International Journal of Nutrition and Food Sciences
2026, Vol. 15, No. 1, pp. 16–24
https://doi.org/10.11648/j.ijnfs.20261501.13

Research Article

Coingestion of Fructose with Fat Cream Exacerbates Postprandial Lipidemia Primarily by Stimulating Endogenous Non-remnant Triglyceride Secretion

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Abstract

Aim: To investigate the effect of coingestion of fructose with fat cream on postprandial lipid metabolism in Japanese female university students, using remnant and non-remnant indices. Methods: Healthy young Japanese women (n=54) with apolipoprotein E phenotype 3/3 were enrolled. They participated on two occasions, ingesting either fat cream (OFTT cream™, Jomo, Japan; 1 g/kg as cream, 0.35 g/kg as fat) alone or fat cream with fructose (0.5 g/kg). Venous blood samples were collected before (0 h) and at 0.5, 1, 2, 4, and 6 h after ingestion. Results: After ingestion of fat cream alone, serum concentration of triglyceride increased at 2 and 4 h, but non-remnant triglyceride concentration did not rise during the experiment. In contrast, after ingesting fructose in combination with fat cream, both serum triglyceride and non-remnant triglyceride levels increased at 4 h and did not return to baseline by the end of the experiment. The ratio of remnant triglyceride to total triglyceride, and an index of particle size were higher in the fructose with fat cream than fat cream alone at 4 h after ingestion. Conclusion: Coingestion of fructose with fat cream, but not fat cream alone, exacerbated postprandial lipidemia primarily by stimulating endogenous non-remnant triglyceride secretion.

Keywords

Fat-Ingestion Test, Fructose, Postprandial Lipidemia, Remnant, Triglyceride

1. Introduction

Postprandial hyperlipidemia contributes to atherogenic dyslipidemia, characterized by hypertriglyceridemia, low high-density lipoprotein-cholesterol (HDL-C), and an increase in small, dense low-density lipoprotein (LDL) particles [1]. This dysmetabolic state results from the excessive postprandial accumulation of triglyceride-rich lipoprotein (TRL) particles via two distinct secretory pathways: (a) the production and secretion of very low-density lipoprotein (VLDL) in the liver and chylomicron (CM) secretion in the small intestine, and (b) defective clearance of TRL from the circulation [2]. Elevated levels of plasma triglyceride (TG), TRL, and TRL remnants are causally

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Received: 15 January 2026; **Accepted:** 27 January 2026; **Published:** 20 February 2026

related to an increased risk of cardiovascular events including myocardial infarction, stroke, and aortic valve stenosis and all-cause mortality [3, 4].

An epidemiological study in Japan found that a 1 mmol/L (88.6 mg/dL) increase in non-fasting TG raised the risk of coronary artery disease (CAD) by 1.29 times in men and 1.42 times in women. The CAD risk increased at non-fasting TG levels of 115 mg/dL or higher, even after adjusting for HDL-C [5]. Fasting TG levels as low as 100 mg/dL can also be associated with a cluster of metabolic abnormalities, including the accumulation of TRL and remnants [6], as well as increased cardiovascular disease risk [7, 8]. In addition, we showed that in young women, the ingestion of a moderate amount of fat cream induces oxidative stress by increasing superoxide radicals and hypochlorite ions, with the degree of oxidative stress positively correlated with the postprandial rise in TG [9]. Therefore, even when fasting TG levels are within the normal range, it is necessary to monitor the increase in TG levels after meals.

It is widely accepted that excessive fructose consumption can negatively impact human health. Diets that derive more than 15% of their energy from fructose, compared to glucose, are associated with increased fasting and postprandial TG concentrations [10, 11]. We previously showed that fructose-containing beverages combined with fat cream delay the clearance of both exogenous and endogenous TRL, exacerbating postprandial lipidemia even in young women [12]. Moreover, soft drink consumption has been linked to a higher risk of ischemic stroke in Japanese women [13]. Furthermore, the World Health Organization has launched a major initiative urging countries to increase the real prices of sugary drinks by at least 50% by 2035 through health taxes, aiming to reduce chronic diseases and generate essential public revenue. Sugary soft drinks containing high-fructose corn syrup are major contributors to diseases such as heart disease, cancer, and diabetes, which are leading causes of death and disability worldwide [14].

