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Predictability of Postprandial Lipidemia by Fasting Values in Young Women

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Abstract: For postprandial hyperlipidemia, screening large numbers of subjects by fat-ingestion tests is inconvenient and unrealistic. In the present study, we performed a fat-ingestion test and searched for fasting items to predict postprandial lipidemia. Methods: Healthy young Japanese women (n=54, age 21.1 ± 1.0 y) with apolipoprotein E phenotype 3/3 were enrolled. They ingested fat cream (OFTT cream™, Jomo, Japan; 1 g/kg as cream, 0.35 g/kg as fat). Venous blood samples were taken before (0 h) and at 0.5, 1, 2, 4, and 6 h after ingestion. Results: In multiple regression analyses, the area under the curve (AUC) and the peak of serum triglyceride (TG) were predictable by fasting (f)-TG itself, the AUC of remnant-like particle-TG (RP-TG) was predictable by f-TG and f-RP-TG, the AUC and the peak of remnant lipoprotein-cholesterol (RLP-C) were predictable by f-RLP-C itself, and those of apolipoprotein B-48 (ApoB48) were predictable by f-ApoB48 itself. The AUC and the peak of TG–RP-TG, an index of non-remnant TG, were predictable by f-TG. Conclusion: Postprandial lipidemia may be predictable by the measurement of the set of fasting serum TG, RLP-C, and ApoB48. For daily medical practice, without performing a fat-ingestion test, the set may provide a useful device for predicting postprandial lipidemia.

Keywords: Predictability, Fat-Ingestion Test, Postprandial Lipidemia, Triglyceride, Remnant Lipoprotein-Cholesterol, Apolipoprotein B-48, Women

1. Introduction

The clinical significance of postprandial or non-fasting plasma glucose levels has already been established, and the oral glucose tolerance test (OGTT) is widely used. For the significance of postprandial lipidemia, Zilversmit first proposed that atherosclerosis is a postprandial phenomenon [1]. We recently showed that even in young healthy women, the ingestion of a moderate amount of fat (0.35 g/kg) caused oxidative stress by increasing superoxide radicals and hypochlorite ions and that the degree of the oxidative stress was positively correlated with the postprandial rise of TG [2]. However, different from the OGTT, the fat-ingestion test has not been put to practical use. One reason is that it takes 6 h or more for the fat-ingestion test [3, 4], compared to 2 or 3 h for the OGTT. Accordingly, screening large numbers of subjects by fat-ingestion tests is inconvenient and unrealistic. The definition or the standard method for predicting postprandial hyperlipidemia also has never been established. Therefore, developing easy and inexpensive fasting markers for predicting postprandial lipidemia is practical.

In this study, we aimed to investigate the possibility of using the fasting values to predict postprandial lipidemia and searched for the fasting items useful for predicting postprandial lipidemia. We enrolled young women because the highest consumers of high-fat fast food are adolescents and young adults [5].

2. Methods

2.1. Subjects

Healthy young Japanese women (n=54, age 21.1 ± 1.0 y) with a normal ovarian cycle and apolipoprotein E phenotype 3/3 were enrolled. They were non-smokers, were not suffering from any apparent acute or chronic illness and were not taking any medication or dietary supplements. These subjects

analyzed in the present study are the same as those reported previously [4]. This study was approved by the Institutional Review Board of Sugiyama Jogakuen University School of Life Studies (Nos. 2013-3, 2014-2, 2014-22, and 2014-23). The subjects provided written informed consent. The 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 according to a standard method. The waist circumference was assessed as the abdominal girth at the level of the umbilicus, and the hip circumference was measured at the level of the greater trochanters. The body mass and composition, including the %body fat (%BF) and visceral fat area (VFA), were analyzed using an 8-polar bioelectrical impedance method (InBody720, BioSpace, Tokyo, Japan). The body mass index (BMI) and the waist-to-hip (W/H) ratio were calculated.

2.3. Experimental Design

The subjects ingested fat cream (OFTT cream™, Jomo, Takasaki, Japan; 1 g/kg body mass as cream, 0.35 g/kg as fat). The OFTT cream was used as described [6]. The fat beverage was prepared by mixing 1 g/kg of OFTT cream with the same final amount of distilled water. The subjects abstained from consuming alcohol on the day before the trial and ingested the beverage after a 12 h overnight fast. Venous blood samples were taken before ingestion (0 h) and at 0.5, 1, 2, 4, and 6 h after beverage ingestion. During the test, the subjects avoided exercise and eating but had free access to water 1 h after ingestion. Blood samples were taken with the subject in a supine position.

