Time-restricted feeding increases fat oxidation rate but not affect postprandial lipemia: a crossover trial

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Abstract

Background: Studies have revealed that time-restricted feeding affects the fat oxidation rate; however, its effects on the fat oxidation rate and hyperlipidemia following high-fat meals are unclear. This study investigated the effects of 5-day time-restricted feeding on the fat oxidation rate and postprandial lipemia following high fat meals.

Methods: In this random crossover experimental study, eight healthy male adults were included each in the 5-day time-restricted feeding trial and the control trial. The meals of the time-restricted feeding trial were provided at 12:00, 16:00, and 20:00. The meals of the control trial were provided at 08:00, 14:00, and 20:00. The contents of the meals of both trials were the same, and the calories of the meals met the 24-hour energy requirement of the participants. After 5 days of the intervention, the participants consumed high-fat meals on the sixth day, and their physiological changes were determined.

Results: The fasting fat oxidation rate ($p<0.001$) and postprandial fat oxidation rate ($p=0.019$) of the time-restricted feeding trial were significantly higher than those of the control trial. The 24-hour energy consumption and postprandial triglyceride, blood glucose, insulin, glycerol, and free fatty acid concentrations of the two trials showed no significant differences ($p>0.05$).

Conclusion: The results revealed that 5 days of time-restricted feeding effectively increased the fasting and postprandial fat oxidation rate, but it did not affect postprandial lipemia.

Introduction

Consuming high-fat meals increases the triglyceride (TG) level in blood plasma. Studies have discovered that large increases in postprandial TG concentration lead to high risks of cardiovascular diseases and metabolic syndrome (1). Compared with the fasting TG concentration, the postprandial TG concentration is a more precise predictor of the risks of cardiovascular diseases and metabolic syndrome (2).

Consuming high-fat meals increases the levels of biochemical substances in blood plasma, such as TG, free fatty acids, and remnant cholesterol. Studies have reported that these biochemical substances are major risk factors for metabolic syndrome, atherosclerosis, myocardial infarction, and coronary heart disease, all of which are associated with high mortality (3, 4). The high TG level after the consumption of high-fat meals can last for 6–8 hours. As three meals daily are typically consumed by the general population, high levels of TG may be constantly occurring in the body. Therefore, investigating methods to reduce the high TG level after eating high-fat meals is crucial for reducing the development of metabolic syndrome.

Studies have demonstrated that a single session of endurance exercise can effectively reduce the TG level after the consumption of high-fat meals (5-8). In addition, performing a single session of endurance exercise 8 hours before meals can effectively reduce the increase in TG after the consumption of high-fat meals, and the effect can last for 48–60 hours (9, 10). Endurance exercises effectively reduce the increase in postprandial TG because they increase lipoprotein lipase activity and insulin sensitivity, and

they reduce the release of very low-density lipoprotein by the liver (10). Recent studies have shown that increasing the fat oxidation rate after eating high-fat meals is crucial for reducing the postprandial TG level (11, 12); however, the results have remained inconsistent. Some studies have described that performing high-intensity interval training is positively correlated with decreases in the postprandial TG level (13), whereas other studies have discovered that the increased fat oxidation rate following high-fat meals did not affect the postprandial TG level (14).

Studies have discovered that time-restricted feeding can improve insulin sensitivity (15), increase the fat oxidation rate (16), and decrease the fasting TG level (17). However, whether time-restricted feeding can exert health benefits in terms of effectively reducing the increase in the TG level following high-fat meals remains unclear. This study investigated the effects of 5-day time-restricted feeding on the fat oxidation rate and postprandial lipemia after the consumption of high-fat meals.

Methods

Participants

This study recruited eight healthy male adults as research participants (age: 22 ± 1.3 yr, height: 170.1 ± 4.7 cm, weight: 75.4 ± 17.5 kg). All the participants had not undergone physical training; they did not exercise regularly; and they did not have any diseases that would prevent them from performing exercises, such as high blood pressure, hyperlipidemia, heart disease, joint disease, and osteoporosis. All the participants fully understood the experimental process before experiment initiation and were notified of the possible risks; they agreed to the terms of the experiment and provided their written consent. A similar number of participants and a similar recruitment method have been employed by this research team in the past. This study was approved by the Institutional Review Board of Jen-Ai Hospital (110-10) in Taiwan.

