Acute exercise and postprandial lipemia in young people

Running head: Postprandial lipemia in young people

Abstract

Exaggerated postprandial triacylglycerol concentrations ([TAG]) independently predict future cardiovascular events. Acute exercise and diet interventions attenuate postprandial [TAG] in adults. This paper aims to examine the exercise postprandial lipemia studies published to date in young people. Nine studies satisfied the inclusion criteria adopted for this summary. The majority of studies are in boys (22% girls) and have shown a single ~60 min session of moderate intensity exercise, performed 12 to 16 h before a standardised meal, reduces postprandial [TAG]. Manipulations of exercise duration and intensity suggest an exercise energy expenditure dose-dependent response is not supported directly in healthy young people. Studies investigating alternative exercise bouts have reported lower postprandial [TAG] after simulated intermittent games activity, high intensity interval running and cumulative 10-min blocks over several hours, which may appeal to the spontaneous physical activity habits of young people. Although extension of these initial findings is warranted, exercise may be an effective strategy to promote regular benefits in TAG metabolism in children and adolescents; this may contribute to an improved cardiovascular disease risk profile early in life.

Introduction

The aim of this article is to draw attention to the burgeoning area of postprandial lipemia research in young people by highlighting primary trends and suggesting future directions. Since the 1970s, numerous research papers have been published investigating the effects of exercise on postprandial lipemia in adults (30, 38, 46); however, this has only been extended to young people since 2007 (3). Postprandial lipemia is most often characterised by the elevation in circulating concentrations of triacylglycerol (TAG) following the consumption of a high fat meal (Figure 1). However, it was suggested recently that there is no widely agreed definition of postprandial lipemia (40). This is largely because of wide variation in study design, which has made it difficult to compare studies directly and may have hindered the widespread application of common findings to influence public health. For the purposes of this article, postprandial lipemia is defined as the increase in plasma triacylglycerol concentration ([TAG]) after consumption of a meal, drink or single food item (Figure 1).

For the reasons outlined below, continued research examining postprandial lipemia in young people is important when considering factors that influence future cardiovascular disease risk. However, this field of study is in its infancy precluding what could reasonably be described as a systematic review. Similarly, there are insufficient data to conduct a meta-analysis, but we have provided effect sizes in what is most appropriately described as a summary of evidence available currently linking acute exercise with postprandial lipemia. This allows us to highlight primary trends and directions for future research. Also, the commonly employed two-day experimental model, described below, may be very well suited to other areas of

research with young people outside of postprandial lipemia.

Although this is not a review, our methods were systematic and included a computerised search of the literature using PubMed, SciVerse Scopus, Web of Science, Google Scholar and SportDiscus. Keywords searched independently or in combination included: postprandial, triglyceride, triacylglycerol, lipemia, lipaemia, exercise, energy expenditure, child, adolescent, boy and girl. All relevant sources were cross-referenced to maximise our search for manuscripts that matched our inclusion criteria. For inclusion, studies were required to (1) be an intervention study in which one of the aims of the study was examining postprandial lipemia after an exercise intervention; (2) include young people <18 years as participants of study at baseline; (3) include a measure of acute exercise manipulation performed up to 18 h before ingestion of a high-fat meal; (4) include a resting control condition; (5) include a measure of postprandial lipemia; (6) be published in the English Language in a peer reviewed journal up to and including July 2013.

Since Zilversmit (88) suggested atherosclerosis was a postprandial phenomenon, evidence from case-control studies support the association between elevated postprandial [TAG] and cardiovascular disease (28, 59). Subsequent prospective epidemiological studies confirm that elevated postprandial [TAG] is linked with an increased incidence of future cardiovascular events in men and women, independent of baseline cardiac risk factors, other lipid concentrations and markers of insulin resistance (1, 35, 58, 68). Moreover, postprandial [TAG] may be a better predictor of cardiovascular disease risk than traditional fasting measures (1).

The majority of the daytime is spent typically in a postprandial state (14 to 16 h·24 h^{-1} for adults) (40). Considering [TAG] reaches a peak approximately 4 h following the consumption of a meal in adults (58), the metabolic perturbations evident following a meal are unlikely to subside before subsequent meals are consumed. Regular exposure to elevated postprandial [TAG] facilitates the exchange of lipids between triglyceride- and cholesterol-rich lipoproteins and, therefore, promotes a more atherogenic lipid profile of TAG-rich lipoprotein remnants, small, dense low-density lipoprotein and low concentrations of high-density lipoprotein (7). The greater exposure of dietary lipoproteins to the arterial wall may contribute to the development of atherosclerotic plaques that precipitate the manifestation of clinically overt symptoms (47).

