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The Latest on the Effect of Prior Exercise on Postprandial Lipaemia

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Abstract

This review examines the effect of prior exercise on postprandial triacylglycerol (pTAG) concentrations, an independent risk factor for cardiovascular diseases. Numerous studies have shown that a single bout of exercise reduces pTAG concentrations; however, several modulators such as exercise energy expenditure/deficit, mode of exercise (aerobic/resistance/high intensity/intermittent exercise or combinations), type of meal (moderate or high fat), time frame between exercise and meal and target group may individually or in conjunction influence this effect. On the other hand, at least for aerobic exercise, training reduces pTAG concentrations transiently (~2 days); therefore, exercise sessions should be frequent enough to maintain this clinically significant improvement. For the healthy population, it seems that a subject's preference and ability determine which type of exercise to undertake to attenuate pTAG concentrations; an energy expenditure of ~30kJ/kg of body mass (or ~2–2.5 MJ) not combined with a corresponding increase in energy intake is required; for resistance or intermittent exercise, for those following a moderate rather than a high-fat diet, and for those with obesity (expressed as kJ/kg of body mass), a smaller energy expenditure is probably sufficient. More studies are needed to investigate dose-response/plateau effects, as well as the threshold of energy expenditure in those with diabetes mellitus and other high-risk populations. Finally, investigation of the underlying mechanisms may be clinically helpful in individualizing the appropriate intervention.

1 Introduction

Accumulated evidence suggests that hypertriacyglycerolaemia, i.e., elevated levels of plasma triacylglycerols (TAG), is associated with increased risk for atherosclerosis, independently of other known cardiovascular risk factors[1]; this association becomes more robust when it refers to the postprandial rather than the postabsorptive state, extensively reported as postprandial lipaemia (PPL)[2]. Therefore, interventions that improve postprandial TAG (pTAG) metabolism may be valuable in reducing the risk of cardiovascular diseases (CVD).

PPL is assessed using the oral fat tolerance test (OFTT), i.e., a test that examines responses to high-fat meal consumption (lipid load). The 'idea' is similar to the oral glucose tolerance test (OGTT), in which postprandial glycaemia is assessed after consumption of a high-carbohydrate meal (glucose load). Although there are no official guidelines for the OFTT

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procedure (such as exist for the OGTT), it usually lasts 6 h and requires hourly blood sampling; however, recent studies suggest that sampling may be less frequent[3, 4]. Interestingly, recent studies examine PPL after consumption of a moderate (0.35–0.7g of fat/kg body mass), rather than a high-fat meal. Whatever the fat load, PPL is mainly expressed as (1) mean pTAG concentrations; (2) peak pTAG concentrations; (3) total area under the TAG concentration-versus-time curve (AUC); and (3) incremental areas under the TAG concentration-versus-time curve (iAUC), calculated by using the trapezoidal rule. AUC and iAUC are used more frequent and are thought to be of more significant importance[5], while some researchers suggest that iAUC is the most representative measure of PPL[6], as it contains 'correction' for changes/differences observed in fasting TAG concentrations.

Increased physical activity is associated with lower risk for CVD[7]; however, the effects of exercise specifically on pTAG metabolism have not been fully elucidated, mainly due to methodological differences between studies. The aim of this review was to examine the latest findings on the effects of prior exercise (either a single bout or repeated bout, i.e., training) on PPL, to investigate which factors modulate these effects the most, and to discuss the underlying mechanisms presenting recent findings from kinetic studies.

2 Literature Search

Studies for this review were retrieved via advanced PubMed search. Search terms used included the following: 'exercise' or 'physical activity' combined with 'postprandial lipemia', or 'postprandial triglyceride', or 'postprandial triacylglycerol' or 'postprandial TG', or 'postprandial TAG'. The search was restricted to original articles published in English between January 1996 and October 2012. The reference list of retrieved articles and other relevant published reviews were also reviewed.

References were excluded if (1) study was conducted in animals; (2) were not relevant to the topics of this review; (3) if exercise was performed only during the postprandial period (we excluded these studies to avoid the acute effects of exercise on gastric emptying influencing results[8, 9]); (4) control trial or group was lacking; or (5) results of interest (pTAG) were duplicated.

In addition, studies examining the effects of one bout of exercise on PPL that did not report exercise energy expenditure or/and exercise-induced energy deficit, meal composition and mode of exercise were not included in summary Tables 1–3 and excluded from this review or not extensively analysed, since previous reviews in this area[80, 144, 145] suggested that these are primary mediators of the hypoTAG effect of exercise. Most studies included in this review were published during the last decade; however, earlier important relevant studies are also reported.

3 Effects of a Single Bout of Exercise on Postprandial Lipaemia

3.1 Acute Effects (<4 h)

Early studies have shown that, during or immediately after exercise, fasting plasma TAG concentrations are unchanged or sometimes increased, probably due to increased lipolysis and consequently increased free fatty acid delivery to the liver[10, 11]. Meal consumption in the hours after a bout of exercise is a common phenomenon, since increased energy expenditure sometimes boosts appetite in the hours after exercise (although relative energy intake, i.e. after accounting for the energy expended during exercise, remains lower after exercise compared with rest)[12–14]; hence, some studies chose to investigate the effects of exercise on pTAG concentrations immediately after, or for a few hours following the end of the exercise bout. Most of these studies, including the one in a non-healthy population

(overweight/obese with the metabolic syndrome)[15], showed a reduction in TAG responses to a high-fat meal given immediately or up to 4 h following the exercise bout (Table 1).

On the other hand, studies in which a moderate-rather than a high-fat meal was given failed to show an effect, independently of whether exercise was of low or moderate intensity[20, 22, 23] and independently of whether subjects were healthy or overweight/obese with the metabolic syndrome[21]. In addition, very low-intensity exercise[16] did not reduce PPL, while resistance exercise[18] produced the adverse effect (i.e. increases in PPL), presumably due to skeletal muscle damage associated with this mode of exercise.

However, given the fact that exercise does reduce PPL when a meal is given several hours after exercise (>12 h; see Sect. 3.2), regardless of whether the meal is of moderate fat[26, 27], or exercise is of low intensity[27, 28] or resistance[29, 30], an interaction between timing, type of meal and mode of exercise may influence the exercise effect. Early studies have suggested that the hypotricylgycerolaemic effect of exercise is attributed mainly to an exercise-induced increase in lipoprotein lipase (LPL) activity (i.e. the key enzyme responsible for circulating TAG hydrolysis, which peaks from 4 to 18 h post-exercise), and not to acute exercise-induced changes in blood flow or energy stores[31]. Therefore, many more studies have investigated the effect of exercise on TAG responses to a meal given several hours after the exercise bout (12–20 h between exercise and meal consumption). In addition, it seems that the delayed effect of exercise on PPL is more robust (in terms of percentage of decrease) than the acute effect[19, 32]. Although studies investigating the delayed effect of exercise on PPL are of great importance (see Sect. 3.2), the acute effect should not be underestimated, since, as discussed below, a growing body of evidence suggests that increased LPL mass or activity is not the sole explanation for the hypotriacylglycerolaemic effect of exercise. Moreover, from a practical point of view, feeding and exercise may be spread throughout the day; therefore, the importance of exercise in reducing PPL should include the period of the whole day. Interestingly, a recent study examined the immediate (same-day) effect of accumulating short bouts of exercise on TAG responses to moderate-fat meals (breakfast and lunch), rather than the responses to a single meal either after the last exercise bout or on the next day, and found that plasma TAG concentrations were lower in the exercise trial than in the control (rest) trial[24].