In this study, we thoroughly examined the effects of the coingestion of fructose and fat cream on postprandial lipid metabolism, using the remnant and non-remnant indices we previously proposed [15]. For this purpose, we enrolled female university students, as this generation of women is thought to be among the highest consumers of high-fructose soft drinks and high-fat fast food, such as cola and hamburgers [16-18].

2. Methods

2.1. Subjects

Japanese female university students (n=54) with a normal ovarian cycle and apolipoprotein E phenotype 3/3 were enrolled. All participants were non-smokers, did not have any apparent acute or chronic illness, and were not taking any medication or dietary supplements. The subjects analyzed in this study are the same as those reported previously [15, 19]. This study received approval from the Institutional Review Board of Sugiyama Jogakuen University School of Life Studies (Nos. 2013-3, 2014-2, 22, and 23), and the subjects provided written informed consent. All procedures were conducted in accordance with the Helsinki Declaration of 1975, as revised in 1983.

2.2. Anthropometric and Body Composition Measurements

Body height was measured using standard methods. Waist circumference was assessed at the level of the umbilicus, while hip circumference was measured at the level of the greater trochanters. Body mass and composition, including visceral fat area (VFA), were analyzed using an 8-polar bioelectrical impedance method (InBody720, BioSpace, Tokyo, Japan). The body mass index (BMI) and waist-to-hip (W/H) ratio were calculated.

2.3. Experimental Design

Each subject participated in two test occasions conducted on separate days, with intervals of 4 or more weeks to ensure that their ovarian cycles were consistent. During each trial, the subjects ingested either fat cream (OFTT cream™, Jomo, Takasaki, Japan; 1 g/kg as cream, 0.35 g/kg as fat) alone (F trial) or fat cream with fructose (0.5 g/kg) (FFr trial) in a randomized crossover design. The OFTT cream was utilized as described previously [20]. Beverages were prepared at a concentration of 10% (w/v).

The subjects abstained from consuming alcohol on the day before each trial and ingested the beverage after a 12-h overnight fast. Venous blood samples were collected before ingestion (0 h) and at 0.5, 1, 2, 4, and 6 h after ingestion. During the test, subjects avoided exercise and eating but had free access to water 1 h after ingestion. Blood samples were collected with the subject in a supine position.

2.4. Biochemical Analysis

Blood and serum samples were immediately refrigerated (4°C) or frozen at −80°C until analysis. Glucose concentration was measured using the mutarotase-glucose oxidase method (Wako, Osaka, Japan). Insulin levels were determined via chemiluminescent enzyme immunoassay (Fujirebio, Tokyo). Hemoglobin A1c (HbA1c) was measured using a latex agglutination method (Fujirebio) and expressed as a National Glycohemoglobin Standardization Program (NGSP) value. Insulin resistance was evaluated using the homeostasis model assessment for insulin resistance (HOMA-IR) [21]. Total cholesterol (TC) was measured enzymatically (Sysmex, Hyogo, Japan), while HDL-C was measured using a direct method (Fujirebio). LDL-C was calculated using the Friedewald formula [22]. For TC, LDL-C, and HDL-C, only

fasting values were considered.

TG levels were measured enzymatically (Sekisui Medical, Tokyo). Remnant-like particle-TG (RP-TG) was assessed using an immunosorbent assay (Otsuka Pharmaceutical, Tokyo) [23]. Remnant lipoprotein-cholesterol (RLP-C) was measured via a homogeneous assay (MetaboRead RemL-C, Kyowa Medex, Tokyo) [24]. Apolipoproteins (Apo) A-I, A-II, B, C-II, C-III, and E were measured using the immunoturbidimetric method (Sekisui Medical). Apolipoprotein B-48 (ApoB48) was quantified using a chemiluminescent enzyme immunoassay (Fujirebio). The concentration of apolipoprotein B-100 (ApoB100) was calculated by subtracting the value of ApoB48 from the total ApoB [25]. The ApoE phenotype was determined using the isoelectric electrophoresis method (Phenotyping ApoE IEF System, Joko, Tokyo).

We also calculated nonRP-TG = TG − RP-TG (TG contained in lipoproteins other than remnants), RP-TG/TG (the ratio of remnant TG to total TG), and RP-TG/RLP-C (an index of remnant particle size) as described previously [15].