Although the clinical manifestations of atherosclerotic disease are not apparent typically until mid-adulthood, the process of atherosclerosis is initiated during childhood and adolescence, and progresses over the lifespan (19, 47). Fatty streaks are considered to be the earliest lesion of atherosclerosis (47), with the presence of aortic fatty streaks identified in the first decade of life (33) and coronary fatty streaks in the second decade of life (74). Although fatty streaks may be fairly innocuous, there is convincing evidence suggesting that they can progress gradually over time into clinically significant atherosclerotic lesions (29, 72). Indeed, the prevalence and extent of fatty streaks and clinically significant atherosclerotic lesions increases rapidly in the arteries of adolescents and young adults (73).

Several prospective cohort studies indicate that cardiovascular disease risk factors, including abnormal lipid and lipoprotein concentrations, during childhood and

adolescence can track into adulthood (2, 14, 57), and are associated with the development of subclinical atherosclerosis in later life (11, 17, 64). The presence of multiple risk factors augments the extent of atherosclerotic legions in young people (4). Moreover, childhood fasting [TAG] is associated independently with young adult cardiovascular disease (54). However, substantial variation is evident in fasting [TAG] in children (79), highlighting the importance of studying [TAG] in the postprandial period. Accordingly, lifestyle interventions, including manipulations in exercise energy expenditure and dietary energy intake, that moderate postprandial lipemia may delay atherogenic progression and should be initiated during childhood and adolescence (19, 47). Therefore, this manuscript will examine the effect of acute exercise interventions on postprandial lipemia in children and adolescents. Considering the infancy of this research area, evidence from adults will be summarised briefly before highlighting key findings to date in children and adolescents.

Exercise and postprandial lipemia in adults

The literature is replete with adult-based studies examining the effect of exercise and dietary manipulations on postprandial lipemia; these have been reviewed elsewhere and we encourage interested readers to access these for a more thorough understanding of the platform that they provide for studies with young people (e.g., 21, 30, 38, 46, 60, 62). The effect of exercise on postprandial [TAG] appears short-lived and dependent on the proximity of the most recent exercise session (31). The notion that acute continuous, moderate intensity exercise reduces postprandial [TAG] is well established in men and women (46). An important determinant of the exercise-induced reduction in postprandial [TAG] is the associated acute exercise

bout energy expenditure (83), although meaningful attenuations in postprandial lipemia are still evident following modest doses of moderate intensity exercise energy expenditure (~1 MJ, 240 kcal) (49, 50). Although a meta-analysis suggested that energy expenditure may be the primary driver of changes in postprandial lipemia, once two outlying studies were accounted for, the strength of this effect was only moderate (62). However, the authors conceded that the only basis for removing these outliers was their large effects relative to other studies included in the analysis as opposed to methodological flaws. Manipulations of exercise intensity and duration support a dose-dependent response in adults (26, 39), although variations in design characteristics preclude the identification of a so-called optimal or minimal dose. It should be noted that the exercise energy expenditure dose-response relationship, described above, applies only to moderate- to vigorous-intensity, aerobic exercise and does not include resistance (e.g., 61, 86) or high intensity (e.g., 18, 20) exercise in adults.

Another avenue of research has highlighted that accumulating shorter bouts (≤10 min) of moderate intensity exercise throughout the day appear equally effective in reducing postprandial [TAG] as a similar duration of continuous exercise (49, 50, 51). This may be important from a public health standpoint as it aligns with international recommendations for daily habitual physical activity accumulation (55). More recently, acute high intensity exercise (ranging from 90% peak $\dot{V}O_2$ to maximal sprint intensity), where the total exercise dose is probably considerably less than that reported in many of the moderate intensity exercise studies, has been shown to reduce postprandial [TAG] in healthy adults (16, 18, 20). It has even been suggested that this type of exercise may be more efficacious than moderate intensity exercise

(82).

A small number of studies with adults have examined the combined effect of manipulations in dietary intake and exercise energy expenditure to determine whether energy deficit or muscle contraction is the primary determinant of the commonly reported reduction in postprandial lipemia following an acute bout of exercise. Gill and Hardman (25) found that an exercise-induced energy attenuation of TAG was almost three times larger than the reduction seen when dietary energy intake was restricted in the same group of postmenopausal women the day before the postprandial measurements. Moreover, they reported quantitative and qualitative differences in carbohydrate and lipid metabolism between the energy deficit manipulations. After a thorough appraisal of the insulin, glucose, TAG, and fatty acid responses during the fasting and postprandial phases of the study, they concluded that “over the long term, energy deficit may improve triacylglycerol metabolism but the mechanisms by which exercise enhances triacylglycerol metabolic capacity are independent of, or at least additive to, the effects of a whole-body energy deficit” (p.470). A technical error in the exercise condition measurements meant that the energy deficit was not the same as the dietary intake restriction (1.73 vs. 1.44 MJ; ~17% difference); however, the authors indicated that the stark difference in outcome measures between the conditions meant that this did not affect the general interpretation of the results. Two recent studies have examined the efficacy of combining acute exercise and energy-intake restriction protocols to attenuate postprandial [TAG] in healthy, young women (44, 45). Although energy-intake restriction alone did not exceed the reduction in postprandial [TAG] observed for exercise alone, the combination of exercise and energy-intake restriction did at least

