Increasing meal frequency in combination with exercise mitigates postprandial triacylglycerol

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Meal frequency, exercise and postprandial lipemia

Abstract

Background: This study examined how manipulating meal frequency, with and without exercise, affects postprandial triacylglycerol (TAG). Methods: Fourteen sedentary men completed four 2-day trials in a non-counterbalanced random crossover order: (i) consumption of one large high fat milkshake without exercise (1-CON); (ii) consumption of two smaller high fat milkshakes without exercise (2-CON); (iii) consumption of one large high fat milkshake with exercise (1-EX); and (iv) consumption of two small high fat milkshakes with exercise (2-EX) – total energy intake was standardized across trials. On Day 1, participants rested (1-CON and 2-CON) or walked briskly for 60 minutes (1-EX and 2-EX). On Day 2, participants consumed either a single large high-fat milkshake (75% fat) (1-CON and 1-EX) for breakfast or two smaller iso-energetic milkshakes (2-CON and 2-EX) for breakfast and lunch. Plasma TAG were measured fasting and for 7 hours after breakfast. Results: Peak incremental TAG was 30% lower on 2-EX than 1-CON ($P = .041; d = 0.38$). Postprandial TAG increased more rapidly in the first 4 hours in 1-CON than other trials, but at 6 hours TAG was exaggerated in 2-CON compared with 1-CON. Conclusions: Increasing meal frequency after exercise, without altering overall fat intake, attenuates postprandial TAG.
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Introduction

High non-fasting triacylglycerol (TAG) concentrations are an independent risk factor for cardiovascular diseases. The postprandial state represents the habitual metabolic state for most individuals consuming regular meals each day with the fasted state only occurring in the first hours of the morning. Strong experimental evidence supports continuous aerobic exercise as a strategy to mitigate postprandial TAG responses to a high fat test meal (>50 g fat). However, in some studies where meals contained a more moderate fat content (30-50 g fat), exercise was reported to have a smaller or no effect on postprandial TAG, although this finding is inconsistent. Timing of meal ingestion in relation to exercise may be one factor explaining the difference among studies. Alternatively, the diminished ability of exercise to impact TAG following meals of moderate fat content may stem from the dose-response relationship between fat intake and the magnitude of the postprandial response, with the consumption of smaller doses of fat attenuating the overall response to a single meal.

Based on the preceding argument individuals could potentially minimize TAG disturbances by increasing meal frequency and decreasing absolute fat consumption at each meal. Such a strategy might suggest little capacity for exercise to further lower postprandial TAG. However, TAG clears slowly from the circulation and rarely returns to fasting concentrations between meals consumed across the waking day. Thus, simply consuming less fat and increasing meal frequency may not attenuate diurnal TAG concentrations. Moreover, increased meal frequency may actually exaggerate TAG by provoking the release of TAG stored in enterocytes or the lymphatic circulation from the previous meal. Alternatively, lower insulin concentrations with smaller meals could evoke less activity in adipose tissue.
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lipoprotein lipase (LPL) - the enzyme responsible for TAG clearance - than large meals, thereby
hydrolyzing circulating TAG at a slower rate for uptake into adipocytes.\textsuperscript{13}

Some studies have investigated the effects of exercise on postprandial TAG responses to
multiple meals.\textsuperscript{2,7,12,15,16} These demonstrate that exercise reduces postprandial TAG after two or
three meals of moderate or high fat content compared with no-exercise control trials. In these
investigations much of the reduction in TAG was after the second or third meal which coincides
with the period of maximal TAG extraction by LPL.\textsuperscript{2,7,12,15} What is presently uncertain is how
the addition of exercise interacts with changes in meal frequency to influence postprandial TAG.

No available studies appear to have compared the role of exercise in response to a single large
high fat meal versus smaller meals equivalent in fat and energy content. Certainly, if the aim of
lifestyle interventions is to minimize diurnal disturbances in postprandial TAG concentrations
then it is pertinent to examine the interaction between exercise and meal frequency.