Postprandial changes in the concentrations of glucose, fructose, insulin, TG, RP-TG, RLP-C, ApoB48, ApoB100, and nonRP-TG were calculated as the difference from the baseline mean value (set to 0 at 0 h) and are represented as ΔTG, ΔRP-TG, ΔRLP-C, ΔApoB48, ΔApoB100, and ΔnonRP-TG, respectively. For TG, RP-TG, RLP-C, ApoB48, and nonRP-TG, the incremental area under the curve (ΔAUC) was also calculated, defined as the difference between the area under the curve and the area below baseline (0 h) from 0 h to 6 h [12, 26].

2.5. Statistical Analyses

The statistical analyses were conducted using SPSS ver. 28 software (IBM, Tokyo). Due to the non-normal distribution of the data, as determined by the Shapiro–Wilk test, all data were expressed as medians with the first and third quartiles (Q1, Q3). Differences in the time course compared with the fasting values in each trial were analyzed using the Friedman test, followed by the Dunnett test. Differences between trials at each time point were assessed using the Mann–Whitney U test. A significance level of $p<0.05$ was adopted for all analyses.

3. Results

3.1. Subject Characteristics

Anthropometric characteristics and fasting blood chemical data are presented in Table 1. The fat cream was tolerated well by all subjects. None of the participants met the Japanese criteria for obesity (BMI ≧ 25 kg/m^2) as defined by the Japan Society for the Study of Obesity, nor did they meet the criteria for metabolic syndrome as defined by the Japan Atherosclerosis Society. The physiques of the subjects were considered average for young Japanese women, consistent with values reported in the National Nutritional Survey [27].

Table 1. *Physical characteristics and fasting blood chemical data.*

Age (years)	21.0 (21.0, 22.0)
Height (cm)	158.3 (154.6, 160.4)
Mass (kg)	51.0 (46.0, 54.6)
BMI (kg/m^2)	20.0 (18.5, 21.5)
W/H	0.78 (0.74, 0.82)
VFA (cm^2)	25.5 (17.4, 35.5)
HOMA-IR	1.21 (0.78, 1.72)
HbA1c (%)	5.20 (5.10, 5.40)
TC (mg/dL)	169.0 (156.0, 183.0)
LDL-C (mg/dL)	93.7 (80.6, 109.8)
HDL-C (mg/dL)	64.0 (57.0, 69.0)
ApoA-I (mg/dL)	147.5 (139.0, 158.0)
ApoA-II (mg/dL)	25.3 (23.5, 27.8)
ApoC-II (mg/dL)	2.10 (1.70, 2.80)
ApoC-III (mg/dL)	6.60 (6.10, 7.70)
ApoE (mg/dL)	3.95 (3.30, 4.40)

All values are presented as median (Q1, Q3).

3.2. Fat-Ingestion Tests

Blood chemical data and calculated indices from the tests are presented in Table 2. The time courses of ΔTG (A), ΔRP-TG (B), ΔRLP-C (C), ΔApoB48 (D), ΔApoB100 (E), ΔnonRP-TG (F), RP-TG/TG (G), and RP-TG/RLP-C (H) are illustrated in Figure 1.

3.2.1. Glucose, Fructose, and Insulin

In the F trial, glucose levels did not increase and decreased at 6 h. In the FFr trial, serum glucose concentration significantly increased at 0.5 h compared to baseline. Fructose levels did not significantly change in the F trial throughout the experiment, whereas they significantly increased at 0.5, 1, and 2 h in the FFr trial. Compared to baseline, insulin levels significantly increased at 0.5, 1, and 2 h in both trials, with levels significantly higher in the FFr trial than in the F trial.

