptake iii. Rest (control)	56 kJ/kg bm 1.11 g fat/ kg bm	14	TAG AUC: i. Sprints: 9.5 (3.5) mmol·l^{-1}·6h^{-1} ii. Continuous: 8.6 (3.1) mmol·l^{-1}·6h^{-1} iii. Rest: 9.3 (1.9) mmol·l^{-1}·6h^{-1} No difference among trials, P>0.05
Tan et al 2014	12 F	21.3 (2.1)	i. Sixty 8-s cycle sprints ii. Rest (control)	Standardised: 4.17 MJ	13.5	TAG AUC: i. Sprints: 5.84 (1.08) mmol·l^{-1}·4h^{-1}

[56]	98 g fat	ii. Rest: 6.71 (1.63) $mmol \cdot l^{-1} \cdot 4h^{-1}$ P<0.05 between trials

Reported values for all studies are mean (SD)

M, male; F, female; HRR, heart rate reserve; MAS, maximal aerobic speed; bm, body mass; ffm, fat free mass; TAG, triacylglycerol; AUC, total area under the concentration versus time curve.

Table 2. Studies examining the effect of high-volume submaximal interval exercise on postprandial triacylglycerol.

Reference	n Sex	Age (y)	Study design	Test meal energy and fat content	Time from exercise cessation to test meal consumption (h)	Main findings
Ferreira et al 2011 [43]	20M	21.5 (3.5)	i. 3-min interval sprint runs at 115% of AT until 500 kcal ii. Continuous running at 85% of AT until 500 kcal iii. Rest (control)	50 kJ/kg bm 1.0 g fat/ kg bm	0.5	TAG AUC: i. Sprints: 9.49 (3.64) $mmol \cdot l^{-1} \cdot 4h^{-1}$ ii. Continuous: 9.16 (3.05) $mmol \cdot l^{-1} \cdot 4h^{-1}$ iii. Rest: 11.22 (4.38) $mmol \cdot l^{-1} \cdot 4h^{-1}$ Sprints & continuous $P<0.05$ *vs.* rest
Trombold et al 2013 [44]	6M	25.0 (2.9)	i. Interval exercise: 2 min cycling at 90% peak oxygen uptake followed by 2 min at	67 kJ/kg bm 1.02 g fat/ kg bm	12	i. Mean TAG AUC after interval exercise 69.4 (17.1) % of rest , $P=0.021$ *vs.* rest ii. Mean TAG AUC after continuous

			25% peak oxygen uptake; isoenergetic to continuous cycling ii. Continuous cycling at 50% peak oxygen uptake for 60 minutes iii. Rest (control)			exercise 81.1 (16.0)% of rest , P=0.102 *vs.* rest iii. No difference in TAG AUC after interval and continuous cycling (P=0.276)
Tyldum et al 2009 [45]	8M	42 ± 4 (mean ± SE)	i. Four 4-min sprints at 85-95% of HRmax isoenergetic to continuous exercise ii. Continuous exercise at 60-70 HRmax iii. Rest (control)	Standardised: 3.8 MJ 48.3 g fat	16-18	No significant difference among trials, P>0.05
Barrett et al 2006 [46]	12M	21.1 ± 0.4 (mean ± SE)	i. Four blocks of interval exercise of walk, sprint, cruise and jog[a] ii. Four 15-min blocks of	69 kJ/kg bm 1.25 g fat/ kg bm	16	TAG AUC: i. Interval: 7.41 ± 0.61 $mmol \cdot l^{-1} \cdot 6h^{-1}$ ii. Continuous: 8.02 ± 0.85 $mmol \cdot l^{-1} \cdot 6h^{-1}$ iii. Rest: 9.85 ± 0.77 $mmol \cdot l^{-1} \cdot 6h^{-1}$

			continuous uphill walking at 60% of maximal oxygen uptake iii. Rest (control)			P=0.001 interval *vs.* rest P=0.028 continuous *vs.* rest (mean ± SE)
Barrett et al 2007 [47]	19M	15.4 ± 0.1 (mean ± SE)	9 boys: i. Four blocks of interval exercise of walk, sprint, cruise and jog[a] ii. Rest (control) 10 boys: i. Four 15-min blocks of continuous uphill walking at 60% of maximal oxygen uptake ii. Rest (control)	69 kJ/kg bm 1.25 g fat/ kg bm	16	TAG AUC: i. Interval: 6.92 ± 0.79 mmol·l^{-1}·6h^{-1} ii. Rest: 9.38 ± 1.25 mmol·l^{-1}·6h^{-1} P=0.002 interval *vs.* rest i. Continuous: 7.26 ± 0.82 mmol·l^{-1}·6h^{-1} ii. Rest: 8.39 ± 0.75 mmol·l^{-1}·6h^{-1} P=0.050 continuous *vs.* rest (mean ± SE)

[a]Protocol is Loughborough Intermittent Shuttle Test (LIST)

Reported values for are mean (SD) excepted where stated

M, male; HRmax, maximal heart rate; bm, body mass; SE, standard error; TAG, triacylglycerol; AUC, area under the concentration versus time curve; AT, anaerobic threshold.