Thus, the present study examined how manipulating meal frequency, combined with and
without exercise, affects postprandial TAG concentrations. To do this we used a simple design
of a single high fat meal, known to substantially elevate postprandial TAG, versus two smaller
isoenergetic fat meals provided 3 hours apart. We hypothesized that compared with consuming a
single large high fat meal, dividing fat consumption into two smaller meals in the presence of
prior exercise would mitigate the postprandial TAG to the greatest extent compared with two
small meals without preceding exercise or a single large high fat meal with exercise.

Material and methods

Recruitment and screening
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All procedures took place after university Institutional Review Board approval. Male volunteers were recruited by advertisement and provided written informed consent after an explanation of the procedures involved before testing began. Participants were screened and recruited if they met the following criteria: (i) 21 to 35 years; (ii) self-reported body mass was stable in the previous two months; (iii) body mass index < 25 kg/m^2; (iv) engaging in structured exercise < 2 times per week for < 20 minutes per bout by self-report; (v) no personal history of cardiovascular disease, diabetes mellitus, metabolic disease or dyslipidemia; (vi) were not dieting; (vii) were not vegetarian; (viii) were non-smokers; (ix) consumed alcohol < 3 times per week with < 3 drinks per time by self-report; (x) were not using medications that influence lipid or carbohydrate metabolism; and (xi) had no joint problems or injuries to the lower body that affected walking.

Stature was measured using an electronic wall-mounted stadiometer to the nearest 0.1 cm (Seca242, Seca, Germany), body mass was recorded using a scale to the nearest 0.1 kg (Mettler-Toledo ID1 Plus, Mettler-Toledo S.E.A Pte Ltd, Singapore) and umbilical waist circumference was measured using a flexible measuring tape to the nearest 0.1 cm. Body composition was estimated via dual energy X-ray absorptiometry (Hologic Discovery QDR 4500W, Bedford, MA, USA) and seated blood pressure measured in duplicate using a manual sphygmomanometer (Empire N, Riester, Germany). Fourteen participants met the entrance criteria and completed all trials (Table 1).

Pre-testing

****Table 1 near here****
After screening, participants completed a walking peak oxygen uptake (peak VO₂) exercise test using a modified ramp protocol. Treadmill speed was constant throughout the test at 6.5 km/h; whereas the initial gradient was 1% and increased by 1% per minute until volitional exhaustion. Expired air was measured continuously throughout the test via a mouthpiece attached to an automated metabolic cart (Parvomedics MMS-2400, Parvomedics, Sandy, UT, USA). Heart rate was monitored using short-range telemetry (Polar RS400, Polar, Oulu, Finland) and ratings of perceived exertion (RPE) assessed periodically during the test. Data from the tests were used to establish the individual participant steady-state relationship between oxygen uptake and treadmill gradient when walking at 6.5 km/h for subsequent use in the main trials.

Main Trials

Each participant completed four 2-day trials in a non-counterbalanced random crossover manner determined from a simple random number generator: i) consumption of a single large meal without exercise (1-CON); ii) consumption of two smaller, isoenergetic meals without exercise (2-CON); iii) consumption of a single large meal with exercise (1-EX); and iv) consumption of two smaller, isoenergetic meals with exercise (2-EX). All trials were separated by a minimum of 3 days. Postprandial TAG concentrations rapidly increase in the absence of recent exercise and return to pre-exercise levels within 48-60 hours after the last bout of exercise performed. Thus, 3 days was considered sufficient washout to prevent any carry-over effects of exercise on subsequent trials. On Day 1 of their first visit participants prospectively recorded their food and drink intake, based on the portion size method, in a diary and, thereafter, replicated that food intake the day before each subsequent visit. Participants were asked to avoid
Day 1

On Day 1 participants arrived at the laboratory at 1700; for 1-EX and 2-EX they walked on a treadmill for 1 hour at 6.5 km/h and a gradient designed to elicit 60% of their peak VO₂. Similar exercise intensity and duration has caused reductions in postprandial TAG in a previous study.²⁰ Heart rate (monitored using short-range telemetry: Polar RS400, Polar, Oulu, Finland) and RPE¹⁸ were assessed every 5 minutes during walking. In contrast, for CON-1 and CON-2, participants sat in the laboratory for 60 minutes engaging in passive activities such as working or reading. At the end of the hour, they were fed a choice of one of two standardized evening meals from a commercial food chain (McChicken®, Fries and Coca-Cola®: 3.96 MJ, 19 g protein, 34 g fat and 142 g carbohydrate; or Filet-O-Fish®, Fries and Coca-Cola®: 3.74 MJ, 19 g protein, 30 g fat, 137 g carbohydrate) which they replicated on all subsequent trials. Nine participants chose the first meal and six the second as their option. Whilst the evening meal replaced the energy used in walking, the same relative energy deficit between the two exercise and no-exercise trials was maintained, similar to previous investigations.¹⁵,¹⁶ After the meal participants were asked to spend the remainder of the evening at home and not to consume any other food or drinks except plain water ad libitum.