2.4. Biochemical Analysis

Blood and serum samples were immediately refrigerated (4°C) or frozen (−80°C) until analysis. The glucose concentration was measured using a mutarotase–glucose oxidase method (Wako, Osaka, Japan). Insulin was measured via chemiluminescent enzyme immunoassay (Fujirebio, Tokyo). Hemoglobin A1c (HbA1c) was measured using a latex agglutination method (Fujirebio) and was expressed as a National Glycohemoglobin Standardization Program (NGSP) value. Insulin resistance was evaluated using the homeostasis model assessment for insulin resistance (HOMA-IR) [7]. Total cholesterol (TC) was measured enzymatically (Sysmex, Hyogo, Japan). HDL-C was measured using a direct method (Fujirebio). LDL-C was calculated using the Friedewald formula [8]. The non-HDL-C level was calculated by subtracting the HDL-C value from the TC value. For TC, LDL-C, HDL-C, and non-HDL-C, only fasting values were measured.

The level of TG was measured enzymatically (Sekisui Medical, Tokyo). Remnant-like particle-TG (RP-TG) was measured by an immunosorbent assay (Otsuka Pharmaceutical, Tokyo) [9]. Remnant lipoprotein-cholesterol (RLP-C) was measured via homogeneous assay (MetaboRead RemL-C, Kyowa Medex, Tokyo) [10]. 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 measured via chemiluminescent enzyme immunoassay (Fujirebio). The concentration of apolipoprotein B-100 (ApoB100) was calculated by subtracting the value of ApoB48 from the value of ApoB [11]. The ApoE phenotype was measured using the isometric electrophoresis method (Phenotyping ApoE IEF System, Joko, Tokyo). We also calculated TG−RP-TG (an index of non-remnant TG level), RP-TG/TG (an index of the ratio of remnant TG to total TG), and RP-TG/RLP-C (an index of remnant particle size) as described previously [4].

2.5. Statistical Analyses

Statistical analyses were performed using SPSS ver. 25 software (IBM, Tokyo). The normal distribution of data was verified using the Shapiro–Wilk test for skewness and kurtosis of distribution. Non-normally distributed data showed a normal distribution when logarithmically transformed, and they were analyzed using parametric statistics. The data were presented as the mean ± SEM. The difference in the time course as compared with the fasting value was analyzed by performing a repeated-measures analysis of variance (ANOVA), followed by the Dunnett test. For all data, $p<0.05$ was considered significant.

For multiple regression analyses, the stepwise method was employed. The 95% confidence interval (95%CI) was presented. For the prediction of dependent variables, the standard partial regression coefficient (β) was calculated and, when significant ($p<0.05$), the prediction accuracy was estimated by adjusted R^2, and $R^2 > 0.5$ was considered to be 'highly predictable'. Furthermore, the multicollinearity between independent variables was denied when the variance inflation factor (VIF) was <10. If the VIF was ≥10, the data were not adopted.

3. Results

3.1. Subject Characteristics

Anthropometric characteristics and fasting blood chemical data are shown in Table 1. The fat cream was tolerated well by all subjects. None of the subjects met the Japanese criteria for obesity (BMI ≥25 kg/m^2), according to the definition by the Japan Society for the Study of Obesity, or metabolic syndrome, as defined by the Japan Atherosclerosis Society. The physiques of the subjects were considered average for young Japanese women, similar to the values reported in the National Nutritional Survey [12].

3.2. Fat-Ingestion Test

Blood chemical data and calculated indices in the fat-ingestion test are presented in Table 2. Correlation analyses between physical characteristics and fasting blood chemical values and the AUC, ΔAUC, or the peaks of

glucose and insulin (Table 3), TG, RP-TG, and RLP-C (Table 4), ApoB48 and ApoB100 (Table 5), and TG−RP-TG, RP-TG/TG, and RP-TG/RLP-C (Table 6) are shown.