3.2 Delayed Effects (>12 h, i.e. Next Day)

Several studies have shown that a single bout of aerobic exercise reduces pTAG concentrations the next day (Tables 2 and 3), and that this effect is abolished 24–40 h after the exercise bout[53, 73]. Early studies have shown that ‘exercise energy expenditure’ is a significant predictor of the exercise-induced reduction in PPL[79]. For the healthy population and for continuous aerobic exercise, it seems that an energy expenditure of ~30 kJ/kg of body mass (or ~2–2.5 MJ) is enough to produce a significant reduction in TAG responses to a high-fat meal given the next day (Table 2). However, in most of these studies, the extra energy expenditure due to exercise relative to rest period (net exercise energy expenditure) has not been compensated for with an increase in energy intake; therefore, subjects in these studies were in ‘energy deficit’. Moreover, as discussed in the following section, regular physical activity does not influence pTAG concentrations when the effects of the last exercise bout, and hence of exercise-induced energy deficit, are no longer present[80–84]. Additionally, a recent study[41] found that both a diet- and exercise-induced energy deficit of a similar magnitude (~2 MJ) reduced TAG concentrations in the fasting and postprandial states. However, the reduction in the postprandial state was greater after exercise than after energy restriction, suggesting that (1) either exercise and diet lower triacylglycerolaemia via different mechanisms; (2) maybe the effect of exercise on TAG metabolism is not mediated only by the energy deficit induced by exercise; or (3) the energy restriction threshold may

need to be higher than the exercise-induced energy deficit to affect TAG metabolism. We recently confirmed the latest two hypotheses in a series of studies in the fasting state[85, 146].

To separate the effects of exercise *per se* from those of exercise-induced energy deficit, two recent studies included, in addition to an exercise and control trial, a third trial in which they fed subjects the amount of energy[76] or carbohydrate[55] they spent during the exercise session. Both studies found that only 'exercise without energy/carbohydrate replacement' reduces pTAG concentrations, suggesting an important role for energy deficit in the hypotriacylglycerolaemic effect of exercise. In another recent study[57], aerobic exercise without concomitant energy deficit lowered pTAG concentrations in African American women but not in White women. Unfortunately, this study lacked a comparison group that did not receive an energy-balanced post-exercise meal, and therefore we cannot conclude whether energy deficit plays an important role only in the White population, or whether ethnicity counteracts energy deficit to augment the hypotriacylglycerolaemic effect of exercise in African American women. In addition, another recent study[50] found that very high-intensity cycling, even without energy deficit, may reduce pTAG concentrations. However, the effect was significantly lower than that of the same exercise with a concomitant exercise-induced energy deficit. Although previous studies investigating the effect of 'intensity' as a mediator of the hypotriacylglycerolaemic effect of exercise have consistently concluded that total energy expenditure and not intensity of the exercise bout is of primary importance[28, 37, 86], at least in adults, the intensity used in these studies was much lower than that of the aforementioned cycling study[50]. The importance of intensity or 'type of exercise' as a mediator of the hypotriacyglycerolaemic effect also comes from recent studies, which showed that the energy expenditure threshold for resistant[29, 45, 52, 53] or intermittent exercise[46] may be less than that for aerobic of low or moderate intensity (Table 2), although energy deficit remains necessary[58].

It may be hypothesized that this difference is attributed to different underlying mechanisms of different modes of exercise. However, at least in the fasting state, recent studies in our laboratory have shown that a bout of high-intensity interval aerobic exercise or resistance exercise may reduce fasting plasma TAG the next day in a similar magnitude and via a similar mechanism as moderate-intensity aerobic exercise of almost twice the energy expenditure[87–89, 147]. These data suggest that the underlying mechanism for the hypotriacylglycerolaemic effect of exercise, albeit the same, is triggered by a lower volume of exercise (or is manifested more readily) when exercise is of high intensity or resistance. Alternatively, methodological problems in estimating energy expenditure during different types of exercise (resistance, very high intensity or intermittent), or higher excess post-exercise oxygen consumption (EPOC) may also account for these results[90]. This is particularly relevant to sprint-interval exercise. Studies involving sprint-interval exercise did not account for energy expenditure during warm-up and during resting periods between sprints (unloaded cycling)[50, 55, 64]. Moreover, a very recent study found that only 2 minutes (4 × 30 s) of sprint-interval cycling elicits 24-h of oxygen consumption (VO_2; and hence energy expenditure) similar to that of 30 min of continuous cycling at ~70 % maximal VO_2 (VO_{2max})[91]. Therefore, in studies in which PPL was investigated the day after sprint-interval exercise[50, 55, 64], underestimations of exercise energy expenditure due to long-lasting EPOC in addition to non-estimation of energy expenditure during warm-up and resting periods, may explain differences between the effects on PPL of this type of exercise versus continuous walking[64], and may suggest that, in the aforementioned cycling study with energy compensation[50], subjects remained in negative energy balance even in the energy-compensation trial.

Combinations of different types of exercise[19] or exercise accompanied with energy restriction[41, 56] have also been shown to reduce PPL (Table 2). In more detail, two recent

studies have shown that aerobic exercise of low intensity and hence low energy expenditure has hypotriacylglycerolaemic effects when combined with mild-to-moderate dietary energy restriction to augment energy deficit[41, 56]. In another study, 30 min running along with 45 min resistance exercise reduced pTAG concentrations[19], presumably due to the augmentation of total exercise energy expenditure[46, 51].

Another possible mediator of the hypotriacylgycerolaemic effect of exercise may be the type of meal. Recent studies investigated the effects of exercise on TAG responses to a moderate- rather than high-fat meal (Table 2), in an attempt to better illustrate real conditions and follow recent dietary guidelines. Although it may be speculated that the effect of exercise would be less intense under a lesser fat load, results from these studies seem not to support this hypothesis, since ~15 kJ/kg (half of the required energy expenditure for a high-fat meal, Table 2) has been shown to be an adequate exercise expenditure to reduce postprandial response to a moderate-fat meal[27, 62, 63, 66]. Similar results were also found when studying responses to a high-carbohydrate meal[67]. The interaction of the type of meal with exercise has also been confirmed in a recent study, in which exercise produced lower TAG responses to an olive-based high-fat meal compared with the effects of no exercise on TAG responses to a butter-based high-fat meal[92]. These results suggest that the combination of exercise and high-monounsaturated fatty acid diet may be a more effective strategy to control PPL than each intervention alone.