Design

This study used a crossover design for the experiment. The participants were divided into the time-restricted feeding trial and the control trial; the participants in the control trial did not practice intermittent fasting methods. The participants of both trials consumed the same meals for 5 days. The time-restricted feeding group used the 16:8 methods to practice intermittent fasting. The meals were provided at 12:00, 16:00, and 20:00. The meals of the control group were provided at 08:00, 14:00, and 20:00, but the consumption time was not limited. On the morning of the sixth day, all participants returned to the laboratory to consume high-fat meals, and their TG blood levels after the meal was determined. The participants were randomly assigned to different arms of the study to receive different treatments, and an interval of at least 14 days was maintained between the tests to avoid any effects of the preceding test on the succeeding test. Studies have discovered that 4 days of intermittent fasting effectively increased the fat oxidation rate and reduced blood glucose (18, 19). Therefore, 5 days of intermittent fasting should provide sufficient intervention time to stimulate fat oxidation rate changes.

Protocol

Pretest

In the pretest, gas analyzers (Vmax Series 29C, Sensor Medics, CA, USA) were used to assess the energy consumption of the participants while they were resting and performing nonmaximal intensity exercises for precisely calculating the daily calorie consumption of each participant. After the participants arrived at the laboratory, they wore heart rate monitors (Polar, Finland), and their energy consumption was examined using gas analyzers. Next, they quietly rested for 20 minutes in the supine position, and their resting heart rate and energy consumption were recorded. They then performed nonmaximal intensity exercises for measuring their energy consumption during low-intensity activities. The participants stood on a treadmill with a slope of 0° for 10 minutes to record their energy consumption during standing. The participants then walked or ran on the treadmill at five speeds, namely 1, 2, 3, 4, and 5 miles per hour. The participants maintained each speed for 3 minutes for examining the relationship between the energy consumption and heart rate of the participants during low-intensity activities.

After the pretest, the participants were asked to perform their normal daily life activities while wearing the heart rate monitor; their heart rates were recorded for 24 hours. The 24-hour heart rate, the regression curve of the heart rate, and energy consumption calculated in the pretest were used to calculate the total energy consumption of the participants. In the experiment, meals were adjusted so that the total calories of each meal met the 24-hour energy requirement of each participant. The method used to record energy consumption and the brand of heart rate monitors (Polar) used in this study have been described elsewhere (12, 20, 21). Moreover, 24-hour energy consumption was recorded the day before the experiment, and the calories of meals were adjusted to meet the 24-hour energy requirement of each patient. The same methods were repeated on the fifth day of the experiment to confirm the 24-hour energy consumption changes of the two trials.

Formal Experiment

The experiment was conducted over 6 days. On the first day, the participants arrived at the laboratory at 08:00 and quietly rested for 20 minutes in the supine position; gas analyzers were used to record their energy consumption. Subsequently, the participants were randomly allocated to the time-restricted feeding trial or the control trial. The meals of the time-restricted feeding trial were provided at 12:00, 16:00, and 20:00, and the participants were required to consume all the food during this time. The meals of the control trial were provided at 08:00, 14:00, and 20:00, but the consumption time was not limited. In addition to regular meals, a snack with approximately 200 calories was provided to the participants for consumption. The participants in the time-restricted feeding trial were only allowed to consume the snack from 12:00 to 20:00, whereas no restrictions were imposed on the control trial for snack consumption. The meals of the participants were provided by dietitians. Based on the results of the pretest, the calories of each meal met the daily energy requirement of the participants. The macronutrient consumption for TRF and CON were listed in table 1.

After experiment completion on the fifth day, the participants returned to the laboratory on the eighth day from 08:00 to 09:00. They rested for 10 minutes in the supine position, and gas analyzers were used to collect the gas data of the participants for 20 minutes. Next, a catheter was inserted into the forearm of each participant to collect fasting blood samples. After blood sample collection, the participants were provided with a specific high-fat meal. The participants rested quietly in the laboratory for 4 hours, and their blood lipid changes during this period were observed.