Table 1. *Physical characteristics and fasting blood chemical data of the subjects.*

Age (years)	21.1	±	0.1
Height (cm)	158.3	±	0.6
Mass (kg)	50.6	±	0.8
BMI (kg/m^2)	20.2	±	0.3
%BF	25.4	±	0.6
W/H	0.78	±	0.01
VFA (cm^2)	26.1	±	1.9
HOMA-IR	1.3	±	0.1
HbA1c	5.23	±	0.04
TC (mg/dL)	174.2	±	3.2
LDL-C (mg/dL)	97.6	±	3.0
HDL-C (mg/dL)	65.0	±	1.4
non-HDL-C (mg/dL)	109.2	±	3.2
ApoAI (mg/dL)	151.2	±	2.6
ApoAII (mg/dL)	25.9	±	0.5
ApoCII (mg/dL)	2.3	±	0.1
ApoCIII (mg/dL)	7.0	±	0.2
ApoE (mg/dL)	4.0	±	0.1

Values are the mean ± SEM

3.2.1. BMI, %BF, W/H, and VFA

BMI and %BF were positively correlated with the *Δ*AUC of TG, RLP-C, ApoB48 (%BF only), and TG–RP-TG. The VFA was positively correlated with the AUC and the peak of TG, *Δ*AUC of ApoB48, and the AUC, *Δ*AUC and the peak of TG–RP-TG. VFA was also positively correlated with the peak of insulin. None of the items was significantly correlated with the W/H ratio (data not shown).

3.2.2. f-Glucose, f-Insulin, HOMA-IR, and HbA1c

f-Glucose was positively correlated with the AUC and the peak of glucose and negatively correlated with the *Δ*AUC of glucose. f-Insulin was positively correlated with the AUC and the peak of insulin and negatively correlated with the *Δ*AUC of insulin. f-Insulin was also positively correlated with the AUC of RP-TG and negatively with the *Δ*AUC of ApoB100. HOMA-IR was positively correlated with the AUC and the peaks of glucose and insulin and negatively correlated with the *Δ*AUC of insulin and ApoB100. HOMA-IR was also positively correlated with the AUC of RP-TG. None of the items were significantly correlated with HbA1c (data not shown).

3.2.3. TC, LDL-C, HDL-C, and Non-HDL-C

TC, LDL-C, and non-HDL-C were positively correlated with the AUC and the peaks of RLP-C and ApoB100 and negatively correlated with those of RP-TG/RLP-C. HDL-C was negatively correlated with the AUC and the peaks of TG and RLP-C, the AUC, *Δ*AUC, and the peak of RP-TG, and the AUC and the peak of TG–RP-TG.

3.2.4. f-TG, f-RP-TG, f-RLP-C, f-ApoB48, and f-ApoB100

f-TG, f-RP-TG, f-RLP-C, and f-ApoB48 were all positively correlated with the AUC and the peaks of TG, RP-TG, RLP-C, ApoB48, and TG–RP-TG. f-TG and f-RP-TG were positively correlated with the AUC and the peak of insulin. f-TG was positively correlated with the *Δ*AUC of RP-TG and the AUC and the peak of ApoB100. f-RP-TG was negatively correlated with the *Δ*AUC of ApoB100. f-RLP-C was positively correlated with the AUC and the peak of ApoB100 and negatively correlated with the AUC and the peak of RP-TG/RLP-C. f-ApoB100 was positively correlated with the AUC and peaks of RLP-C and ApoB100 and negatively correlated with those of RP-TG/RLP-C.

3.2.5. f-TG–RP-TG, f-RP-TG/TG, and f-RP-TG/RLP-C

f-TG–RP-TG was positively correlated with the AUC and peaks of insulin, TG, RP-TG, RLP-C, ApoB48, ApoB100, and TG–RP-TG. f-TG–RP-TG was also positively correlated with the *Δ*AUC of RP-TG and negatively with the AUC of RP-TG/TG. f-RP-TG/TG was negatively correlated with the AUC and peaks of TG and TG–RP-TG and with the *Δ*AUC of RP-TG, RP-TG/TG, and RP-TG/RLP-C. f-RP-TG/RLP-C was positively correlated with the peak of insulin and the AUC and the peak of RP-TG/RLP-C and negatively correlated with those of RLP-C and ApoB100.