Table 2. *Blood chemical data and indices in the ingestion tests.*

	Trial	0 h	0.5 h	1 h
Glucose	F	88.9 (79.6, 96.2)	88.2 (80.8, 92.4)	88.4 (81.8, 95.8)
(mg/dL)	FFr	90.2 (81.0, 95.8)	96.6 (85.5, 101.5)*#	93.6 (84.4, 98.5)
Fructose	F	0.43 (0.18, 0.68)	0.52 (0.09, 0.74)	0.50 (0.10, 0.68)
(mg/dL)	FFr	0.45 (0.16, 0.63)	5.49 (3.87, 7.38)*#	5.84 (4.35, 7.29)*#
Insulin	F	5.1 (4.0, 7.2)	7.2 (6.0, 11.0)*	7.2 (3.8, 7.3)*
(mU/L)	FFr	5.7 (3.7, 7.6)	12.6 (9.5, 16.8)*#	11.2 (8.7, 13.7)*#
TG	F	56.5 (42.0, 74.0)		59.5 (44.0, 76.0)
(mg/dL)	FFr	52.0 (46.0, 65.0)		55.0 (46.0, 70.0)
RP-TG	F	7.9 (6.6, 10.8)		10.1 (7.3, 15.1)
(mg/dL)	FFr	8.3 (7.0, 9.9)		9.3 (7.3, 11.3)
RLP-C	F	4.50 (3.40, 58.0)		4.90 (3.50, 6.50)
(mg/dL)	FFr	4.80 (3.60, 5.70)		4.70 (3.60, 5.80)
ApoB48	F	2.00 (1.20, 2.70)		2.60 (1.90, 3.70)
(mg/L)	FFr	1.90 (1.35, 2.50)		2.05 (1.20, 2.50)#
ApoB100	F	67.8 (58.5, 80.7)		70.7 (57.3, 78.9)
(mg/dL)	FFr	69.3 (57.9, 77.8)		69.3 (58.8, 76.9)
nonRP-TG	F	48.6 (31.1, 59.6)		49.0 (33.7, 63.1)
(mg/dL)	FFr	43.5 (39.3, 56.6)		46.4 (37.5, 58.2)
RP-TG/TG	F	0.15 (0.13, 0.19)		0.18 (0.15, 0.22)
	FFr	0.15 (0.13, 0.18)		0.17 (0.14, 0.19)#
RP-TG/RLP-C	F	1.80 (1.54, 2.24)		2.27 (1.85, 2.76)
	FFr	1.75 (1.50, 2.48)		2.03 (1.63, 2.52)#

Table 2. *Continued.*

	Trial	2 h	4 h	6 h
Glucose	F	90.5 (84.0, 96.2)	86.4 (78.7, 91.4)	82.3 (75.7, 87.3)*
(mg/dL)	FFr	90.6 (82.3, 96.0)	88.4 (81.2, 94.3)	86.7 (81.9, 92.3)#
Fructose	F	0.47 (0.07, 0.81)	0.36 (0.05, 0.77)	0.40 (0.12, 0.54)
(mg/dL)	FFr	2.17 (1.55, 3.02)*#	0.63 (0.27, 0.90)#	0.47 (0.15, 0.60)
Insulin	F	5.1 (3.8, 7.3)*	3.5 (2.3, 4.6)	3.0 (2.2, 3.8)*
(mU/L)	FFr	7.4 (5.7, 11.2)*#	3.7 (2.9, 6.4)	3.1 (2.5, 4.2)*
TG	F	68.5 (53.0, 91.0)*	66.0 (51.0, 90.0)*	45.0 (36.0, 63.0)
(mg/dL)	FFr	64.0 (51.0, 83.0)	91.5 (67.0, 125.0)*#	70.5 (51.0, 88.0)*#
RP-TG	F	17.3 (12.2, 24.5)*	15.3 (10.4, 23.6)*	9.1 (7.0, 12.1)
(mg/dL)	FFr	15.0 (9.9, 21.4)*	24.0 (14.9, 34.0)*#	13.1 (10.1, 16.8)*#
RLP-C	F	5.30 (3.90, 6.90)	5.60 (4.40, 7.90)*	4.75 (3.60, 6.20)