Day 2

On Day 2 participants reported to the laboratory at 0800 where they lay in a supine position on a bed and a cannula was inserted into an antecubital or forearm vein. A fasting 6 mL
blood sample was taken after 10 minutes and participants then consumed either a large (1-CON, 1-EX) or small (2-CON, 2-EX) high fat test milkshake within 10 minutes. The milkshakes consisted of Magnolia milk, Emborg whipping cream and Haagen-Dazs vanilla ice cream. The single large milkshake provided 1.21 g of fat, 0.62 g of carbohydrate, 0.29 g of protein and 0.061 MJ per kg of body mass. Energy composition was 75% fat, 17% carbohydrate and 8% protein. For a 70 kg male, the meal provided 4.26 MJ (1014 kcal). On the 2-CON and 2-EX trials, a second small milkshake was provided 3 hours after the first. The two small milkshakes each provided the same relative composition, but half the energy of the single large fat milkshake (0.60 g fat, 0.31 g carbohydrate, 0.15 g protein and 0.031 MJ per kg of body mass). Two meals with the equivalent amount of fat have previously been shown to continuously elevate TAG over 5 hours. Further blood samples were taken each hour, for seven hours, after participants started to consume the first milkshake; the cannula was removed and participants went home after the final sample.

**Sample analysis**

Blood samples were collected into precooled EDTA vacutainers, separated in a refrigerated centrifuge (Rotina 420R, Andreas Hettich GmbH & Co., Tuttlingen, Germany) and the plasma stored at -80°C for later analysis. All samples were analysed for TAG on an Abbott Architect c4000 Clinical Chemistry Analyzer (Abbott Laboratories, Illinois, USA) using a glycerol phosphate oxidase method; intra-assay and inter-assay coefficients of variation were < 5%.

**Statistical justification**
Power analysis conducted using G*Power v3.1 suggested that for a within subject repeated measures ANOVA 14 participants would be sufficient to detect, with a power of 0.82 and 5% alpha, a small effect ($f = 0.34$) on postprandial TAG concentrations among trials.

**Calculations and statistics analysis**

Total area under the plasma TAG concentration *versus* time curve (AUC-TAG) was calculated using the trapezium rule$^{22}$ and the incremental area calculated using the same method after correcting for fasting concentrations (iAUC-TAG); no values recorded below fasting on all trials. Peak TAG was defined as the highest TAG concentration during each trial and time to peak TAG was recorded. Peak incremental TAG was calculated as the highest TAG concentration minus fasting TAG concentration and represents the highest postprandial increase in TAG. Peak and AUC responses are important indicators of the maximal increase in and magnitude of postprandial TAG, respectively, and have been used as indicators of clinical risk.$^{1,3,11}$

Data were analyzed using standard statistical software (SPSS Version 23.0, Chicago, IL) and checked for normality using Shapiro-Wilk tests. Pre-trial dietary data were compared using a one-way ANOVA. Exercise heart rate and RPE (log transformed) were compared between 1-EX and 2-EX trials using a paired t-test. Fasting and postprandial TAG data were not normally distributed and a natural log transformation applied to the data. Fasting TAG, AUC-TAG, iAUC-TAG, peak TAG, peak incremental TAG and time to peak TAG were compared among trials using one-way ANOVA. Postprandial TAG concentrations were compared between trials and over time using a two-way ANOVA. When a significant main effect of trial or a trial $\times$ time interaction presented, planned contrasts were conducted between the intervention trials (2-CON,
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1-EX, 2-EX) with control (1-CON). To determine if there were any confounding variables, the relationship of AUC-TAG, iAUC-TAG and peak TAG with BMI, waist circumference and body fat percentage was examined. The 95% confidence intervals for ratio differences (95% CI) derived from the log-transformed differences in geometric means are reported. Cohen’s $d$ was used as an estimate of effect size for significant differences; calculated by dividing the difference between the mean values of the intervention and control with the control trial (1-CON) standard deviation (SD). In the absence of a clinical anchor, absolute effect sizes of 0.00 – 0.19 were considered trivial, 0.20 – 0.49 as small, 0.50 – 0.79 as medium and $\geq 0.80$ as large. Data in text, tables and figures are reported as mean (SD) and reflect observed (not log transformed) values. Significance was set as $P \leq .05$.