match it (45). The authors concluded that this combination may be attractive for individuals when regular prolonged low- to moderate-intensity exercise is not feasible because of lifestyle constraints. Collectively, these studies suggest that although exercise energy expenditure may be a determinant in attenuating postprandial lipemia, the role of muscle contraction *per se* may need to be examined more thoroughly to determine its function when the exercise dose is low.

Burton et al. (5) followed-up Gill's work with Hardman (25) by comparing exercise energy deficit (-2.8 MJ) and exercise with dietary compensatory replacement (balance) with a resting control condition in overweight and obese middle-aged men. Both exercise conditions resulted in metabolic changes compared with a resting control; however, only the exercise deficit attenuated fasting and postprandial [TAG]. The changes in [TAG] were ascribed largely to changes in hepatic very-low density lipoprotein (VLDL) metabolism. The authors concluded that, to maximise the potential beneficial effects of exercise on postprandial lipemia, an exercise-induced energy deficit is required. Thus, dietary compensatory energy replacement may need to be monitored in individuals wishing to experience the 'full' benefit of exercise.

Exercise and postprandial lipemia in children and adolescents

Current international physical activity guidelines recommend that children and adolescents engage in 60 to 90 min of moderate intensity daily physical activity (13, 36). However, depending on how this is quantified, many children and adolescents fail to meet the guidelines (65), and physical activity participation has been shown to decline from childhood through adolescence (56). Therefore, it is likely that exercise interventions in young people need to be engaging and sustainable in order for long-

term benefits in metabolic health to emerge. This section will summarise the published exercise postprandial lipemia studies with young people. It has not been possible to identify published studies that have compared directly the effect of exercise and dietary manipulations on postprandial lipemia in young people.

Nine studies have investigated the effect of acute exercise on postprandial lipemia in young people using the criteria adopted for this summary (3, 41, 42, 69, 70, 76, 78, 80, 81). A summary of these studies is presented in Table 1. In line with the adult literature, a single session of moderate to vigorous intensity exercise inducing an exercise energy expenditure ≥1.0 MJ (240 kcal) attenuates postprandial [TAG] in boys and girls (3, 41, 42, 70, 78, 80, 81). Although the magnitude of change in postprandial [TAG] after exercise varies in these studies (Table 1), on average the changes are moderate with effect sizes ranging from 0.26 to 0.86 in those that reported statistically significant attenuations compared to a resting control condition. Thus, an exercise-induced deficit is efficacious, but a closer examination of the exercise characteristics might be more enlightening and will be used to compare and contrast the main outcomes with the adult-based literature appraised above. Before doing this, however, a brief description of the standardised methods and two-day experimental model may help those thinking of adding postprandial (lipemic) research to their portfolio.

Standardised methods and two-day experimental model

The standard two-day model that has been employed in the majority of the published exercise studies to date with young people is depicted in Figure 2. Each participant completes the two or three conditions in a random order separated by a

standardised period (minimum of 7 days). For the 48 hours preceding day 2 of the first counter-balanced condition, participants are usually asked to minimise and record their habitual physical activity and record their food and drink intake. These records are used subsequently to replicate these extraneous factors, which are likely to affect the study outcome measures across the conditions. The level of care in controlling or accounting for habitual physical activity and diet in the studies appears to have increased since the first study was published six years ago, which places an added burden on the participants. Some studies have introduced a lunch meal to better reflect normal dietary practice and a specific evening snack to standardise the fasting period. A critical feature of the two-day model is that the exercise intervention precedes the first blood sample by 12 to 18 hours (Table 1). This is because lipoprotein lipase (LPL) activity appears to peak in this period after exercise (71) and has been implicated mechanistically in reducing postprandial [TAG] (46).

