

Postprandial Triglyceride Response in Men: Role of Overweight, Abdominal Fat and Nutrition

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Key Words

Postprandial triglyceride response · Postprandial triglyceride peak value · Triglyceride-area under curve · Fasting triglyceride levels · Cardiometabolic risk

Summary

Objective: The study analyses i) the effect of overweight, waist circumference and dietary habits on postprandial (pp) triglyceride (TG) response and compares ii) pp TG response with fasting TG levels and iii) pp TG peak values with TG-AUC (area under curve) with respect to cardiometabolic risk assessment. **Methods:** In 100 men (44–68 years) body composition (air-displacement plethysmography), dietary habits, cardiometabolic risk and pp lipid metabolism (standardised lipid load) were assessed. A pp TG peak value of 260 mg/dl was used as a cut-off to classify TG normal- and high-responders. **Results:** pp TG response had positive associations with BMI ($r = 0.24$; $p < 0.05$), fat mass ($r = 0.28$; $p < 0.01$), waist circumference ($r = 0.33$; $p < 0.01$), systolic blood pressure ($r = 0.21$; $p < 0.05$), fasting ($r = 0.29$; $p < 0.01$) and pp glucose ($r = 0.40$; $p < 0.001$), fasting ($r = 0.38$; $p < 0.001$) and pp insulin levels ($r = 0.46$; $p < 0.001$), and inverse associations with HDL-C ($r = -0.43$; $p < 0.001$) and intake of dietary fibre ($r = -0.31$; $p < 0.05$). Fasting TG levels had a greater contribution to the variance in 12 of 14 cardiometabolic risk factors compared to pp TG response. TG-AUC was highly correlated to cardiometabolic risk. **Conclusion:** Overweight, abdominal fat and a low intake of dietary fibre were determinants of increased pp TG response. Contrary to findings in younger normal-weight men, fasting TG levels had a stronger association with cardiometabolic risk compared to pp TG response. This might be explained by differences in fat mass.

Background and Aims

Obesity and its co-morbidities have become a major public health concern. Fasting triglyceride (TG) levels are positively associated with body weight and are considerable risk factors for atherosclerosis [1]. Because TG levels raise in response to dietary intake and TG-rich lipoproteins like VLDL, IDL and chylomicron remnants have a high atherogenic potential, it was proposed to assess postprandial (pp) TG levels after an oral lipid load in order to estimate cardiometabolic risk [2]. Fasting TG levels cannot precisely predict pp TG response (that is the response of TG levels to an oral lipid load and reflects the individual capacity to handle the fat intake) since some fasting normotriglyceridaemics display an exacerbated pp TG response [3]. However, a standardised and reproducible lipid load test as well as a consistent characterisation is so far lacking [4]. Pp lipaemia tests differ regarding types of test meals (e.g. milk shakes, fluids with defined ingredients, mixed meals), meal sizes and compositions (e.g. different fat contents, different amounts of other nutrients like carbohydrates, fatty acids, cholesterol and alcohol which can alter pp TG response) [4]. In addition, test meals with the same amount of nutrient/energy for every subject irrespective of body weight are used [5]. By contrast other test meals are referred to body surface area (BSA) [3] and body weight [6]. Characterisation of pp TG response is performed using pp TG peak values (TG_{ppmax}) [2] or TG-AUC (TG area under curve) which reflects occurrence, accumulation and elimination of TG in plasma [3].

Among others, pp TG response is determined by overweight, abdominal obesity [7–9] and nutrition. Pp TG response was found to be associated with cholesterol [10], energy intake [11, 12] as well as the intake of protein [13], fat, alcohol [14] dietary fibre [5] and n-3 fatty acids [6]. Furthermore, cardiometabolic risk factors such as elevated LDL-cholesterol

[2], low HDL-cholesterol, elevated glucose and insulin levels [3] as well as a high blood pressure [8] are also related to pp TG response. Taken together, a comprehensive view of pp TG response compared with basal TG levels is missing. To bring all these points together this study aims to investigate:

- i) the influence of overweight, abdominal obesity, body composition and dietary habits on pp TG response,
- ii) the importance of pp TG response when compared with fasting TG levels for assessing cardiometabolic risk,
- iii) different possibilities to characterise pp TG response (TG_{ppmax} vs. TG-AUC).

To follow these questions we have used a standardised lipid load and followed a standardised test protocol.

Material and Methods

Study Design and Population

The Metabolic Intervention Cohort Kiel (MICK) is a prospective as well as an intervention cohort as part of the research network in Kiel (Germany) 'Fat and Metabolism – Gene Variation, Gene Regulation and Gene Function'. 750 men aged 45 to 65 years were recruited through requisition of the local registration office. Exclusion criteria were endocrinological dysfunction, known disturbances in fat metabolism, diagnosed diabetes mellitus, impaired liver or kidney function, tumours and alcohol dependency. 100 men (60.3 ± 5.6 years) of Caucasian descent participated in this sub-cohort analysis and pp lipid metabolism, anthropometry, body composition and dietary habits as well as cardiovascular risk factors were assessed. The study was approved by the local ethic committee of the medical faculty of Christian-Albrechts-University in Kiel. Written informed consent was obtained from each subject. The study meets the standards of the Declaration of Helsinki in its revised version of 1975 and its amendments of 1983, 1989 and 1996.