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

	0 h			0.5 h			1 h			2 h			4 h			6 h		
Glucose (mg/dL)	88.2	±	1.8	87.1	±	1.5	88.1	±	1.7	89.4	±	1.6	85.5	±	1.4*	81.8	±	1.4*
Insulin (mU/L)	5.93	±	0.41	8.53	±	0.52*	7.35	±	0.39*	5.60	±	0.33	3.71	±	0.24*	3.15	±	0.20*
TG (mg/dL)	57.8	±	3.0				61.3	±	3.1	73.9	±	3.8*	71.3	±	4.0*	51.3	±	3.1*
RP-TG (mg/dL)	8.9	±	0.5				11.9	±	0.8*	19.3	±	1.3*	18.1	±	1.4*	10.2	±	0.6
RLP-C (mg/dL)	4.90	±	0.26				5.02	±	0.26	5.60	±	0.29*	6.22	±	0.34*	5.19	±	0.28
ApoB48 (mg/L)	2.24	±	0.18				2.98	±	0.19*	4.11	±	0.24*	4.26	±	0.27*	3.31	±	0.25*
ApoB100 (mg/dL)	69.4	±	1.9				68.7	±	1.9	68.5	±	1.9*	69.4	±	1.9	70.9	±	1.9*
TG−RP-TG	48.9	±	2.5				49.4	±	2.6	54.6	±	2.8	53.2	±	3.2	41.1	±	2.6
RP-TG/TG	0.16	±	0.01				0.20	±	0.01	0.26	±	0.01*	0.26	±	0.02*	0.21	±	0.01*
RP-TG/RLP-C	1.95	±	0.11				2.51	±	0.17	3.62	±	0.22*	2.98	±	0.19*	2.03	±	0.08

Values are the mean ± SEM. *p<0.05 vs. the fasting value.

Table 3. *Correlation analysis between physical characteristics and fasting values and the AUC, ΔAUC, or the peaks of glucose and insulin.*

	Glucose			Insulin		
	AUC	**ΔAUC**	**Peak**	**AUC**	**ΔAUC**	**Peak**
BMI	0.199	−0.010	0.172	0.251	0.071	0.140
%BF	−0.047	0.004	−0.074	0.187	0.102	0.231
VFA	0.012	−0.058	0.009	0.200	0.062	0.277*
f-Glucose	0.893**	−0.602**	0.862**	0.082	−0.170	−0.020
f-Insulin	0.182	−0.010	0.176	0.834**	−0.790**	0.695**
HOMA-IR	0.382**	−0.189	0.367**	0.801**	−0.764**	0.647**
TC	−0.159	0.056	−0.176	−0.057	0.092	0.098
LDL-C	−0.155	0.060	−0.185	−0.075	0.131	−0.004
HDL-C	−0.070	−0.047	−0.014	−0.128	−0.064	0.077
non-HDL-C	−0.131	0.078	−0.172	−0.002	0.120	0.065
f-TG	0.101	0.103	0.039	0.372**	−0.035	0.369**
f-RP-TG	−0.081	0.090	−0.154	0.437**	−0.191	0.536**
f-RLP-C	−0.131	0.234	−0.182	0.191	−0.070	0.258
f-ApoB48	0.042	0.053	−0.034	0.190	−0.029	0.224
f-ApoB100	−0.120	0.020	−0.149	0.030	0.111	0.060
f-TG−RP-TG	0.128	0.100	0.071	0.340*	−0.005	0.318*
f-RP-TG/TG	−0.233	0.007	−0.257	0.028	−0.164	0.153
f-RP-TG/RLP-C	0.147	−0.110	0.106	0.224	−0.083	0.314*

*p<0.05, **p<0.01

Table 4. *Correlation analysis between physical characteristics and fasting values and the AUC, ΔAUC, or the peaks of TG, RP-TG, and RLP-C.*