Results

Pre-trial dietary intake

Energy intake on Day 1 was similar among trials: 1-CON: 8.1 (2.0) MJ; 2-CON: 7.5 (1.8) MJ; 1-EX: 8.0 (1.8) MJ; 2-EX: 8.3 (2.4) MJ; $P = .369$. There were no differences in intake of protein (1-CON: 58 (17) g; 2-CON: 59 (14) g; 1-EX: 63 (23) g; 2-EX 63 (20) g; $P = .549$), carbohydrate (1-CON: 272 (71) g; 2-CON: 244 (62) g; 1-EX: 260 (48) g; 2-EX 270 (80) g; $P = .368$) or fat (1-CON: 68 (20) g; 2-CON: 64 (18) g; 1-EX: 72 (24) g; 2-EX 71 (24) g; $P = .437$) among trials on Day 1.

Walking trials
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Mean heart rate (1-EX: 145 (16) b/min vs. 2-EX: 147 (16) b/min; \( t(13) = -0.778, P = .451 \)) and RPE (1-EX: 13 (2) vs. 2-EX: 13 (2); \( t(13) = 0.573, P = .576 \)) during the walking trials were similar.

**Fasting and summary TAG responses**

Fasting and summary postprandial TAG responses are presented in Table 2. Between trial differences in fasting TAG concentrations were small and non-significant (\( F(3,39) = 2.198, P = .104 \); all \( d \leq 0.27 \)). The mean AUC-TAG was lower by 14%, 17% and 18% on the 2-CON, 1-EX and 2-EX trials, respectively, than 1-CON (\( F(3,39) = 2.862, P = .049 \)). Planned contrasts showed smaller AUC-TAG for 1-EX (\( P = .041, d = 0.30 \)) and 2-EX (\( P = .036, d = 0.31 \)) trials but not the 2-CON (\( P = .139, d = 0.24 \)) trial compared with 1-CON. The iAUC-TAG was 17%, 25% and 29% lower on the 2-CON (\( d = 0.22 \)), 1-EX (\( d = 0.32 \)) and 2-EX (\( d = 0.37 \)) trials compared with 1-CON but there was no significant difference among trials (\( F(3,39) = 2.605, P = .065 \)). The AUC and iAUC for TAG did not correlate significantly with BMI (AUC: \( r = 0.170-0.406 \); iAUC: \( r = 0.081-0.341 \)), waist circumference (AUC: \( r = 0.057-0.284 \); iAUC: \( r = -0.048-0.177 \)) or body fat percentage (AUC: \( r = 0.111-0.433 \); iAUC: \( r = 0.012-0.414 \)) on any trial (all \( P > 0.05 \)).

Mean peak TAG concentrations were 18%, 20% and 23% lower on the 2-CON, 1-EX and 2-EX trials than 1-CON trial, respectively (\( F(3,39) = 3.030, P = .041 \)). Planned contrasts showed significantly lower peak TAG for 2-EX (\( P = .023, d = 0.37 \)) but not 2-CON (\( P = .070, d = 0.22 \)) and 1-EX (\( P = .067, d = 0.32 \)) trials compared with 1-CON. Similarly, peak incremental TAG (Figure 1) differed among trials (\( F(3,39) = 3.029, P = .041 \)) being 30% lower on the 2-EX (\( P = .027, 95\% \text{ CI: } 1.0 \) to 13.3, \( d = 0.38 \)) than 1-CON trial but not lower on the 2-CON (\( P = .092, \)
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95% CI: -0.9 to 10.3, \( d = 0.27 \) and 1-EX (\( P = .087 \), 95% CI: -1.0 to 12.5, \( d = 0.32 \)) trials. Peak TAG did not correlate significantly with BMI (\( r = 0.270-0.362 \)), waist circumference (\( r = 0.121-0.261 \)) or body fat percentage (\( r = 0.115-0.362 \)) on any of the trials (all \( P > 0.05 \)).