In the first published exercise postprandial lipemia study with young people, Barrett et al. (3) demonstrated that single sessions of continuous moderate intensity exercise and simulated intermittent games activity (Figure 3) reduced postprandial [TAG] by ~14% and 26% respectively compared with resting control in late adolescent boys (15.4 y). Details of the simulated intermittent games activity can be found in the original publication (3), but briefly consisted of four x 18 min blocks of walking, sprinting, cruising, jogging and rest (Figure 3). The larger reduction in the intermittent games activity was linked tentatively to the greater exercise energy expenditure; however, the evidence is limited by the indirect comparison of two different groups of boys in the between-measures design and absence of energy expenditure quantification during the intermittent games activity (3). Nevertheless, it

is very likely that the intermittent games arm of the study resulted in considerably higher energy expenditure given the total exercise time was 14 min longer and the average intensity was ~17% higher. In addition to being the first of its kind, this study is notable because the intermittent exercise was thought to better match the stop-start nature of young people's activity habits compared with the long-duration continuous exercise that dominates the adult-based postprandial lipemia literature.

In a more recent study, Thackray et al. (76) investigated the acute effect of high intensity interval running on postprandial [TAG] in 11 to 12 year old boys. The exercise intervention adopted in this study involved 10 x 1 min treadmill runs at 100% peak $\dot{V}O_2$ (maximal aerobic speed), with 1 min active recovery between each interval and resulted in a moderate reduction in postprandial [TAG] compared to a non-exercise control condition. The high intensity nature of this exercise precluded the direct measurement of energy expenditure; however, considering the short exercise duration (10 minutes in total), it is likely the energy expenditure was lower than that reported in other studies in young people adopting longer and less intense exercise interventions (3, 41, 42, 70, 78, 80, 81), suggesting that exercise intensity is a key factor influencing postprandial TAG metabolism in young people. Importantly, the exercise protocol was well tolerated by the boys in this study, and recent evidence suggests that pre-pubertal boys prefer moderate intensity exercise interspersed with short bursts of high intensity effort compared with continuous moderate intensity exercise alone (9). Considering children, typically, spend a greater percentage of time engaged in high intensity activities than adults (34), exercise with an intermittent games activity (3) or a high intensity (76) component may be appealing interventions to enable young people to acquire metabolic health

benefits. We encourage further research that adopts alternative modes of exercise to increase the ecological validity of postprandial research, but appreciate that there are challenges to overcome in quantifying the energy expenditure in these studies. Perhaps judicious use of movement detection technologies can be employed to overcome this measurement issue.

In contrast to the study conducted by Barrett and colleagues (3), and studies with adults (26, 39), subsequent studies in healthy boys do not support an exercise energy expenditure dose-dependent response (78, 80). Specifically, performing 60 min of moderate and vigorous intensity exercise were similarly efficacious in attenuating postprandial [TAG] by ~24% and 21% respectively, despite the 45% greater gross exercise energy expenditure at the higher intensity (80). Moreover, despite doubling the gross exercise energy expenditure in the 60 min (1867 kJ) compared with the 30 min condition (982 kJ), the additional attenuation in postprandial [TAG] was trivial (20% vs. 16% respectively) (78). This led the authors to suggest a ‘threshold’ attenuation may exist, above which additional increases in energy expenditure will not reduce postprandial [TAG] further in apparently healthy normolipidemic boys (78). Recently, when the same study design was conducted in healthy adolescent girls, only the 60 min exercise condition attenuated postprandial [TAG] meaningfully and, therefore, does not provide direct support for a dose-response or ‘threshold’ attenuation (81). However, it is worth noting that 56% of the girls in this study experienced a lower postprandial TAG response after the 30 min exercise condition compared with a resting control, suggesting that on an individual level, some girls may benefit from a lower dose of moderate intensity exercise (81).

Some postprandial lipemia studies have included design features that provide insights beyond the energy expenditure focus that has dominated the majority of adult-based studies and the research with young people described above; these are now reviewed. Consumption of meals high in fat, and the associated lipemic response, have been linked to endothelial dysfunction in adults (85). Endothelial dysfunction is considered to be the first stage in atherogenesis (66) and is considered to be a pre-requisite for the development of atherosclerosis (37). In a study of 13 healthy adolescent boys, postprandial endothelial function, indicated by flow mediated dilation (FMD), was reduced by 32% following a high-fat breakfast and 24% after lunch compared with fasting in a non-exercise control condition (70). In contrast, 60 min of treadmill walking at 60% peak $\dot{V}O_2$ prevented the postprandial decline in FMD, once it had been normalised for the post-occlusion shear rate. It is noteworthy that the boys in this study did not demonstrate any of the risk factors for coronary heart disease, yet the consumption of the high-fat meals, common in westernised countries (75), induced endothelial dysfunction to a similar extent seen in adults (70). However, although this maintenance of normal endothelial function coincided with an attenuation in plasma [TAG], FMD was not meaningfully related to [TAG] in either the control or exercise conditions or at any time point ($-0.14 < r < 0.23$; $P \geq 0.46$). Hence, the authors concluded that simultaneous changes in postprandial lipemia and endothelial function were coincidental rather than causative.