All the aforementioned studies have been carried out in healthy populations. Although reducing pTAG concentrations is important to prevent CVD, interventions that lower pTAG concentrations are considerably more valuable in populations with increased risk for CVD, such as those with obesity, the metabolic syndrome, diabetes and hypertriacylglycerolaemia in the fasting state[93].

It seems that, for those with obesity and the metabolic syndrome, less energy expenditure per kg of body mass or even less total energy expenditure[71, 74] is required to reduce pTAG concentrations, whether consuming a high- or moderate-fat meal (Table 3). In a recent study, similar total energy expenditure and, hence, less energy expenditure per kg of body mass produced similar, if not greater, reduction in PPL in obese compared with normal-weight adolescents[43]. Therefore, exercise may be an effective strategy to reduce an important CVD risk factor in obesity and the metabolic syndrome, even in adolescents. In addition, a recent study showed that, in overweight/obese men, moderate aerobic exercise reduces TAG responses to an ad libitum rather than a fixed meal given the next day[75]. In more detail, subjects in this study did not compensate for the exercise-induced energy expenditure by increasing their energy intake during the ad libitum meal, which means that the energy deficit necessary for lowering PPL may be achieved in real life conditions, at least in the short term.

Considering the beneficial effects of physical activity on insulin sensitivity[94], and the inverse association between insulin sensitivity and PPL[95, 96], it may be speculated that exercise would lower PPL to a similar or even greater extent in those with type II diabetes. The only two studies in those with type II diabetes failed to show significant improvement of PPL the day after exercise, although information on energy expenditure was not provided[97, 98]. However, similar exercise regimens[42] in healthy populations did have hypotriacylglycerolaemic effects, suggesting that exercise may not be the intervention of choice for such populations, or that some other type of exercise/deficit or greater energy expenditure/deficit may be effective. Indeed, if improvement in insulin sensitivity is the desired result, exercise energy expenditure greater than 3.5–4.0 MJ is needed to lower TAG concentrations; however, this was only investigated in the fasting, rather than the postprandial, state[99]. Nevertheless, exercise-induced reduction in PPL has been

documented even in the absence of improvement in insulin sensitivity in healthy populations[28, 37, 41, 56, 79]. In addition, in one of the aforementioned studies involving those with type II diabetes, exercise improved insulin sensitivity without affecting PPL[97], suggesting that exercise-induced TAG-lowering is not mediated by an increase in insulin sensitivity[79].

Collectively, these data suggest that several modulators may influence the effect of exercise on pTAG concentrations. Energy expenditure/deficit may be the most important, particularly for aerobic exercise of low to moderate intensity; however, recent data suggest the possibility of interactions between exercise energy expenditure/deficit, mode of exercise, type of meal, time between exercise and meal, and the target group.

4 Mechanisms Underlying the Hypotriacylgycerolaemic Effect of Acute Exercise

The mechanisms responsible for the TAG-lowering effect of acute exercise are only partly understood, particularly with regard to the postprandial period. The hypotriacylglycerolaemic effect of prolonged moderate-intensity exercise has been partially attributed to enhanced muscle LPL activity[80, 100–102]. Increased LPL-mediated hydrolysis likely results in increased clearance of triacylglycerol-rich-lipoprotein (TRL)-TAG across skeletal muscle in postprandial states[33], presumably in order to replenish intramuscular TAG stores depleted by prior exercise[101, 103]. Studies that evaluated whole-body endogenous[85, 88, 104] and exogenous[105, 106] TAG clearance the day after a prolonged bout of moderate-intensity exercise found significantly augmented removal rates of plasma TAG compared with resting conditions, while hepatic secretion of very low-density lipoprotein (VLDL)-TAG and secretion of intestinal-derived TAG were unchanged in men[38, 88, 104, 106] or, for VLDL-TAG, decreased in women[85]. Moreover, a growing body of evidence suggests that the effect of exercise on pTAG concentrations corresponds to lowered VLDL-TAG and not chylomicron-TAG concentrations[38].

The increased VLDL-TAG clearance the day after exercise cannot be attributed solely to increases in skeletal muscle LPL mass or activity, since exercise-induced hypotriacylglycerolaemia is not always accompanied by LPL changes[34, 36, 63, 107]. We hypothesize that increased TAG clearance the day after exercise may be a secondary result to the observed reduction in VLDL-apolipoprotein-B100 secretion[104], and/or changes in VLDL size[104, 106]. Reduction in hepatic secretion of VLDL particles or VLDL-TAG may result in the observed more efficient LPL-mediated chylomicron clearance after exercise[106, 108, 109], due to diminished competition between VLDL and chylomicrons for LPL-mediated hydrolysis in the fed state[110–112]. In addition, at least in men, the liver after exercise secretes the same amount of TAG in fewer VLDL particles relative to resting conditions, meaning that the liver secretes fewer, but TAG-richer and hence larger, nascent VLDL[104, 113]. In a recent kinetic study, the $VLDL_1$-apolipoprotein-B secretion rate was unchanged after exercise; however, exercise increased TAG enrichment of $VLDL_1$ particles and tended to increase $VLDL_1$ particle size[106]. The removal of TAG from the core of TAG-richer (and larger) VLDL particles is more efficient than TAG removal from TAG-poorer (and smaller) VLDL, due to the greater affinity for LPL of peripheral tissues[112], resulting in accelerated whole-body LPL-mediated VLDL-TAG hydrolysis[113]. Finally, recent evidence suggest that increases in blood flow to previously exercised muscles may play a role in exercise-induced reduction in PPL, presumably by increasing hydrolysis of TAG by LPL within capillaries, especially in the face of an exercise-induced increase in insulin sensitivity[70].

5 Effect of Exercise Training on Postprandial Lipaemia

Several cross-sectional studies confirm that PPL is lower in physically active adults compared with their sedentary counterparts. In more detail, early studies have shown that athletes exhibit lower PPL than sedentary subjects matched for age, body weight[114, 115] and fasting TAG concentrations[115]. Studies using an intravenous lipid tolerance test indicated that the protective effect of intense exercise training is attributed to accelerated TAG clearance from the circulation[115, 116]. A more recent study showed that active (participated in at least 90 min of structured vigorous physical activity per week and 30 min of moderate or greater physical activity 5 days/week) middle-aged men had lower (p = 0.057) TAG responses to a high-fat meal than their sedentary counterparts[117]. Later research revealed that even recreationally active (3–5 bouts of non-intense aerobic exercise per week) men and women exhibit lower PPL than sedentary subjects matched for age, body weight and fasting lipid concentrations[118].