	TG			RP-TG			RLP-C		
	AUC	**ΔAUC**	**Peak**	**AUC**	**ΔAUC**	**Peak**	**AUC**	**ΔAUC**	**Peak**
BMI	−0.050	0.283*	−0.055	−0.063	−0.050	−0.138	−0.070	0.350**	−0.039
%BF	0.158	0.290*	0.161	0.189	0.081	0.096	0.189	0.331*	0.204
VFA	0.283*	0.259	0.296*	0.246	0.089	0.147	0.237	0.229	0.245
f-Glucose	0.087	0.149	0.069	−0.011	0.068	−0.063	−0.164	0.084	−0.144
f-Insulin	0.257	0.065	0.228	0.326*	0.130	0.220	0.178	0.079	0.182
HOMA-IR	0.262	0.098	0.231	0.291*	0.130	0.177	0.114	0.093	0.123
TC	−0.018	−0.160	−0.014	−0.013	−0.067	−0.009	0.363**	−0.015	0.345*
LDL-C	0.038	−0.104	0.023	0.021	0.005	0.022	0.393**	0.051	0.363**
HDL-C	−0.532**	−0.185	−0.474**	−0.382**	−0.331*	−0.312*	−0.363**	−0.209	−0.331*
non-HDL-C	0.212	−0.082	0.191	0.153	0.075	0.126	0.524**	0.075	0.492**
f-TG	0.928**	0.100	0.894**	0.699**	0.372**	0.552**	0.766**	0.137	0.752**
f-RP-TG	0.674**	0.079	0.659**	0.668**	0.116	0.463**	0.604**	0.120	0.597**
f-RLP-C	0.700**	0.002	0.690**	0.528**	0.229	0.435**	0.935**	0.062	0.897**
f-ApoB48	0.567**	0.107	0.560**	0.470**	0.237	0.341*	0.533**	0.074	0.514**
f-ApoB100	0.244	−0.095	0.217	0.161	0.078	0.124	0.507**	0.053	0.473**
f-TG−RP-TG	0.920**	0.098	0.885**	0.665**	0.396**	0.537**	0.751**	0.133	0.738**
f-RP-TG/TG	−0.347*	−0.064	−0.324*	−0.093	−0.373**	−0.149	−0.200	−0.079	−0.204
f-RP-TG/RLP-C	0.038	0.090	0.036	0.178	−0.052	0.079	−0.346*	0.084	−0.300*

*p<0.05, **p<0.01

3.2.6. Multiple Regression Analyses

Then we performed multiple regression analyses of the AUC, ΔAUC, and peaks of TG, RP-TG, RLP-C, ApoB48, TG–RP-TG, RP-TG/TG, and RP-TG/RLP-C as dependent variables and f-TG, non-HDL-C, f-RP-TG, f-RLP-C, and f-ApoB48 as independent variables. Only highly predictable results (R^2 >0.5) are shown in Table 7.

The AUC and the peak of TG were predictable (R^2 >0.5) by f-TG only. The AUC of RP-TG was predictable by f-TG and f-RP-TG. The AUC and the peak of RLP-C were predictable by f-RLP-C only. The AUC and the peak of ApoB48 were predictable by f-ApoB48 only. The AUC and the peak of TG–RP-TG were predictable by f-TG only. RP-TG/TG and RP-TG/RLP-C were not predictable by any of the fasting values (data not shown).

Table 5. *Correlation analysis between physical characteristics and fasting values and the AUC, ΔAUC, or the peaks of ApoB48 and ApoB100.*

	ApoB48			ApoB100		
	AUC	**ΔAUC**	**Peak**	**AUC**	**ΔAUC**	**Peak**
BMI	0.051	0.230	0.030	0.095	−0.173	0.098
%BF	0.098	0.326*	0.083	0.191	−0.206	0.203
VFA	0.145	0.277*	0.130	0.237	−0.147	0.257
f-Glucose	−0.033	−0.082	−0.061	−0.114	−0.058	−0.100
f-Insulin	0.132	0.027	0.132	−0.082	−0.285*	−0.072

	ApoB48			ApoB100		
	AUC	⊿AUC	Peak	AUC	⊿AUC	Peak
HOMA-IR	0.119	0.017	0.115	−0.097	−0.289*	−0.085
TC	0.115	0.120	0.168	0.846**	−0.071	0.840**
LDL-C	0.064	0.089	0.099	0.946**	−0.057	0.941**
HDL-C	−0.113	0.045	−0.057	−0.263	0.054	−0.263
non-HDL-C	0.165	0.102	0.194	0.968**	−0.095	0.963**
f-TG	0.545**	0.089	0.525**	0.289*	−0.210	0.290*
f-RP-TG	0.478**	0.105	0.492**	0.137	−0.320*	0.139
f-RLP-C	0.492**	0.046	0.511**	0.529**	−0.072	0.520**
f-ApoB48	0.865**	0.039	0.849**	0.132	−0.183	0.137
f-ApoB100	0.156	0.050	0.181	0.992**	−0.112	0.990**
f-TG−RP-TG	0.526**	0.082	0.502**	0.300*	−0.178	0.301*
f-RP-TG/TG	−0.063	0.056	−0.035	−0.180	−0.115	−0.185
f-RP-TG/RLP-C	0.033	0.132	0.026	−0.423**	−0.258	−0.421**