When 60 min of exercise at 70% peak $\dot{V}O_2$ was accumulated throughout the day in 6 x 10 min bouts by 12.9 y old boys, this exercise was also found to prevent the postprandial decline in FMD (69). However, although the total and incremental areas

under the [TAG] versus time curve were reduced as a result of exercise by 11% and 16%, these differences were not statistically significant. Despite these non-significant changes in postprandial [TAG], the reduction in the total and incremental areas under the [TAG] versus time curve are in line with previous research in adolescents and may still be meaningful physiologically. A significant attenuation of postprandial endothelial dysfunction was observed after the accumulated exercise; thus, providing additional support that exercise-induced alterations in postprandial endothelial function may be independent of changes in postprandial [TAG]. Accumulated exercise may be particularly appealing to young people as they perceive prolonged activity to be more demanding than adults (77). In addition, short bouts of activity may be easier to incorporate into the school day than extended bouts of activity.

Postprandial response in the overweight and obese

Elevated postprandial lipemia or delayed postprandial TAG clearance have been reported consistently in various adult-patient groups (e.g., 28, 59, 63, 67) and some studies have been conducted with adults who are overweight, obese, with metabolic syndrome or diabetic (5, 8, 10, 15, 22, 48, 49, 52, 87). However, direct comparisons between healthy and patient groups are rare and present mixed results. It would appear that only two studies have examined the postprandial lipemic response to exercise in overweight young people (41, 42). MacEneaney et al. (42) found overweight late adolescent boys experienced a similar exercise-induced attenuation in postprandial [TAG] (~20%) compared with similarly-aged, normal [*healthy*] weight boys. It should be noted, however, that the overweight boys in the MacEneaney study (42) had no history of diabetes, heart disease, or liver dysfunction, and were normo-tensive, -lipidemic and -glycemic. Despite the significant body size and

composition differences between the two groups of boys, they were both considered to be healthy. Although the skinfold measures used to estimate body composition could not provide a more detailed pattern of fat distribution in the boys, there was a statistically significant positive association between the total area under the [TAG] versus time curve and sum of skinfolds in both the control (r = 0.49) and exercise (r = 0.47) conditions. Moreover, [TAG] returned to baseline after 6 h in the healthy weight boys, but not in the overweight-boys. The authors suggested this may be due to delayed clearance, but recent evidence in adults may point to differences in hepatic release of fatty acids in TAG (12).

In a study examining the effect of 60 min of moderate intensity cycling on postprandial lipemia in overweight black and white adolescents (41), acute exercise reduced [TAG] in both groups, but the reduction was greater in white (19%) than in black (8%) adolescents. Interestingly, the authors found increased visceral fat was a major contributor to the greater reduction in [TAG] seen in the white, but not black adolescents. As in the MacEneaney study (42), the overweight participants of this study were otherwise healthy. The results of the MacEneaney (42) and Lee (41) studies are supported by other non-exercise studies. Although obese and non-obese adolescents exhibit a similar postprandial TAG profile (53, 84), those with a central pattern of fat distribution displayed signs of impaired TAG clearance (53). Consequently, the potential for exercise to improve this aspect of metabolic health in overweight children and adolescents is promising.

Mechanisms

The mechanisms responsible for the reduction in postprandial [TAG] following acute

exercise in young people have not been investigated currently, largely due to the invasive nature of the techniques required. Consequently, the proposed mechanisms in adults are drawn upon to provide some insight into the exercise-induced reduction in postprandial [TAG] in young people. In simple terms, the mechanisms in adults involve either enhanced removal or diminished appearance of circulating TAG, but most likely a combination of the two. It is postulated that increased LPL activity, the rate limiting enzyme responsible for hydrolysis of circulating TAG, may facilitate the enhanced clearance of circulating TAG; the tissue specific location of this increased LPL activity may be critical, but it is not yet clear whether skeletal muscle is the primary site. However, reductions in postprandial [TAG] have been reported, with little change in postheparin plasma LPL activity (27) or skeletal muscle LPL activity (32). Nevertheless, exercise-induced changes in LPL activity are correlated strongly with the attenuation in postprandial [TAG] following exercise (27, 32). Alternatively, or in addition, moderate intensity exercise has been shown to reduce the concentration of postprandial VLDL-TAG (24, 43). Indirect evidence for altered hepatic VLDL secretion can be drawn from studies reporting an increase in the circulating concentration of fasting and postprandial 3-hydroxybutyrate (3-OHB), a marker of hepatic fatty acid oxidation, alongside the attenuation in postprandial [TAG] (23, 24, 43). This suggests a shift in hepatic VLDL kinetics from fatty acid re-esterification and VLDL synthesis and towards hepatic fatty acid oxidation. A recent stable isotope enrichment study with sedentary obese women indicated that the reduction in postprandial lipemia after endurance and resistance exercise bouts was not achieved by enhanced clearance of dietary fat, but by reduced abundance of endogenous fatty acids in plasma TAG (12). However, this study did not use the two-day model used most commonly in the paediatric literature (described above, Figure

2); therefore, it is possible that the mechanism responsible for any alterations in postprandial metabolism are different.