	Trial	2 h	4 h	6 h
(mg/dL)	FFr	4.80 (3.80, 6.30)	6.85 (5.40, 8.80)* #	6.15 (4.80, 8.30)* #
ApoB48	F	3.80 (2.70, 4.70)*	3.95 (2.90, 5.20)*	2.95 (2.20, 3.60)*
(mg/L)	FFr	3.10 (2.40, 4.70))*	4.90 (4.10, 6.00)* #	3.45 (2.80, 4.70)*
ApoB100	F	67.9 (56.6, 78.6)	69.8 (57.6, 81.6)	71.1 (58.8, 82.8)
(mg/dL)	FFr	68.4 (57.8, 76.7)	69.5 (58.6, 78.3)	71.1 (60.7, 79.5)
nonRP-TG	F	52.2 (39.5, 67.2)	51.3 (36.9, 67.2)	36.2 (28.3, 52.9)
(mg/dL)	FFr	49.7 (38.6, 61.6)	67.0 (51.3, 84.0)* #	57.8 (40.6, 72.1)* #
RP-TG/TG	F	0.26 (0.22. 0.29)*	0.24 (0.20, 0.28)*	0.21 (0.18, 0.24)*
	FFr	0.24 (0.19, 0.29)*	0.26 (0.22, 0.32)* #	0.19 (0.16, 0.22)*
RP-TG/RLP-C	F	3.25 (2.40, 4.45)*	2.79 (2.00, 3.50)*	1.92 (1.64, 2.29)
	FFr	3.24 (2.23, 3.79)*	3.34 (2.64, 4.60)* #	2.05 (1.57, 2.81)

All values are presented as median (Q1, Q3). *p<0.05 compared to the fasting values. # p<0.05 compared between the trials.

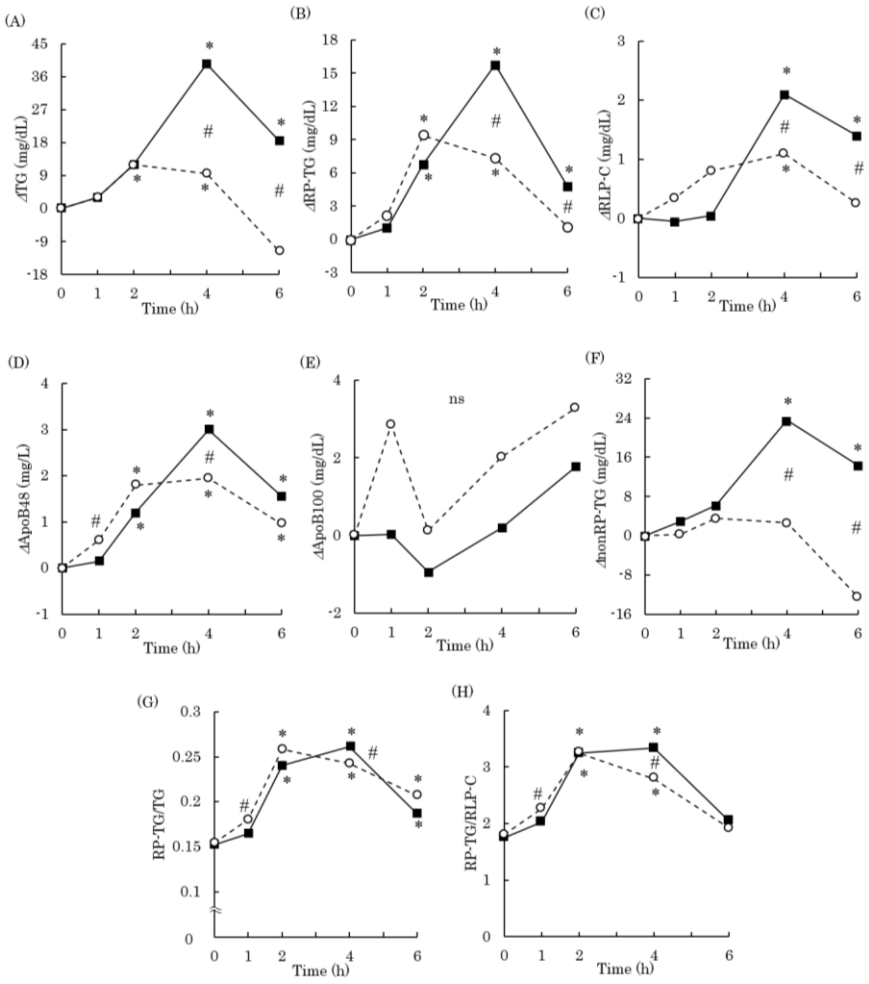

Figure 1. *Time courses for ΔTG (A), ΔRP-TG (B), ΔRLP-C (C), ΔApoB48 (D), ΔApoB100 (E), ΔnonRP-TG (F), RP-TG/TG (G) and RP-TG/RLP-C (H).*

○ F trial, ■ FFr trial. Values are shown as the median. *p<0.05 compared to the fasting values. #p<0.05 compared between the trials. ns: not significant.