*p<0.05, **p<0.01

Table 6. *Correlation analysis between physical characteristics and fasting values and the AUC, ΔAUC, or the peaks of TG−RP-TG, RP-TG/TG, and RP-TG/RLP-C.*

	TG−RP-TG			RP-TG/TG			RP-TG/RLP-C		
	AUC	⊿AUC	Peak	AUC	⊿AUC	Peak	AUC	⊿AUC	Peak
BMI	−0.043	0.386**	−0.089	−0.111	−0.202	−0.180	−0.045	−0.159	−0.153
%BF	0.137	0.316*	0.119	−0.022	−0.100	−0.112	−0.062	−0.083	−0.131
VFA	0.274*	0.273*	0.275*	−0.117	−0.141	−0.174	−0.038	−0.078	−0.126
f-Glucose	0.110	0.146	0.044	−0.218	−0.065	−0.223	0.191	0.065	0.052
f-Insulin	0.218	0.003	0.208	0.162	0.072	0.067	0.200	0.045	0.082
HOMA-IR	0.235	0.044	0.204	0.076	0.033	−0.018	0.239	0.047	0.088
TC	−0.018	−0.161	0.004	0.016	−0.010	0.010	−0.438**	−0.199	−0.360**
LDL-C	0.041	−0.134	0.047	0.012	0.047	0.026	−0.449**	−0.141	−0.364**
HDL-C	−0.539**	−0.032	−0.500**	0.124	−0.137	0.051	−0.021	−0.150	−0.003
non-HDL-C	0.215	−0.149	0.220	−0.038	0.049	−0.013	−0.433**	−0.137	−0.363**
f-TG	0.931**	−0.100	0.925**	−0.263	0.023	−0.198	0.003	−0.001	−0.059
f-RP-TG	0.629**	0.029	0.635**	0.036	−0.216	−0.088	0.157	−0.258	0.037
f-RLP-C	0.702**	−0.137	0.732**	−0.147	0.002	−0.090	−0.423**	−0.094	−0.356**
f-ApoB48	0.556**	−0.010	0.559**	−0.093	−0.008	−0.130	−0.019	0.023	−0.080
f-ApoB100	0.251	−0.168	0.247	−0.084	0.045	−0.057	−0.418**	−0.117	−0.362**
f-TG−RP-TG	0.932**	−0.118	0.925**	−0.302*	0.064	−0.206	−0.024	0.044	−0.072
f-RP-TG/TG	−0.399**	0.147	−0.387**	0.386**	−0.400**	0.141	0.143	−0.389**	0.099
f-RP-TG/RLP-C	−0.006	0.145	−0.033	0.170	−0.190	0.000	0.757**	−0.180	0.548**

*p<0.05, **p<0.01

Table 7. *Multiple regression analyses of the AUC, ΔAUC, or the peaks of TG, RP-TG, RLP-C, and ApoB48 as dependent variables.*

Dependent variable		Independent variable	β	95%CI			VIF	Adjusted R^2
TG	AUC	f-TG	0.928	5.553	-	6.952	1.000	0.858
	Peak	f-TG	0.894	1.061	-	1.404	1.000	0.796
RP-TG	AUC	f-TG	0.453	0.289	-	1.187	2.102	0.526
		f-RP-TG	0.340	0.669	-	6.355		
RLP-C	AUC	f-RLP-C	0.935	5.497	-	6.790	1.000	0.872
	Peak	f-RLP-C	0.897	1.025	-	1.350	1.000	0.801
ApoB48	AUC	f-ApoB48	0.865	5.147	-	7.127	1.000	0.744
	Peak	f-ApoB48	0.849	1.145	-	1.624	1.000	0.716
TG−RP-TG	AUC	f-TG	0.931	4.555	-	5.671	1.000	0.864
	Peak	f-TG	0.925	0.846	-	1.063	1.000	0.854