Implications and future directions

Although from a small number of studies, the evidence from young people demonstrates consistently that a single session of moderate to high intensity exercise promotes reductions in postprandial [TAG] in this population (Table 1). While it appears that a 'threshold' of exercise energy expenditure may be required to promote a meaningful reduction in the postprandial TAG response, the evidence of a dose-dependent response is not yet supported directly in boys or girls (78, 80, 81). The studies in Table 1 have provided high fat meals that may exaggerate the postprandial lipemic effect - although these are not ecologically valid, they have permitted comparison of the various experimental conditions. Eventually, future studies will need to consider meals that young people normally eat. In the absence of a clinical endpoint, the relevance of these findings cannot be determined from a clinical perspective. Nevertheless, the evidence from these studies is encouraging and suggests that exercise may be an effective strategy to attenuate postprandial lipemia in children and adolescents. Consequently, promoting a physically active lifestyle from a young age may stimulate regular benefits in lipid metabolism and have long-term implications regarding cardiovascular disease risk, but additional work is required to increase the evidence-base.

Future acute exercise postprandial lipemia studies should examine the efficacy of innovative and realistic exercise and/or dietary manipulations to strengthen the evidence base in this area. This should include different forms of exercise, such as

resistance exercise and inclusion of girls, both of which are noticeably missing or sparse in studies with young people. In combination with other evidence, this may ultimately influence exercise and health guidelines in young people. Additional exercise-based studies are also required in young people with cardiovascular disease risk factors, such as fasting hypertriglyceridemia, obesity, insulin resistance and Type 2 diabetes mellitus. Furthermore, the influence of factors that may modify the postprandial TAG response following exercise, including sex, maturation, free-living energy expenditure and habitual diet warrant attention. Moreover, some indirect and relatively non-invasive techniques can be employed that may help to develop our understanding of the mechanisms responsible for the exercise-induced attenuation in postprandial [TAG] in children and adolescents (e.g., ultrasound for blood flow or isotope-ratio mass spectrometry for gas analysis).

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Figure legends

Figure 1 Changes in postprandial plasma triacylglycerol concentration across experimental conditions. Black rectangle denotes consumption of a standardised test meal.

Figure 2 Schematic of the standard two-day model employed in the majority of exercise postprandial studies in young people.

Figure 3 Diagram of the Loughborough intermittent shuttle test (LIST) protocol employed by Barrett et al. (3). The protocol was designed to simulate games activity and consisted of repeated and standardised patterns of walking, sprinting, cruising and jogging.

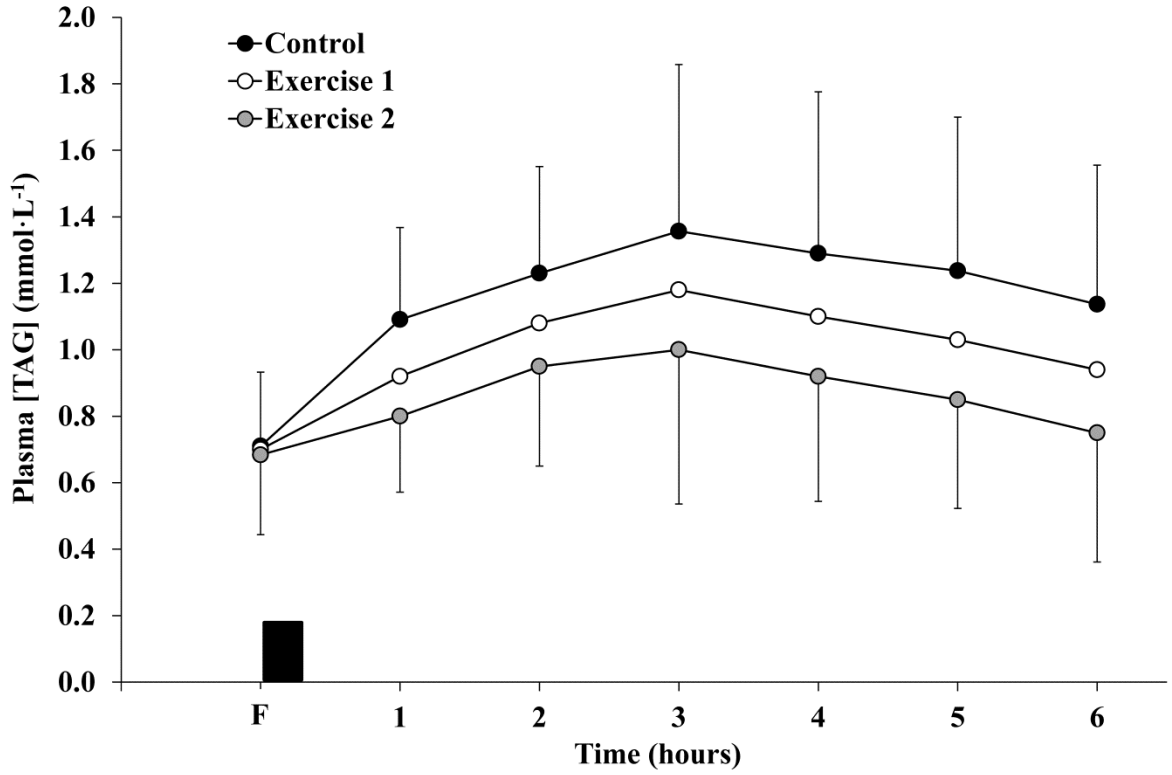
Table 1 Summary of studies examining the effect of acute exercise on postprandial lipaemia in children and adolescents