Oral fat tolerance test (OFTT)

All oral fat tolerance test (OFTT) meals were designed and provided by dieticians, as previously described (12, 22, 23). The meals included toast, butter, cheese, muesli, and cream. For every kg of the body weight of the participant, the meal provided 1.2 g of fat, 1.1 g of carbohydrate, 0.33 g of protein, and 16.5 kcal of energy. The nutritional information was obtained from the nutritional facts on food packages. During the experiment, the participants were required to consume the OFTT meal within 15 minutes.

Blood collection

In the experiment, a catheter (Venflon 20G, Sweden) was inserted into the vein of the forearm, and a three-way stopcock (Connecta Ltd., Sweden) was used to collect 10 mL of blood each time. Blood was collected before meals, 30 minutes after meals, and every hour after meals up to the fourth hour. After each session of blood collection, 10 mL of isotonic saline water was used to clean the catheter to avoid blood clotting in the catheter.

The collected blood was immediately placed in blood collection tubes containing ethylenediaminetetraacetic acid. A cell counter was used to analyze the hematocrit (Sysmax KX-21N, Kobe, Japan). After the analysis, the blood was centrifuged for 20 minutes at $500 \times g$ at 4°C . Blood plasma was obtained and was immediately placed in a -80°C refrigerator for preservation and future biochemical analysis.

Blood biochemical analysis

TG, blood glucose, free fatty acid, and glycerol levels in blood were analyzed using an automated biochemistry analyzer (7020, Hitachi, Japan) and commercial reagents (GOD-PAP method, Randox, Ireland). The insulin concentration in blood plasma was analyzed using a chemiluminescence immunoassay analyzer (Elecsys 2010, Roche Diagnostics, Basel, Switzerland) and commercial reagents (Roche Diagnostics). The fat and carbohydrate oxidation rates were calculated using the following formula(24):

$$\text{Fat oxidation (g/min)} = 1.695 \times \text{VO}_2 - 1.701 \times \text{VCO}_2$$

$$\text{Carbohydrate oxidation (g/min)} = 4.585 \times \text{VCO}_2 - 3.226 \times \text{VO}_2$$

Statistical Analysis

All the data in this study are presented as average $\pm$ standard deviation. First, the normality of the data was tested using the Shapiro–Wilk test. The fasting fat oxidation rate, blood biochemical values, and areas under the fat oxidation rate curve and the TG curve were analyzed using the paired sample t test. The postprandial fat oxidation rate and blood biochemical values were analyzed using two-way ANOVA with repeated measures. If the data were significant, the Bonferroni method was used to perform post hoc comparisons. Using G*power 3, to achieve an alpha value of 5% and a power of 0.8, the sample size of eight was considered sufficient for this study. The significance level was set at $\alpha < 0.05$.

Result

Fasting plasma concentrations

The fasting fat oxidation rate of the time-restricted feeding trial was significantly higher than that of the control trial ($p < 0.001$). The concentrations of TG, blood glucose, insulin, free fatty acids, and glycerol between the two trials were not significantly different ($p > 0.05$) (Table 1).

Postprandial fat oxidation rate and area under the curve

The postprandial fat oxidation rate of the time-restricted feeding trial was significantly higher than that of the control trial (trial $\times$ time, $p = 0.019$; trial, $p = 0.004$; time, $p = 0.256$; Figure 1A). The area under the postprandial fat oxidation rate curve of the time-restricted feeding trial was significantly larger than that of the control trial ($p = 0.02$, Figure 1B).

Postprandial TG concentration and area under the curve

Postprandial TG concentrations were not significantly different between the time-restricted feeding and control trials (trial $\times$ time, $p = 0.531$; trial, $p = 0.850$; time, $p < 0.001$; Figure 2A). The areas under the TG concentration curve were also not significantly different between the two trials ($p = 0.956$, Figure 2B).