However, a limitation of these early studies is that PPL was investigated close enough to the last bout of exercise (12–36 h) in trained subjects, confounding the effect of training status and acute exercise. When participants were asked to abstain from exercise for 60 h prior to measuring PPL, the difference between active (endurance and sprint/strength trained) and inactive counterparts was abolished[81], suggesting that the effect of exercise training is transient and lasts for ~2 days after the last exercise bout. Moreover, two recent studies failed to reveal differences in pTAG concentrations between aerobically or combined aerobically/anaerobically trained and inactive counterparts, even though subjects were instructed to refrain from exercise for only 24 h before the test meal[119, 120].

Additional support for the transient effect of exercise comes from detraining as well as interventional studies. Interrupting training for 60 h[121] or 6–6.5 days[121, 122] increases PPL in endurance-trained subjects. However, it should be noted that no data were given on the effect of detraining on body mass, and since subjects were asked to follow the same diet on the day(s) preceding each PPL assessment, energy surplus[123] and/or increases in body mass[42, 124–127] after detraining may have masked training effects on PPL. Unfortunately, to the best of our knowledge, no study has yet investigated the effects of exercise detraining with a concomitant reduction in energy intake (equivalent to reduction in energy expenditure) on PPL. We believe that this information would be of great importance, not only because it would distinguish exercise effects from those of energy balance, but also because training interruption is a common phenomenon in real-life conditions (e.g., during holidays, athletes during non-training periods, etc.)

Interventional studies have shown that 12 weeks of aerobic training (either walking or running) does not influence PPL, when it is measured 48 h[82, 84], 60 h[83] or 9 days[83] after detraining[82, 83]. These data, along with data from previously described intravenous fat tolerance tests in trained and untrained subjects[115, 116] suggest that the mechanism responsible for the hypotriacylglycerolaemic effect of exercise training is the same, i.e. derived from that of acute exercise (i.e., increased TAG clearance). However, more studies are needed to investigate whether this also applies to resistance or high-intensity exercise training, since recent studies in our laboratory, albeit in the fasting state, showed that high-intensity aerobic interval training reduces VLDL-TAG concentrations via a different mechanism than that of a single-bout of this type of exercise[147], i.e., decreases hepatic VLDL-TAG secretion without affecting clearance[128].

Nevertheless, all the aforementioned studies have been carried out in healthy adult populations. It may be speculated that the effects of exercise training would be more robust, or would persist for a longer period in individuals susceptible to postprandial lipid

dysmetabolism, such as those with obesity and diabetes[95, 129], older populations[130] or smokers[131, 132]. To the best of our knowledge, no study has yet investigated the effects of exercise *training per se* on PPL in a non-healthy population (i.e., without the well known mediators of changes in body weight or body composition[42, 124–127, 133]), such as those with obesity and diabetes. The only relevant study failed to show an effect of a 4-month aerobic exercise training programme (40 min cycling on a cyclometer, 3 times per week) on TAG concentrations at 6 h after the consumption of a high-fat meal, in healthy subjects and subjects with type II diabetes[134]. Unfortunately, effects on mean or peak pTAG concentrations, TAG AUC or iAUC (e.g., the common expressions of PPL, see Sect. 1) were not reported. Regarding the effect of exercise training in older populations, a recent study showed that active older adults exhibit lower PPL than their inactive counterparts, even though subjects were instructed to refrain from exercise for 48 h before the test meal[135]. Moreover, 6 months of aerobic exercise training reduced mean TG response to a high-fat meal in older men and women[136]. On the other hand, the same training protocol did not significantly influence PPL in older overweight men and women, even though subjects were instructed to refrain from exercise for only 24–36 h before the test meal[137]. In addition, exercise training does not seem to improve smokers' PPL, since a recent study showed no significant differences in pTAG concentrations of trained (>2 h per week of structured exercise for a minimum of 12 months) compared with untrained normolipidaemic smokers, even though subjects were instructed to refrain from exercise for only 24 h before the test meal[138]. Whether more exercise is needed to control PPL in smokers is currently unknown.

Collectively, these data suggest that, at least for young healthy non-smoking adults and for aerobic exercise, training transiently reduces PPL; therefore, exercise sessions should be frequent, on a daily or day-to-day basis, to maintain this clinically significant improvement. More studies are needed to investigate (1) the effects of training in non-healthy populations including more age groups (e.g., adolescents, elderly); (2) the effects of different types of training (e.g., resistance, high intensity, etc.); and (3) the relative effects of training and energy balance.

6 Clinical Implications and Recommendations for Further Research

This review has shown that acute continuous aerobic exercise (~2–2.5 MJ or more) combined with energy deficit may transiently reduce TAG responses to a high-fat meal, therefore reducing the risk for CVD[2]. Such moderate energy deficit may be achieved through ~90 min of brisk walking or bicycling. Alternatively, the combination of low-intensity aerobic exercise plus diet[41, 56] or resistance plus aerobic exercise[19] seems to be equally effective. We believe this outcome is clinically significant, since it gives the opportunity to select the most practical and feasible intervention for each individual. For sedentary individuals who may have difficulty exercising at higher intensity, the combination of aerobic exercise of low energy expenditure with mild-to-moderate dietary energy restriction, which is in accordance with current recommendations for body weight control in adults[139, 140], could be a more realistic goal. Moreover, the combination of running with resistance exercise complies with American College of Sports Medicine guidelines[141] and is fairly representative of the programmes offered by many fitness centers in most Western countries.

Additionally, exercise does not need to be a single continuous bout but instead could be spread out throughout the day, since the benefits of intermittent compared with continuous exercise on TAG metabolism are equal or possibly even greater[46]. Furthermore, if a prudent diet (with moderate fat intake, high in MUFA) is followed, exercise may be of less load, while those following a high-fat diet must exercise more. Therefore, it is up to the

subject's preference and ability which type of exercise regimen (± diet) to follow in order to attenuate PPL. However, since training studies have shown that the effect of exercise is transient, exercise should be included as a part of daily living and not performed only occasionally.

In a non-healthy population such as those with obesity, acute exercise combined with energy deficit is effective in lowering pTAG concentrations. However, those with diabetes, who suffer from postprandial dysmetabolism, and may therefore benefit greatly from such interventions, do not seem to benefit from acute low-to-moderate aerobic exercise, at least that of such energy expenditure magnitude. Whether other types of exercise or energy deficit (e.g., resistance exercise or diet) are effective in such populations is currently unknown. Moreover, as seen in Tables 1, 2 and 3, the elderly have not been studied, even though PPL increases with age[130, 142], presumably due to decreases in LPL activity and hence reduced TRL-TAG clearance[142]. Since most studies in adults show that exercise increases TRL-TAG clearance[88, 104, 105, 115, 116], the elderly may benefit from exercise, as some training studies have already shown[135, 136]. Additionally, the effectiveness of exercise in reducing pTAG concentrations in a population with established CVD, and therefore the importance of such interventions in secondary prevention of CVD, is currently unknown.