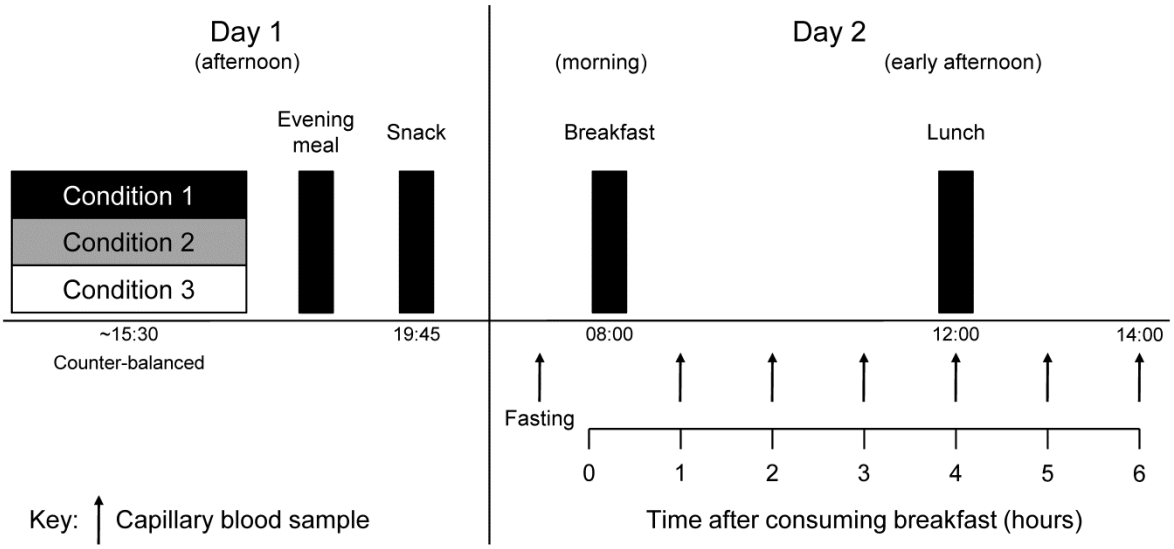
Study	n	Sex	Age (years)	Intervention (INT)	EE (MJ)	INT to meal delay (h)	Amount of fat (g/kg BM)†	Effect size*
Barrett et al. (3)	10 9	M M	15.3 15.4	4 x 15 min TMW @ 59% peak $\dot{V}O_2$ 4 x 18 min LIST @ 69% peak $\dot{V}O_2$	2.0 NA	16.0	1.3	0.46 0.78
Thackray et al. (76)	15	M	11.8	10 x 1 min TMR @ 100% peak $\dot{V}O_2$	NA	15.5	Breakfast 1.5 Lunch 1.1	0.50
Tolfrey et al. (80)	8	M	12.9	6 x 10 min TMR @ 53% peak $\dot{V}O_2$ 6 x 10 min TMR @ 75% peak $\dot{V}O_2$	1.5 2.2	14.7	1.5	0.86 0.67
Tolfrey et al. (78)	11	M	13.3	3 x 10 min TMR @ 55% peak $\dot{V}O_2$ 6 x 10 min TMR @ 55% peak $\dot{V}O_2$	1.0 2.0	14.5	1.5	0.26 0.32
Tolfrey et al. (81)	18	F	13.0	3 x 10 min TMW @ 55% peak $\dot{V}O_2$ 6 x 10 min TMW @ 56% peak $\dot{V}O_2$	0.8 1.5	14.5	1.5	0.10 0.40
Sedgwick et al. (70)	15	M	13.6	60 min TMW @ 60% peak $\dot{V}O_2$	1.9	15.0	Breakfast 1.5 Lunch 1.1	0.71
Sedgwick et al. (69)	14	M	12.9	6 x 10 min TMR @ 72% peak $\dot{V}O_2$ 09:30 start (ex10:50rest) x 6	1.9	18.0	Breakfast 1.5 Lunch 1.1	0.38
MacEneaney et al. (42)	10HW 8 OW	M M	15.6 15.9	59 min TM @ 65% peak $\dot{V}O_2$ 52 min TM @ 65% peak $\dot{V}O_2$	2.5 2.5	12.0 to 14.0	97 g/2 m^2 BSA	0.57 0.72
Lee et al. (41)	21B 17W	9M/12F 12M/5F	15.4B 14.5W	60 min cycling @ 50% peak $\dot{V}O_2$	1.9B 2.1W	14.0	64B & 66W g/2 m^2 BSA	0.18B 0.46W