Postprandial blood biochemical indices

The plasma glucose concentration was not significantly different between the two trials (trial $\times$ time, $p = 0.712$; trial, $p = 0.456$; time, $p = 0.023$; Figure 3A). Figure 3B indicates that the insulin concentration was not significantly different between the trials (trial $\times$ time, $p = 0.564$; trial, $p = 0.558$; time, $p = 0.027$; Figure 3B). Figure 3C indicates that the free fatty acid concentration was not significantly different between the trials (trial $\times$ time, $p = 0.598$; trial, $p = 0.976$; time, $p < 0.001$; Figure 3C). Figure 3D indicates that the glycerol concentration was not significantly different between the trials (trial $\times$ time, $p = 0.925$; trial, $p = 0.064$; time, $p < 0.001$; Figure 3D).

Discussion

In this study, meals were provided that met the 24-hour energy requirement of the participants for 5 days, and the intervention was time-restricted feeding conducted at different parts of the day. The results

revealed that time-restricted feeding effectively increased the fasting fat oxidation rate or the fat oxidation rate after the consumption of high-fat meals. However, the increased fat oxidation rate exerted no effects on the TG level following high-fat meals, 24-hour energy consumption, or reactions of blood biochemical substances.

This study confirmed that 5 days of time-restricted feeding effectively increased the fasting fat oxidation rate and the fat oxidation rate after the consumption of high-fat meals. Studies applying time-restricted feeding have mostly used interventions with a duration of a few weeks, and the results showed that time-restricted feeding decreased body weight and improved metabolism (25, 26). Studies that have utilized short-term time-restricted feeding have discovered that 4 days of early time-restricted feeding (consuming dinner before 15:00) effectively increased the fat oxidation rate and reduced appetite; however, it did not affect 24-hour energy consumption (18). A similar study demonstrated that 4 days of early time-restricted feeding improved the 24-hour blood glucose balance (19). In contrast to the aforementioned studies, this study used late time-restricted feeding (consuming dinner before 20:00). In addition, all the meals were prepared by the research team and were directly provided to the participants; hence, in this study, the diet of the participants could be more precisely controlled, instead of the participants consuming their own food. This study discovered that late time-restricted feeding produced results similar to those achieved by early time-restricted feeding. In addition, compared with the control trial, time-restricted feeding did not affect the 24-hour energy metabolism of the time-restricted feeding trial, and time-restricted feeding effectively reduced the fasting fat oxidation rate and the fat oxidation rate after the consumption of high-fat meals. However, the glycerol and free fatty acid concentrations of the two trials were not different. Therefore, the exact mechanism through which time-restricted feeding increased the fat oxidation rate was unknown.

In this study, time-restricted feeding could effectively increase the fasting fat oxidation rate and the postprandial fat oxidation rate, but it did not affect the TG level after the consumption of high-fat meals. This result indicated that 5 days of short-term time-restricted feeding resulted in a shorter action time for the increase in the fat oxidation rate; thus, the postprandial TG level was not affected. Chiu et al. (2019) used three high-fat meals per day to change the fat oxidation rate of participants; although this method effectively increased the fat oxidation rate, it did not affect the TG level after the consumption of high-fat meals (14). This study demonstrated that the fat oxidation rate of the time-restricted feeding trial was significantly higher than that of the control trial; however, glycerol and free fatty acid concentrations were not significantly different. Therefore, although short-term time-restricted feeding effectively increased the fat oxidation rate, it did not affect the postprandial TG reaction.

Another possible reason for the intervention not affecting the TG level after the consumption of high-fat meals is that 5-day time-restricted feeding did not affect blood glucose and insulin concentrations. Studies have suggested that insulin sensitivity is a major factor that affects the TG level after the consumption of high-fat meals (27). Compared with late time-restricted feeding, early time-restricted feeding reduced postprandial blood glucose concentration to a higher extent in a previous study (28). However, that study did not limit the calorie intake, and participants were 55 years old and were at a high

risk of diabetes. In comparison, this study provided all the meals to the participants during the experiment to ensure that the calorie intake of all the participants was equal. In addition, this study controlled the calorie intake to ensure that it met the 24-hour energy requirement of the participants, and the results revealed that fasting and postprandial blood glucose concentrations and the insulin concentration were unaffected. Accordingly, the insulin sensitivity of the participants remained unchanged; thus, the postprandial TG level was unaffected.