Table 3. ⊿AUC for TG, RP-TG, RLP-C, ApoB48, and nonRP-TG.

Trial	TG (mg·h/dL)	RP-TG (mg·h/dL)	RLP-C (mg·h/dL)	ApoB48 (mg·h/L)	nonRP-TG (mg·h/dL)
F	37.3 (17.0, 70.5)	31.2 (21.0, 50.3)	3.5 (1.7, 5.8)	8.2 (5.1, 11.2)	13.2 (12.5, 28.6)
FFr	83.8 (62.0, 155.0)#	46.2 (26.8, 78.4)#	6.3 (4.2, 11.4)#	9.9 (7.0, 12.8)	46.1 (28.8, 76.9)#

All values are presented as median (Q1, Q3). # $p<0.05$ compared between the trials.

3.2.2. TG, RP-TG, and RLP-C

In the F trial, the serum concentration of TG significantly increased at 2 and 4 h, returning to baseline at 6 h. In the FFr trial, TG levels significantly increased at 4 h and did not return to baseline even at 6 h. The TG levels at 4 and 6 h were significantly higher in the FFr trial compared to the F trial. The concentration of RP-TG significantly increased at 2 and 4 h, returning to baseline at 6 h in the F trial. In the FFr trial, RP-TG significantly increased at 2 and 4 h but did not return to baseline at 6 h. The RP-TG concentration at 4 and 6 h was significantly higher in the FFr trial than in the F trial. The concentration of RLP-C significantly increased at 4 h and returned to baseline at 6 h in the F trial. In the FFr trial, RLP-C significantly increased at 4 h but did not return to baseline at 6 h. The RLP-C concentration at 4 and 6 h was significantly higher in the FFr trial than in the F trial.

3.2.3. ApoB48 and ApoB100

The serum concentration of ApoB48 significantly increased at 2 and 4 h and did not return to baseline even at 6 h in both trials. The ApoB48 level in the FFr trial was significantly lower at 1 h and significantly higher at 4 h compared to the F trial. The serum concentration of ApoB100 did not significantly change in either trial during the experiment and was not different between the two trials.

3.2.4. nonRP-TG, RP-TG/TG, and RP-TG/RLP-C

In the F trial, the concentration of nonRP-TG did not change significantly during the experiment. However, in the FFr trial, it significantly increased at 4 h and did not return to baseline even at 6 h. The nonRP-TG level at 4 and 6 h was significantly higher in the FFr trial than in the F trial. The RP-TG/TG ratio significantly increased at 2, 4, and 6 h in both trials. The RP-TG/TG ratio in the FFr trial was significantly lower at 1 h and significantly higher at 4 h than in the F trial. The RP-TG/RLP-C ratio significantly increased at 2 and 4 h in both trials. The RP-TG/RLP-C ratio in the FFr trial was significantly lower at 1 h and significantly higher at 4 h than in the F trial.

3.2.5. ⊿AUC for TG, RP-TG, RLP-C, ApoB48, and nonRP-TG

⊿AUC for TG, RP-TG, RLP-C, ApoB48, and nonRP-TG are presented in Table 3. The ⊿AUC values for TG, RP-TG, RLP-C, and nonRP-TG in the FFr trial were significantly larger than those in the F trial. In contrast, the ⊿AUC for ApoB48 did not differ between the trials.

4. Discussion

The major findings of this study indicate that the ingestion of fat cream (F trial) did not stimulate endogenous (hepatic) TG secretion. However, the coingestion of fructose with fat cream (FFr trial) markedly stimulated endogenous TG secretion and exacerbated postprandial lipidemia. The RP-TG/TG ratio at 4 h was higher in the FFr trial compared to the F trial, suggesting an increased remnant ratio. Similarly, the RP-TG/RLP-C ratio at 4 h was also higher in the FFr trial, indicating larger remnant particle size. These results suggest that after ingesting fructose with fat, the remnant particles were larger, and the ratio of remnant TG to total TG in the late postprandial phase was higher compared to the ingestion of fat alone.

Plasma levels of TRL are influenced by the rate of production, the efficiency of TG content removal via lipolysis, and the hepatic clearance of remnant particles. However, with moderately elevated plasma TG, overproduction is the primary contributor [28]. The increase in hepatic endogenous TRL secretion may also delay the metabolism of exogenous intestinal TRL by lipoprotein lipase [29].