Another important outcome of this review is the necessity for physical activity control when evaluating hypotriacylglycerolaemic interventions (e.g., pharmaceutical, lifestyle or diet). In addition, as a general clinical guideline, we may propose physicians take into account physical activity when assessing patients' biochemical results.

Finally, elucidating the mechanisms responsible for the hypotriacylglycerolaemic effect of exercise is imperative and clinically useful in order to (1) propose or develop appropriate TAG-lowering drugs or diet that may act synergistically with the exercise; and (2) propose the appropriate intervention to treat particular TAG dysmetabolism (e.g., propose high-intensity interval aerobic training that lowers VLDL-TAG production[128] in populations with high levels of VLDL-TAG secretion, such as obese men[143], or acute resistance exercise that increases VLDL-TAG clearance in populations with low levels of VLDL-TAG clearance, such as obese women[143] and, presumably, the elderly[142]). However, such studies in the postprandial state involve methodological difficulties, since steady state is preferred for tracer studies (which is the main method used to study TAG kinetics).

7 Conclusion

Exercise may attenuate PPL, an important cardiovascular risk factor. The exercise energy expenditure threshold for acute effects seems to be ~2–2.5 MJ, or ~30 kJ/kg of body weight or less in an obese population or a population with the metabolic syndrome; even less with moderate-fat meals, resistance or intermittent exercise, although more studies are needed to confirm the advantage of such types of exercise. Moreover, prolonged aerobic exercise (i.e., exercise training) is effective in reducing PPL, but the effect is abolished with detraining. More studies are needed to investigate possible dose-response and plateau effects, the effect of combinations of different types of exercise, as well as the threshold of exercise energy expenditure in those with diabetes and other vulnerable populations. Finally, investigation of mechanisms underlying these effects would increase our knowledge and may be helpful in the clinical setting in selecting appropriate interventions for individuals.

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Table 1

Studies on the acute (<4 h) hypotriacylglycerolaemic effect of exercise in a healthy and non-healthy population[a]

Sex; (n)	Subjects; important characteristics	Type of Exercise	Regimen	Time between exercise cessation and meal	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
High fat meal											
Exercise with energy deficit											
Aerobic: *continuous*											
M (13)	Active	CAE	240 min walking at 25 % VO_{2max}	1 h	59	4.6	45	3.5	NA	NS	[16]
M (13)	**Active**	**CAE**	**90 min walking at 65 % VO_{2max}**	**1 h**	**59**	**4.6**	**54**	**4.2**	**NA**	**−39**	[16]
M (20)	**Moderate active**	**CAE**	**40 min jogging at 72.5 % VO_{2max}**	**30 min**	**30**	**2.1**	**34**	**2.3**	**−18**	**NA**	[17]
M (35)	**OW/OB, MS**	**CAE**	**51 min walking at 60–70 % VO_{2max}**	**0 min**	**20**	**2.1**	**17**	**1.8**	**−13**	**−32**	[15]
Aerobic: *intermittent*											
M (20)	**Moderate active**	**IAE**	**40 min intense jogging at 99.7 % VO_{2max} (3 min exercise – 1.5 min recovery)**	**30 min**	**30**	**2.1**	**33**	**2.3**	**−15**	**NA**	[17]
Resistance											
M (10)	Resistance trained	RE	90 min resistance exercise at 80 % 12 RM	1 h	21	1.6	14	1.1	+48	+89	[18]
Combination											
M (12)	**Recreational active or trained**	**RE+CAE**	**45 min resistance exercise at 95 % 10 RM +30 min running**	**4 h**	**46**	**3.7**	**41**	**3.3**	**−15**	**NA**	[19]
Moderate fat meal											
Exercise with energy deficit											
Aerobic: *continuous*											
M (16)	Sedentary	CAE	90 min walking at ~50 % VO_{2max}	0 min	38	2.6	32	2.2	NS	NS	[20]
M (13)		CAE	60 min walking at 60 % VO_{2peak}	0 min	30	2.4	27	2.1	NS	NS	[21]
W (13)		CAE	60 min walking at 60 % VO_{2peak}	0 min	27	1.6	23	1.4	NS	NS	[21]
M (16)	Sedentary	CAE	60 min walking at ~50 % VO_2 max	0 min	26	1.8	22	1.5	NS	NS	[20]
M (9)	OW/OB, MS	CAE	60 min walking at 60 % VO_{2peak}	0 min	25	2.3	22	2.0	NS	NS	[21]
W (9)	OW/OB, MS	CAE	60 min walking at 60 % VO_{2peak}	0 min	22	1.7	18	1.4	NS	NS	[21]

Sex; (n)	Subjects; important characteristics	Type of Exercise	Regimen	Time between exercise cessation and meal	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
M (11)	Sedentary	CAE	45 min cycling at 62 % HR_{max} (~39% VO_{2max}) [d]	0 min	18	1.3	15	1.1	NS	NS	[22]
M (16)	Sedentary	CAE	30 min walking at ~50 % VO_{2max}	0 min	13	0.9	11	0.7	NS	NS	[20]
M (12)	Untrained	CAE	30 min cycling at 48 % VO_{2max}	30 min	12	0.9	10	0.7	NS	NS	[23]
M (12)	Untrained	CAE	30 min cycling at 37 % VO_{2max}	30 min	9	0.7	7	0.5	NS	NS	[23]
M (12)	Untrained	CAE	30 min cycling at 26 % VO_{2max}	30 min	6	0.5	4	0.3	NS	NS	[23]
Aerobic: *intermittent*											
M (10)	**Recreational active**	**IAE**	**6×5 min (8:30, 10:00, and 11:30 a.m. and 1:00, 2:30, and 4:00 p.m.) jogging at 70 % VO_{2max}**	**25 min after the first and the third bout; (test meal 9:00 a.m. and 12:00 p.m)**	**25**	**1.8**	**23**	**1.6**	**−10**	**−22**	[24]

[a] For clarity, bold studies reported significant reduction in postprandial TAG responses, while unbolded studies did not.

[b] Most studies reported GEEE values rather than net exercise-energy expenditure/ED. For these studies ED was calculated by subtracting values for energy expenditure during rest from the reported corresponding GEEE values. For studies reporting only net exercise-energy expenditure, GEEE was calculated by adding values for energy expenditure during rest to the reported corresponding net exercise-energy expenditure values.

[c] Percentage of the reduction is presented if it was given, or it is calculated if mean values were given.

[d] For comparison purposes, intensity is also presented as % VO_{2max} estimated as previously described[25].

CAE continuous aerobic exercise, *ED* energy deficit, *GEEE* gross exercise energy expenditure, *HR_{max}* maximal heart rate, *IAE* intermittent aerobic exercise, *M* men, *MS* metabolic syndrome, *NA* not assessed, *NS* not significant – p>0.05, *OB* obese, *OW* overweight, *RE* resistance exercise, *RM* repetition maximum, *TAG AUC* area under the triacylglycerol concentration versus time curve in the postprandial state, *TAG iAUC* incremental TAG iAUC, *VO_{2max}* maximal oxygen consumption, *VO_{2peak}* peak oxygen consumption, *W* women.