The main limitation of this study was the calculation of 24-hour energy consumption. The 24-hour energy consumption was determined through calculation, rather than through measurement by methods such as those using the respiratory chamber. Calculations would not be as accurate as actual measurements. Studies have tested 24-hour energy consumption and yielded robust results using methods similar to that used in the present study (12, 20, 21). Therefore, we believe that this method is still credible. In addition, this study did not collect data on the muscle glycogen or intramyocellular TG levels and did not analyze lipoprotein lipase activity. Therefore, this study could not explain the main reason for the nonsignificant effect of 5-day time-restricted feeding on postprandial TG.

Conclusion

This study discovered that consuming meals with the same amount of calories for 5 days and using time-restricted feeding as the intervention can effectively increase the fasting fat oxidation rate and the fat oxidation rate after the consumption of high-fat meals. However, the increased fat oxidation rate did not increase the TG level after the consumption of high-fat meals. Future studies can extend the duration of time-restricted feeding and can investigate how long-term time-restricted feeding affects the TG level after the consumption of high-fat meals.

Declaration

Ethics approval and consent to participate: This study was approved by the Institutional Review Board of Jen-Ai Hospital (110-10) in Taiwan.

Consent for publication

Not applicable.

Availability of data and materials

All relevant materials are presented in the present manuscript.

Competing interests

The authors declare that they have no competing interests.

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Author Contributions: C.-H.C. (Chih-Hui Chiu) carried out the experiment, blood analysis and assisted the manuscript preparation. C.-H.C. (Che-Hsiu Chen) and M.-H.W. assisted the data analysis and manuscript preparation. Y.-C.H, Y.-Z.L, and B.-W.L assisted the experimental design, data analysis and manuscript preparation. All authors haveread and agreed to the published version of the manuscript.

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Tables

Table 1. The macronutrient consumption for TRF and CON

	TRF	CON	P value
Carbohydrate (g)	340.4±28.0	342.2±31.4	0.904
Protein (g)	124.7±11.3	128.7±31.4	0.599
Fat (g)	74.9±7.9	72.1±10.2	0.552
Total calorie (Kcal)	2533.6±240.9	2532.0±272.0	0.983

Values are mean SD, n = 8. TRF, time-restricted feeding trial; CON, control trial.

Table 2. The participants physiological information and fasting plasma biochemistry.

	TRF	CON	<i>p</i> value
24 h energy expenditure (Kcal)	2541.5±1406.2	2388.6±1370.4	0.419
TG (mmol/L)	1.16±0.4	1.45±0.7	0.077
Glucose (mmol/L)	70.6±5.3	71.0±3.2	0.793
Insulin (pmol/L)	7.8±3.2	7.6±4.5	0.931
NEFA (mmol/L)	0.38±0.17	0.35±0.08	0.655
Glycerol (μ mol/L)	33.4±17.0	27.9±10.5	0.190
Fat oxidation (g/min)	0.14±0.05	0.04±0.03	<0.001
Carbohydrate oxidation (g/min)	0.21±0.12	0.26±0.14	0.167

Values are mean SD, n = 8. TRF, time-restricted feeding trial; CON, control trial; TG, triglyceride; NEFA, Non-esterified fatty acids.

Figures

Figure 1

The postprandial fat oxidation over the 4 hours (A) and the fat oxidation rate area under the curve in 4 hours (B). * TRF was significantly higher than those for the CON.

Figure 2

The postprandial triglycerides concentrations over the 4 hours (A) and the TG area under the curve in 4 hours (B). * mean TRF was significantly higher than those for the CON.

Figure 3

The postprandial glucose concentrations over the 4 hours (A), insulin concentrations over the 4 hours (B), glycerol concentrations over the 4 hours (C) and non-esterified fatty acids concentrations over the 4 hours.