Time to peak TAG concentration differed among trials (\( F(3,39) = 5.395, P = .003 \), with an extended time to peak of \(~1 \) hour on 2-CON (\( P = .002 \)) and 2-EX (\( P = .010 \)) compared with 1-CON. When examining the no-exercise trials, 9 out of 14 participants took longer whilst 4 participants took the same time to reach peak TAG in 2-CON than 1-CON. One participant reached peak TAG in a shorter time on 2-CON than 1-CON.

****Table 2 near here****

****Figure 1 near here****

**Postprandial TAG**

Two-way ANOVA revealed a significant difference in plasma TAG concentrations among trials (main effect of trial, \( F(3,39) = 2.847, P = .050 \)). Planned contrasts supported lower TAG on the 1-EX (\( P = .045 \); 95% CI: 0.1 to 7.4) and 2-EX (\( P = .037 \); 95% CI: 0.3 to 7.5) trials compared with 1-CON, but not a significant difference between 1-CON and 2-CON (\( P = .126 \); 95% CI: -0.8 to 5.8). The TAG concentrations increased more rapidly in 1-CON over the first 4 hours compared with the other trials, but at 6 hours were elevated on 2-CON compared with 1-CON (trial × time interaction, \( F(21,273) = 6.200, P < .001 \)). Subsequent planned contrasts across individual time points supported these observations (Figure 2).

****Figure 2 near here****
Discussion

This study demonstrates that apportioning dietary fat into two small meals in the presence of prior exercise can reduce disturbances in postprandial TAG. Our data suggest that the reduction in postprandial TAG results from two factors. Firstly, less fat intake at the first meal in combination with exercise mitigated the early TAG response (1 hour) compared with a single large high fat meal. Moreover, the peak postprandial TAG was also attenuated, an effect not seen with a small first meal in isolation or when exercise preceded the single large high fat meal.

Secondly, exercise diminished the extended elevation and exaggeration in TAG that occurred in response to the second meal in the late postprandial period (6 hours) without exercise (2-CON).

Thus, the combination of smaller, more frequent fat intake along with recent exercise may represent the optimal strategy for individuals wishing to attenuate diurnal TAG.

Elevations in postprandial TAG are influenced by several factors including the amount of fat in a meal.\textsuperscript{3,5} Although increases in postprandial TAG have a direct dose-dependent relationship with the amount of fat consumed, simply apportioning fat into smaller, more frequent doses does not necessarily reduce the magnitude of TAG across the day as TAG concentrations typically do not return to fasting between meals.\textsuperscript{2,12,24} Moreover, successive meals lead to further exaggerations in TAG or biphasic peaks in TAG.\textsuperscript{2,12,15,16,24} This is likely from triggering the release or outflow of TAG in chylomicrons stored in enterocytes or the lymphatic circulation from the first meal, or alternatively displacing pre-formed chylomicrons by newly formed chylomicrons.\textsuperscript{14} The present study confirmed these observations by demonstrating that ingestion of a single large milkshake exaggerated TAG concentrations early in the postprandial period compared with a single small milkshake. Subsequent ingestion of a second
small milkshake after 3 hours prolonged TAG appearance in the circulation with higher
concentrations in the late postprandial period (6 hours). Thus, there was no overall difference in
the postprandial TAG response after consuming the single large and two small milkshakes as
evidenced by the summary TAG measures (AUC and iAUC) and postprandial comparison.

Individuals wishing to minimize TAG perturbations across the day cannot therefore necessarily
rely on the tactic of ingesting smaller amounts of fat across more meals.