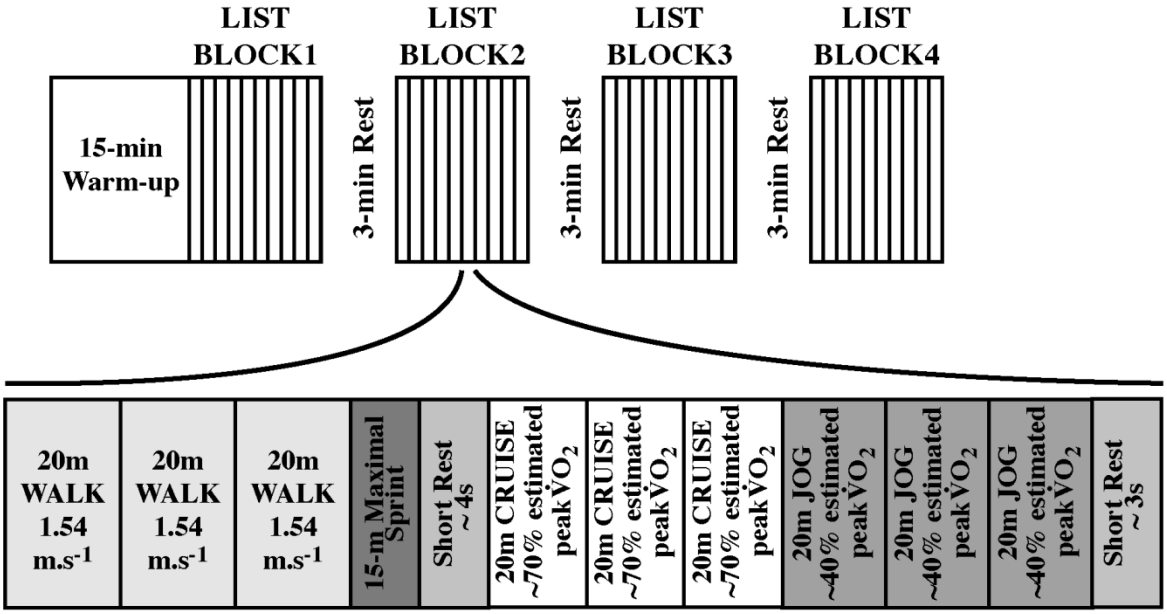
Studies are arranged to match the critical appraisal in the text. †Unless indicated specifically, test meals were consumed as breakfast

*An effect size of 0.2 represents the minimum important difference, 0.5 moderate and 0.8 large (6); all values represent effects for exercise-induced total area under the time curve for triacylglycerol (TAUC-TAG) compared with a non-exercise control condition - taken directly from published studies or calculated from (Exercise $\bar{x}$ – Control $\bar{x}$)/pooled SD.

EE energy expenditure; M male; F female; TM treadmill – W walk, R run; $\dot{V}O_2$ oxygen uptake; BM body mass; BSA body surface area; LIST Loughborough intermittent shuttle test; HW healthy weight; OW overweight; B black; W white


Control
Exercise 1
Exercise 2
Plasma [TAG] (mmol·L-1)
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
F
1
2
3
4
5
6
Time (hours)


Day 1
(afternoon)
Day 2
(morning)
(early afternoon)
Evening meal
Snack
Breakfast
Lunch
Condition 1
Condition 2
Condition 3
~15:30
Counter-balanced
19:45
08:00
12:00
14:00
Fasting
0
1
2
3
4
5
6
Key:
Capillary blood sample
Time after consuming breakfast (hours)



Pattern repeated 10 times to form one 'BLOCK' of exercise