Table 2

Studies on the delayed (>12 h but <24 h) hypotriacylglycerolaemic effect of exercise in a healthy population [a]

Sex (n)	Subjects' important characteristics	Type of exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
High fat meal										
Exercise with energy deficit										
Aerobic: *continuous*										
M (8)	**Recreational active or trained**	**CAE**	**120 min running at 65 % VO_{2max}**	***96***	**7.2**	***88***	**6.6**	**−34**	**NA**	[33]
B (9)	**Recreational active**	**CAE**	**90 min walking at 60 % VO_{2max}**	**69**	**4.3**	**48**	**3.9**	**−32**	**R**	[28]
B (9)	**Recreational active**	**CAE**	**180 min walking at 30 % VO_{2max}**	**67**	**4.2**	**48**	**3.4**	**−33**	**R**	[28]
M (12)	**Recreational active or trained**	**CAE**	**90 min running at 60 % VO_{2max}**	***65***	**4.9**	***59***	**4.4**	**−18**	**−22%**	[31]
M (8)	**Recreational active**	**CAE**	**90 min cycling at 60% VO_{2max}**	***60***	**4.5**	***54***	**4.0**	**−28**	**−42%**	[34]
W (9)	**Trained**	**CAE**	**90 min walking at 60 % VO_{2max}**	**55**	**3.4**	**49**	**3.0**	**−30**	**R**	[35]
W (9)	**Active, premenopausal**	**CAE**	**120 min walking at 50 % VO_{2max}**	**52**	**3.2**	**44**	**2.7**	**−23**	**−42%**	[36]
B (12)	**Recreational active**	**CAE**	**90 min walking at 60 % VO_{2max}**	***51***	**3.5**	***44***	**3.0**	**−26**	**−31%**	[37]
M (11)	**Recreational active or trained**	**CAE**	**90 min walking at 60 % VO_{2max}**	**45**	**3.4**	**39**	**2.9**	**−25**	**−27%**	[38]
M (12)	**Recreational active**	**CAE**	**60 min exercise at 60 % VO_{2max}**	***41***	**3.1**	***37***	**2.8**	**−19**	**ns**	[39]
M 10	**Recreational active**	**CAE**	**60 min jogging at 60 % VO_{2max}**	**40**	**3.1**	**36**	**2.8**	**NS**	**R**	[40]
W (13)	**Untrained**	**CAE**	**90 min walking at 60 % VO_{2max}**	**39**	**2.3**	**33**	**1.9**	**−16**	**R**	[35]
W 6	**Sedentary, Premenopausal**	**CAE**	**90 min walking at 60 % VO_{2peak}**	**39**	**2.4**	**30**	**1.8**	**−23**	**ns**	[41]
M (10)		**CAE**	**90 min walking at 50 % VO_{2max}**	***39***	**2.9**	***32***	**2.4**	**−25**	**−25%**	[42]
M (10)	**Adolescents**	**CAE**	**60 min walking or slow jogging at 65 % VO_{2max}**	**38**	**2.5**	**34**	**2.2**	**−17**	**ns**	[43]
W (11)	**Postmenopausal, moderate active**	**CAE**	**90 min walking at 65 % VO_{2max}**	***33***	**2.1**	***27***	**1.7**	**−20**	**R**	[44]
B (12)	Recreational active	CAE	90 min walking at 30 % VO_{2max}	*25*	1.7	*19*	1.3	NS	NS	[37]
B (14)	Recreational trained	CAE	90 min walking at 30–45 % VO_{2max}	22	1.6	16	1.2	NS	NA	[45]
B (18)	Sedentary	CAE	30 min continuous running at 60 % VO_{2max}	14	1.0	12	0.9	NS	NS	[46]
Aerobic: *intermittent*										

Sex (n)	Subjects' important characteristics	Type of exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
M (8)	Adolescents	IAE	6 × 10 min treadmill exercise at 75 % VO_{2peak} (10 min rest between each bout)	49	2.2	44	2.0	−20 NS; p= 0.07	NS	[47]
M (11)	**Adolescents**	**IAE**	**6 × 10 min jogging at 55 % VO_{2peak} (10 min rest between each bout)**	**38**	**2.0**	**34**	**1.7**	**−16**	NS	[48]
M (8)	**Adolescents**	**IAE**	**6 × 10 min treadmill exercise at 53 % VO_{2peak} (10min rest between each bout)**	**34**	**1.5**	**30**	**1.3**	**−24**	NS	[47]
M (10)	**Adolescents recreational active**	**IAE**	**4 × 15min uphill walking at 60 % VO_{2peak} (3 min rest between each block)**	**32**	**2.0**	**27**	**1.7**	**−14**	NS	[49]
M (11)	**Adolescents**	**IAE**	**3×10 min treadmill exercise at 55 % VO_{2peak} (10 min rest between each bout)**	**19**	**1.0**	**17**	**0.9**	**−13 NS; p = 0.06**	NS	[48]
B (12)	**Recreationally active**	**IAE**	**18 min sprint interval cycling (4× [30 s very high-intensity cycling + 4 min cycling without resistance])**	**17**	**1.2**	**16**	**1.1**	**−21**	NS	[50]
B (18)	**Sedentary**	**IAE**	**3 × 10 min running at 60 % VO_{2max} (20 min rest between each bout)**	**14**	**1.0**	**12**	**0.9**	NS	**−27**	[46]
Resistance: *continuous*										
W (5)	**Recreationally active but untrained, premenopausal**	**RE**	**95 min moderate-intensity whole-body resistance exercise**	**53**	**2.9**	**47**	**2.6**	**−24**	**NA**	[30]
M (11)	Recreational active, not resistance trained	RE	90 min resistance exercise at 80 % 10 RM	27	2.3	21	1.8	NS	NS	[51]
B (14)	**Recreational weight trained**	**RE**	**90 min resistance exercise at 10 RM**	**23**	**1.7**	**18**	**1.3**	**−14**	**NA**	[45]
M (10)	**Resistance trained**	**RE**	**90 min resistance exercise at 100 % of 8 RM**	**21**	**1.8**	**16**	**1.4**	**−35**	NS	[52]
M (10)	Resistance trained	RE	90 min resistance exercise at 50 % of 8 RM	19	1.6	13	1.1	−26 NS, p = 0.052	NS	[52]
M (10)	**Recreational experienced in weight lifting**	**RE**	**80 min resistance exercise at 12 RM**	**18**	**1.4**	**13**	**1.0**	**−24**	**−45**	[29]
M (10)	**Recreational experienced in weight lifting**	**RE**	**40 min resistance exercise at 12 RM**	**10**	**0.8**	**7**	**0.6**	**−20**	NS	[29]
M (9)	**Sedentary untrained**	**RE**	**26 min resistance exercise at 6 RM**	**8**	**0.6**	**6**	**0.5**	**−12**	NS	[53]
Resistance: *intermittent*										
M (24)	**Active with some participating in regular resistance training**	**IRE**	**5 × 45 min intermittent (throughout the day) resistance exercise at 30–40 % 1 RM**	**67**	**5.1**	**46**	**3.5**	**−12**	**−18**	[54]
Combination										