4. Discussion

Although the subjects in the present study were all lean and non-obese, BMI and %BF indices of obesity or adiposity were positively correlated with the ⊿AUC of TG, RLP-C, TG–RP-TG, and ApoB48 (%BF only). Meanwhile, VFA, an index of visceral adiposity, was correlated with the AUC, ⊿AUC, and peak of TG–RP-TG. Abdominal obesity has been reported to be associated with postprandial hyperlipidemia [13, 14] and men (mainly middle-aged) with the "hypertriglyceridemic waist" phenotype (waist ≥90 cm and TG >2.0 mmol/L) had exaggerated postprandial lipidemic responses as compared with those without it [15]. The present results suggest that, even within a normal range, adiposity may be a factor in increased postprandial lipidemia and that VFA, also in the normal range, may be related to postprandial non-remnant TG metabolism in young women.

f-Glucose was positively correlated with the AUC and the peak of glucose and negatively with the *Δ*AUC, suggesting that the higher the fasting value, the higher the total AUC and the peak were; conversely, the additional increase after fat ingestion was lower. f-Insulin also showed similar tendencies. The AUC and the peak of insulin were also correlated with f-TG, f-RP-TG, and f-TG–RP-TG, suggesting that fasting remnant and non-remnant TG levels are associated with insulin metabolism. HOMA-IR was positively correlated with the AUC and peak of both glucose and insulin, but HbA1c had no correlation with any of the items. This suggests that HOMA-IR may be a more sensitive marker of glucose metabolism or insulin resistance than HbA1c.

f-TG, f-RP-TG, f-RLP-C, and f-ApoB48 were all related to postprandial TG, RP-TG, RLP-C, ApoB48, and TG–RP-TG, suggesting that these items are involved in the overall postprandial lipid and lipoprotein metabolism and work in close cooperation with each other. f-TG, non-HDL-C, and f-RLP-C were also positively correlated with the AUC and peak of ApoB100. ApoB100 particles are present, in large part, in LDL particles, so the fluctuation in the fat-ingestion test is small; however, f-ApoB100 was positively correlated with the AUC and the peak of RLP-C, which is the index of the mainly endogenous apoB100-containing remnant particle number. HDL-C was negatively correlated with these items, particularly the AUC and peak of TG–RP-TG, namely non-remnant TG, suggesting that the HDL-C level can be an index of non-remnant, but not remnant, TG levels.

In multiple regression analyses, postprandial TG (AUC and peak) was predictable by f-TG itself. Similarly, RP-TG (AUC) was predictable by f-TG and f-RP-TG, and RLP-C (AUC and peak) was predictable by f-RLP-C itself. ApoB48 (AUC and peak) was predictable by f-ApoB48 itself. TG–RP-TG (AUC and peak) was predictable by f-TG. However, RP-TG/TG and RP-TG/RLP-C were not predictable by f-TG, indicating that f-TG can be a marker of postprandial non-remnant TG level, but not remnant TG level. Actually, major atherogenic changes in TG-rich lipoproteins mainly occur during the postprandial phase. Secondary analyses of the Women's Health Study showed that TG levels measured 2–4 h postprandially had the strongest association with cardiovascular events [16]. Genetic studies such as Mendelian randomization studies also demonstrated that TG-rich remnant lipoproteins are causally related to atherosclerotic cardiovascular disease [17]. This may be one reason that the fasting TG level is inferior to the postprandial or non-fasting TG level as a risk factor for predicting cardiovascular disease. It has been reported that even subjects with fasting TG levels of 106–149 mg/dL are associated with increased mortality in the long-term follow-up [18].

The present results indicate that, in order to predict postprandial lipidemia, measuring a single fasting item may not be enough. Simultaneous measurement of fasting levels of TG, RLP-C, and ApoB48 may be useful for predicting postprandial lipidemia in daily practice. The set of measurements—all available clinically—may be able to substitute for fat-ingestion tests as the surrogate.

This study has several strengths. The number of subjects we enrolled was larger than in our previous studies, and we examined the utility of fasting values and the remnant and non-remnant indices proposed previously [4]. We also used a fat amount that was not excessive for Japanese women [3, 4]. This study also has some limitations. Because we restricted examinees to healthy Japanese young women with the apoE3/3 phenotype, the results may not be applicable for other populations or ethnicities.

5. Conclusion

The fasting serum TG level can predict the postprandial non-remnant, but not remnant, TG level. Postprandial lipidemia may be predictable by the measurement of the set of fasting serum TG, RLP-C, and ApoB48. For daily medical practice, without performing a fat-ingestion test, the set may be a useful device for predicting postprandial lipidemia.

Conflict of Interests

The authors declare that they have no competing interests.

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