The hepatic metabolism of fructose is independent of energy status and results in unregulated uptake by the liver, leading to increased lipogenesis [30]. A single bolus of fructose rapidly increases hepatic de novo lipogenesis [31] and stimulates VLDL-TG production and secretion [32]. Fructose also inhibits hepatic lipid oxidation, favoring fatty acid reesterification [33].

ApoB is the major structural protein in TRL, existing as either ApoB100, produced in the liver, or a truncated form, ApoB48, made in the intestine [28]. During lipolysis, ApoB remains associated with the TRL on which it was secreted. Since there is one ApoB molecule per particle, ApoB concentration serves as a measure of particle number. The intestine produces large chylomicrons containing ApoB48, which are then metabolized into remnant particles. However, the intestine secretes VLDL-sized particles containing ApoB48 [34]. Thus, rather than size or density, the isoforms

of ApoB, namely ApoB48 and ApoB100, accurately categorize the TRL class. In this study, we define CM as the intestinally secreted TRL and VLDL as the hepatically secreted TRL.

In the present study, the coingestion of fructose with fat cream exacerbated postprandial lipidemia primarily by stimulating the secretion of endogenous non-remnant TG, i.e., VLDL-TG secretion. However, the ingestion of fat cream alone did not stimulate the secretion of VLDL-TG at all. Accordingly, the postprandial rise in TG following the ingestion of fat alone is suggested to be mostly due to exogenous TRL-TG. We previously reported that the coingestion of glucose with fat cream did not increase endogenous TRL-TG [12]. Additionally, the coingestion of fructose with fat cream slightly but significantly delayed the metabolism of both exogenous and endogenous remnants. We also demonstrated that this combination caused a delay in the clearance of ApoB48-containing TRL from the circulation [26]. In the present study, the ApoB48 concentration in the FFr trial was significantly higher at 4 h compared to the F trial, suggesting that more exogenous lipoprotein particles were circulating in the late postprandial phase. The simultaneous ingestion of fructose and fat cream may have also delayed the absorption of fat in the intestine and/or the secretion of ApoB48-containing TRL particles from the intestine.

This study has strengths and limitations. First, the number of subjects enrolled was larger than in our previous studies [12, 26]. We also used various indices such as nonRP-TG, RP-TG/TG, and RP-TG/RLP-C in addition to TG, RP-TG, RLP-C, ApoB100, and ApoB48. However, further investigations are needed to clarify whether these measurements and indices are suitable for estimating postprandial lipid and lipoprotein metabolism. Finally, since only Japanese young women were examined, the results may not be applicable to other populations.

5. Conclusion

Although the ingestion of fat cream alone did not stimulate the secretion of hepatic endogenous TG, coingestion of fructose with fat cream exacerbated postprandial lipidemia not only by delaying the metabolism of both exogenous and endogenous TRL but also by stimulating endogenous TG secretion.

Abbreviations

BMI	Body Mass Index
CAD	Coronary Artery Disease
CM	Chylomicron
HbA1c	Hemoglobin A1c
HDL-C	High-density Lipoprotein-cholesterol
HOMA-IR	Homeostasis Model Assessment for Insulin Resistance
LDL	Low-density Lipoprotein
NGSP	National Glycohemoglobin Standardization Program
RLP-C	Remnant Lipoprotein-cholesterol
RP-TG	Remnant-like particle-triglyceride
TC	Total Cholesterol
TG	Triglyceride
TRL	Triglyceride-rich Lipoprotein
VFA	Visceral Fat Area
VLDL	Very Low-density Lipoprotein
W/H	Waist-to-Hip
ΔAUC	Incremental Area Under the Curve

Acknowledgments

The authors are grateful to Hiromi Ishii, Chiharu Iijima, Kaori Kuzawa for their technical assistance.

Author Contributions

Erika Mizutani-Watanabe: Conceptualization, Data curation, Formal Analysis, Investigation, Visualization, Writing - original draft

Michitaka Naito: Data curation, Funding acquisition, Investigation, Project administration, Supervision

Funding

This work was supported by Grants-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology, Japan (21300259, 24500874, and 15K00855).

Data Availability Statement

The data supporting the outcome of this research work has been reported in this manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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