Sex (n)	Subjects' important characteristics	Type of exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
M (8)	**Recreational active**	**CAE+IAE**	**90 min cycling at 70 % VO_{2peak} followed by 10 × 1 min sprints (1 min rest between each sprint)**	**76**	**6.3**	**70**	**5.8**	**−41**	**−47**	[55]
M (12)	**Recreational active or trained**	**RE+CAE**	**45 min resistance exercise at 95% of 10 RM +30 min running**	**46**	**3.7**	**41**	**3.3**	**−26**	**NA**	[19]
W (6)	**Sedentary, Premenopausal**	**CAE+D**	**100 min walking at 30 % VO_{2peak} + energy restriction of 1 MJ**	**23**	**1.4**	**36**	**2.1**	**−19**	NS	[41]
W (8)	**Sedentary, Premenopausal**	**CAE+D**	**100 min walking at 30 % VO_{2peak} + energy restriction of 1.4 MJ**	**22**	**1.4**	**38**	**2.4**	**−19**	NS	[56]
Exercise without energy deficit										
Aerobic: *continuous*										
W (6)	White	CAE	90 min walking at 60 % VO_{2max} + energy replacement	17	1.2	0	0	NS	NA	[57]
W (6)	**African American**	**CAE**	**90 min walking at 60 % VO_{2max} + energy replacement**	**16**	**1.2**	**0**	**0**	**−62**	**NA**	[57]
Aerobic: *intermittent*										
B (12)	**Recreationally active**	**CAE**	**18 min sprint interval cycling (4 × [30 s very high-intensity cycling + 4 min cycling without resistance]) + energy replacement**	**14**	**1.0**	**0**	**0**	**−12**	NS	[50]
Resistance										
B (10)	Resistance trained	RE	90 min at 75 % of 1 RM + energy replacement	39	2.6	0	0	NS	NA	[58]
B (10)	Resistance trained	RE	50 min at 75 % of 1 RM + energy replacement	21	1.4	0	0	NS	NA	[58]
B (10)	Resistance trained	RE	20 min at 75 % of 1 RM + energy replacement	9	0.6	0	0	NS	NA	[58]
Combination										
M (9)	Recreational active	CAE	90 min cycling at 70 % VO_{2peak} + followed by 10 × 1 min sprints (1 min rest between each sprint) + energy/carbohydrate replacement	76	6.3	0	0	NS	NS	[55]
Moderate fat meal										
Exercise with energy deficit										
Aerobic: *continuous*										
W (8)	**Rowers**	**CAE**	**80 min rowing at 55 % of maximal aerobic power**	**68**	**4.6**	**63**	**4.2**	**−35**	NS	[59]

Sex (n)	Subjects' important characteristics	Type of exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
B (8)		CAE	90 min treadmill exercise at 65% HR_{max} (~43 % VO_{2max}) [d]	40	2.4	34	2.1	−23 NS; $p = 0.053$	NS	[60]
M (9)		**CAE**	**60 min cycling at 70–75 % HR_{max} (~44 % VO_{2max})** [d]	**31**	**2.6**	**27**	**2.3**	**−26**	NS	[61]
M (10)	**Recreational active**	**CAE**	**30 min running at 70 % VO_{2max}**	**24**	**2.0**	**23**	**1.8**	**−22**	**−31**	[62]
M (15)	**Recreational active**	**CAE**	**30 min walking at 40 % VO_{2max}**	**15**	**1.1**	**13**	**1.0**	**−16**	NS	[27]
M (12)	**Recreational active**	**CAE**	**30 min cycling at 65 % HR_{max} (~44% VO_{2max})** [d]	**13**	**0.9**	**11**	**0.8**	**−30**	**−33**	[63]
M (9)	Recreational active	CAE	30 min walking at VO_2 20 ml/kg (~38–47 % VO_{2max}) [e]	13	1.0	11	0.9	NS	NS	[64]
B (14)	Recreational active 45–63 years	CAE	30 min walking at 53 % VO_{2max} (every day for 3 days + the morning before the test meals)	12	0.9	10	0.7	NS	NS	[65]
M (15)		**CAE**	**30 min walking at 60 % HR_{max} (~36% VO_{2max})** [d]	**10**	**0.7**	**8**	**0.5**	**−18**	**NA**	[66]
Aerobic: *intermittent*										
M (10)	**Recreational active**	**IAE**	**10×3 min running at 70% VO_{2max} (30 min rest between each bout)**	**25**	**2.0**	**23**	**1.9**	**−24**	**−32**	[62]
M (15)	**Recreational active**	**IAE**	**10 × 3 min (30 min rest between each bout) walking at 40 % VO_{2max}**	**15**	**1.1**	**13**	**1.0**	**−16**	NS	[27]
M (9)	**Recreational active**	**IAE**	**5 × (30 s maximal sprints and 4 min unloaded cycling between each sprint)**	**5**	**0.4**	**5**	**0.4**	**−18 NS; $p = 0.056$**	**−34**	[64]
High carbohydrate meal										
Exercise with energy deficit										
Aerobic: *continuous*										
W (10)	**Sedentary**	**CAE**	**60 min cycling at 60 % VO_{2peak}**	**20**	**1.2**	**16**	**1.0**	**R**	**NA**	[67]

[a] For clarity, bold studies reported significant reduction in postprandial TAG responses, while unbolded studies did not.

[b] Most studies reported GEEE values rather than net exercise-energy expenditure/ED. For these studies ED was calculated by subtracting values for energy expenditure during rest from the reported corresponding GEEE values. For studies reporting only net exercise-energy expenditure, GEEE was calculated by adding values for energy expenditure during rest to the reported corresponding net exercise-energy expenditure values. Italics indicate that subjects' mean weight was not given and a body mass of 75 kg for men and 65 kg for women was used arbitrarily for the calculations.

[c] Percentage of the R is presented if it was given, or it is calculated if mean values were given.

[d] For comparison purposes, intensity is also presented as % VO_{2max} estimated as previously described [25].

[e] For comparison purposes, intensity is also presented as corresponding % VO_{2max} of untrained males [68, 69]

B both sexes, *CAE* continuous aerobic exercise, *D* hypocaloric diet, *ED* energy deficit, *GEEE* gross exercise energy expenditure, *IAE* intermittent aerobic exercise, *IRE* intermittent resistance exercise, HR_{max} maximal heart rate, *M* men, *NA* not assessed, *NS* not significant – $p > 0.05$, *R* reduction, *RE* resistance exercise, *RM* repetition maximum, *TAG AUC* area under the triacylglycerol concentration versus time curve in the postprandial state, *TAG iAUC* incremental TAG AUC, VO_{2max} maximal oxygen consumption, VO_{2peak} peak oxygen consumption, *W* women.