The single large milkshake used here probably exaggerated the magnitude and duration
of the postprandial response compared with a normal dietary meal. Total fat consumed in the
single large milkshake equated to 85 g for a 70 kg male, which is equivalent to a daily intake for
many individuals. Most individuals typically consume much less fat than that used in
experimental studies with normal meals containing 20-40 g of fat in the usual daily pattern of
three to four meals, which is similar to the two smaller milkshakes used here. Moreover,
normal meal intake leads to smaller variations in TAG concentrations as opposed to the large
increases observed when a high fat meal is ingested. Nevertheless, the use of high fat meals
persists in experimental studies to establish differences in TAG concentrations among
individuals and in response to interventions. It should also be recognized that some
individuals do frequently consume large doses of fat. For example, in Singapore the average
reported fat intake for men is 103 g/day, but for men in the 90th percentile and above, reported fat
consumption is > 160 g/day, or > 50 g per meal based on a three meal intake frequency. Thus,
comparing a single large bolus of fat versus two smaller boluses of fat in this study is still
relevant.

The present study provides a clear experimental comparison between a single large and
two smaller doses of fat equivalent in energy. Future investigations nevertheless need to
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establish fat responses over several large versus small meals to mimic a full day. Dose response
studies show that very small amounts of fat, typically < 15g in a meal, have little impact on
altering circulating TAG but larger doses of fat increase TAG in a dose dependent manner.5,9,10
Thus, apportioning fat intake into very small amounts across many meals should limit
disturbances in TAG because each meal provides little challenge in terms of TAG clearance.
However, a previous study in eight obese women found that when they were given food, with a
total energy intake of 6.3 MJ (1500 kcal) and total fat intake of 1.3 MJ (315 kcal), in a three or
six meal pattern, the three meal pattern lead to significantly lower TAG concentrations over a 12
hour day.13 The authors suggested that the higher insulin concentrations observed in the three
meal pattern may have stimulated adipose tissue LPL activity which functions to hydrolyze
circulating TAG for subsequent uptake into the adipocytes. More investigations are needed to
properly characterize variations in TAG concentrations with multiple meals.

Continuous aerobic exercise is an effective strategy for reducing postprandial TAG in
response to a single high fat meal4,5 or multiple meals taken throughout the day2,12 and in the
present study exercise attenuated TAG concentrations to the single high fat meal as expected.
The novel finding was that exercise before the two small milkshakes also reduced peak
postprandial TAG, the 3-4 hours postprandial TAG, and mitigated the exaggeration in TAG seen
during the late postprandial period with two meals alone (6 hours). Aerobic exercise is thought
to reduce circulating TAG via improved clearance, by increasing skeletal muscle LPL activity,
and/or reducing TAG appearance in hepatic very low density lipoproteins.26

Although the mechanism for TAG reduction could not be elucidated here it is notable that
the reductions with the exercise trials were during the later postprandial period (≥3 hours). This
differs from investigations which have observed differences in fasting TAG with exercise,
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contributing to TAG attenuation during the early postprandial response (<3 hours) as well.\textsuperscript{16} Several previous studies, where two or three meals were fed after exercise, have reported a similar finding to ours, with reductions in TAG most notable after the second meal (≥3 h).\textsuperscript{2,12,15} That TAG attenuations with exercise are apparent late in the postprandial period corresponds with peak concentrations and the period of maximal TAG extraction from the circulation.\textsuperscript{12} In the present study, exercise was performed the evening before the meals. Skeletal muscle LPL activity peaks ≥ 8 hours after exercise and returns to baseline after approximately 24 hours.\textsuperscript{27} Thus, it is feasible that exercise-induced increases in LPL activity increased TAG clearance after the second meal when circulating TAG concentrations peaked. Another possibility is that insulin concentrations after the second meal were reduced with exercise.\textsuperscript{28} This could have increased skeletal muscle LPL activity, which is decreased by insulin,\textsuperscript{27} and subsequently led to attenuated postprandial TAG concentrations. However, we did not measure insulin and the relationship between insulin and postprandial TAG after exercise has been challenged.\textsuperscript{29}

Several limitations exist for this study. The aforementioned relevance of using high fat loads to examine postprandial TAG is questionable when many individuals consume meals containing smaller amounts of fat along with the use of liquid meals as a fat challenge. In addition, this study only used an experimental one versus two meal design shown to elevate TAG previously. Nevertheless, future studies need to move toward ecologically relevant designs where individuals consume a typical three meal pattern of daily foods with normal fat intakes as the standard control group. These studies should include mechanistic data (e.g. insulin, lipoprotein lipase activity) and be conducted in clinically relevant populations, such as individuals with overweight or obesity, in order to provide a better picture of how meal frequency and exercise interact to lower postprandial TAG.
Conclusion