Table 3

Studies on the delayed (>12 h but <24 h) hypotriacylglycerolaemic effect of exercise in a non-healthy population [a]

Sex (n)	Subjects' Important Characteristics	Type of Exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
High fat meal										
Exercise with energy deficit										
Aerobic: *continuous*										
M (8)	**OW, inactive**	**CAE**	**90 min walking at 60 % VO_{2max}**	**40**	**3.7**	**36**	**3.3**	**−29**	**−29**	[26]
M (8)	**OW, inactive**	**CAE**	**90 min walking at 60 % VO_{2max}**	**38**	**3.5**	**34**	**3.1**	**−22**	**−34**	[70]
M (10)	**MS (OW/OB, IR, HTAG), sedentary**	**CAE**	**60 min jogging at 70 % VO_2max**	**31**	**3.0**	**27**	**2.6**	**NA**	**−39**	[71]
M (8)	**OB, Adolescents**	**CAE**	**50 min walking or slow jogging at 6 5% VO_{2max}**	**29**	**2.5**	**26**	**2.2**	**−27**	NS	[43]
M (10)	**MS (OW/OB, IR, HTAG), sedentary**	**CAE**	**60 min jogging at 60 % VO_{2max}**	**28**	**2.7**	**24**	**2.3**	**NA**	**−31**	[71]
M (10)	**MS (OW/OB, IR, HTAG), sedentary**	**CAE**	**60 min walking at 60% VO_{2max}**	**28**	**2.5**	**23**	**2.1**	**NA**	**−33**	[72]
M (10)	**OW/OB, IR, HTAG, sedentary**	**CAE**	**60 min jogging at 60 % VO_{2max}**	**27**	**2.7**	**23**	**2.3**	**NA**	**−33**	[73]
M (10)	**MS (OW/OB, IR, HTAG), sedentary**	**CAE**	**45 min walking at 60 % VO_{2max}**	**21**	**1.9**	**18**	**1.6**	**NA**	**−31**	[72]
M (14)	OW/OB, MS, inactive	CAE	60 min walking at 65% VO_{2peak}	19	2.1	17	1.8	NA	NS	[74]
M (14)	**OW/OB, MS, inactive**	**CAE**	**100 min walking at 40 % VO_{2peak}**	**19**	**2.1**	**14**	**1.6**	**NA**	**−27**	[74]
M (10)	**MS (OW/OB, IR, HTAG), sedentary**	**CAE**	**60 min jogging at 40 % VO_{2max}**	**18**	**1.8**	**14**	**1.4**	**NA**	**−30**	[71]
M (10)	MS (OW/OB, IR, HTAG), sedentary	CAE	30 min walking at 60 % VO_{2max}	14	1.3	12	1.1	NA	NS	[72]
Aerobic: *intermittent*										
M (14)	OW/OB, MS, inactive	IAE	2 × 30 min walking at 65 % VO_{2peak} (separated by 3–5 h)	19	2.1	16	1.8	NA	NS	[74]
Moderate fat meal										
Exercise with energy deficit										
Aerobic: *continuous*										
M (8)	**OW, inactive**	**CAE**	**90 min walking at 60 % VO_{2max}**	**41**	**3.8**	**36**	**3.4**	**−26**	**−32**	[26]

Sex (n)	Subjects' Important Characteristics	Type of Exercise	Regimen	GEEE (kJ/kg)	GEEE (MJ)	ED (kJ/kg)[b]	ED (MJ)[b]	TAG AUC (%)[c]	TAG iAUC (%)[c]	References
M (10)	**OW sedentary to moderately active**	**CAE**	**90 min walking at 50 % VO_{2max}**	**39**	**3.5**	**33**	**3.0**	**−27**	NS	[75]
M (13)	**OW/OB**	**CAE**	**90 min walking at 50 % VO_{2peak}**	**NA**	**3.3**	**NA**	**2.8**	**−14**	NS	[76]
M (7)	**OW/OB, sedentary**	**CAE**	**30 min cycling at 60 % HR_{max} (~36 % VO_{2max})** [d]	**10**	**0.8**	**8**	**0.7**	**−18**	NS	[77]
M (10)	**Centrally OB**	**CAE**	**30 min cycling at 62 % HR max (~39% VO_{2max})** [d]	**9**	**0.9**	**8**	**0.7**	**−9**	**NA**	[78]
M (15)		**CAE**	**30 min cycling at 60 % HR_{max} (~36 % VO_{2max})** [d]							
Aerobic: *intermittent*										
M (7)	**OW/OB, sedentary**	**IAE**	**10 × 3 min cycling at 60 % HR_{max} (~36 % VO_{2max}) d (30 min rest between each bout)**	**10**	**0.8**	**8**	**0.7**	**−15**	NS	[77]
Exercise without energy deficit										
Aerobic: *continuous*										
M (14)	OW/OB	CAE	90 min walking at 50 % VO_{2peak} + energy replacement	**NA**	**3.3**	NA	0	NS	NS	[76]
High carbohydrate meal										
Exercise with energy deficit										
Aerobic: *continuous*										
W (10)	**OW/OB, sedentary**	**CAE**	**60 min cycling at 60 % VO_{2peak}**	**19**	**1.6**	**15**	**1.3**	**R**	**NA**	[67]

[a] For clarity, bold studies reported significant reduction in postprandial TAG responses, while unbolded studies did not.

[b] Most studies reported GEEE values rather than net exercise-energy expenditure/ED. For these studies ED was calculated by subtracting values for energy expenditure during rest from the reported corresponding GEEE values. For studies reporting only net exercise-energy expenditure, GEEE was calculated by adding values for energy expenditure during rest to the reported corresponding net exercise-energy expenditure values.

[c] Percentage (%) of the reduction is presented if it was given, or it is calculated if mean values were given.

[d] For comparison purposes, intensity is also presented as % VO_2max estimated as previously described [25].

CAE continuous aerobic exercise, *ED* energy deficit, *GEEE* gross exercise energy expenditure, *HR_{max}* maximal heart rate, *HTAG* hypertriacylglycerolaemics, *IAE* intermittent aerobic exercise, *IR* insulin resistant, *M* men, *MS* metabolic syndrome, *NA* not assessed, *NS* not significant – p > 0.05, *OB* obese, *OW* overweight, *TAG AUC* area under the triacylglycerol concentration versus time curve in the postprandial state, *TAG iAUC* incremental TAG AUC, *VO_{2max}* maximal oxygen consumption, *VO_{2peak}* peak oxygen consumption, *W* women