In summary, dividing dietary fat into two small meals in the presence of previous exercise minimized peak postprandial responses and perturbations in postprandial TAG. Consumption of a smaller amount of fat attenuated early postprandial TAG concentrations in comparison with a large bolus of fat, but TAG concentrations were subsequently exacerbated after a second fat meal. The additional role of exercise appears to be in minimizing exaggerations in TAG resulting from ingestion of a second intake of fat. Future studies should examine how exercise interplays with smaller fat intakes spread frequently throughout the day on circulating TAG.

Acknowledgements

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Funding Source

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References
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Table 1. Physical characteristics of participants

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<td>Waist circumference (cm)</td>
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<td>Body fat (%)</td>
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<td>Peak oxygen uptake (mL/kg/min)</td>
<td>35.4 (4.7)</td>
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Values are mean (SD), n = 14.
Table 2. Fasting and summary postprandial triacylglycerol (TAG) measures over 7 hours on the four trials.

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<td>0.84 (0.30)</td>
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<td>AUC-TAG (mmol × 7h/L)</td>
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<td>iAUC-TAG (mmol × 7h/L)</td>
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<td>95% CI</td>
<td>-3.9 to 12.6</td>
<td>-0.9 to 18.5</td>
<td>-0.2 to 18.9</td>
<td>-0.2 to 18.9</td>
<td></td>
</tr>
<tr>
<td>Peak TAG (mmol/L)</td>
<td>3.04 (1.97)</td>
<td>2.49 (1.13)</td>
<td>2.43 (1.09)</td>
<td>2.33 (1.00)a</td>
<td>.041</td>
</tr>
<tr>
<td>95% CI</td>
<td>-0.5 to 12.0</td>
<td>-0.6 to 14.1</td>
<td>1.3 to 15.0</td>
<td>1.3 to 15.0</td>
<td></td>
</tr>
<tr>
<td>Time to peak TAG (h)</td>
<td>3.9 (0.7)</td>
<td>4.9 (0.9)a</td>
<td>4.1 (0.9)</td>
<td>4.6 (0.9)a</td>
<td>.003</td>
</tr>
<tr>
<td>95% CI</td>
<td>-15.1 to -3.5</td>
<td>-7.5 to 3.2</td>
<td>-11.9 to -1.2</td>
<td>-11.9 to -1.2</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean (SD), n = 14.

* Two-way repeated measures ANOVA.

† 95% confidence interval for the ratio differences.

a Significantly different from 1-CON (P ≤ .05).
Meal frequency, exercise and postprandial lipemia

Figure Captions

Figure 1. Mean (open rectangles) and individual peak incremental triacylglycerol (TAG) concentrations over 7 hours after a single high fat milkshake (1-CON, diamonds), two small high fat milkshakes (2-CON, squares), a single high fat milkshake taken after exercise (1-EX, triangles) or two high fat milkshakes taken after exercise (2-EX, rosettes). n = 14. *Significantly different 1-CON vs. 2-EX (P = .041).

Figure 2. Mean triacylglycerol (TAG) concentrations over 7 hours after a single high fat milkshake (1-CON, diamonds with solid line), two small high fat milkshakes (2-CON, squares with dotted line), a single high fat milkshake taken after exercise (1-EX, triangles with dashed line) or two high fat milkshakes taken after exercise (2-EX, rosettes with dot and dash line). Black arrows indicate milkshake timings. n = 14. Main effect of trial, P = .050: 1-CON vs. 1-EX, P = .045; 1-CON vs. 2-EX, P = .037. Main effect of time, P < .001. Trial × time interaction, P < .001: *, significantly different 1-CON vs. 2-CON (P ≤ .05); b significantly different 1-CON vs. 1-EX (P ≤ .05); c significantly different 1-CON vs. 2-EX (P ≤ .05).
Figure 1

254x190mm (96 x 96 DPI)
Figure 2

254x190mm (96 x 96 DPI)