Anthropometric Measurements and Body Composition Analysis

Body height was measured with a stadiometer to the nearest 0.5 cm (SECA, Modell 220; Vogel and Halke, Hamburg, Germany). Body weight was determined to the nearest 0.1 kg on an electronic scale coupled to the Bod Pod® system (Bod Pod®; Body Composition System; Life Measurement Instruments, Concord, CA, USA), and BMI was calculated as 'body weight (kg) / body height² (m²)'. Waist circumference was measured midway between the lowest rib and the iliac crest while the subjects were at minimal respiration. Body composition (fat mass (FM) and fat free mass (FFM)) was assessed by air-displacement plethysmography (Bod Pod), which is described elsewhere [15]. Manufacturer's software was used to calculate body density and % body fat using Siri's equation [16]. FFM was calculated as 'body weight (kg) – FM (kg)'.

Assessment of Energy and Nutrient Intake

Subjects were instructed to complete a 7-day dietary record to assess their usual dietary habits. These records were analysed for energy and nutrient intake using the software program Prodi® (PRODI 4.5 LE 2001 Expert; Wissenschaftliche Verlagsgesellschaft, Stuttgart, Germany). Underreporting (that is the incomplete declaration of ingested foods) was checked by calculating resting energy expenditure (REE) according to Müller et al. [17]. In 36 subjects REE multiplied with 1.2 showed a higher value than the declared energy intake. These dietary records were discarded because of underreporting. Underreporter (n = 36) were not older than normal-reporter (n = 61) (59.9 ± 5.2 years vs. 60.6 ± 5.6 years; p > 0.05), but had a higher BMI (29.6 ± 4.4 kg/m² vs. 26.5 ± 2.7 kg/m²; p < 0.001). Three dietary records were not sent back, leaving 61 7-day dietary records for analysis.

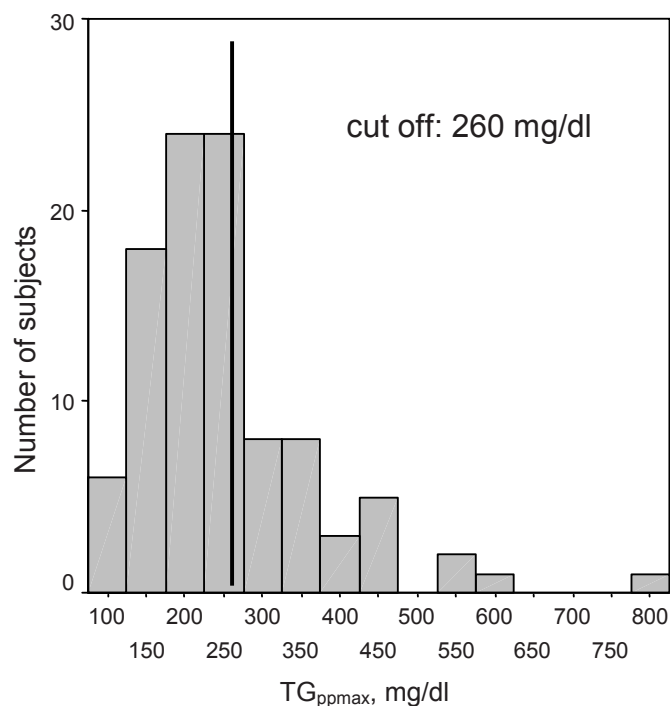


Fig. 1. Frequency distribution of subjects (n = 100) according to TG_{ppmax}.

Assessment of Cardiometabolic Risk Factors

Blood pressure was measured with a standard manual sphygmomanometer. After a minimum of 12 h fasting, a blood sample was taken for analysing total-, HDL- and LDL-cholesterol as well as TG levels enzymatically by Konelab 20i Analyzer (Konelab, Espoo, Finland). The intra-assay CVs were 1.2% for total-cholesterol, 3.5% for HDL-cholesterol, 2.7% for LDL-cholesterol and 2.5% for triglycerides. Glucose levels were determined enzymatically with Konelabs testkit (Thermo Clinical Lab-systems; intra-assay CV < 2.2%). Serum insulin levels were assessed by radioimmunoassay (Biochem Immunosystems, Freiburg i. Br., Germany; testkit of Adaltis Dt. GmbH, Freiburg i. Br., Germany; CV < 5.4%).

Excluding subjects with a fasting glucose level >7.0 mmol/l (n = 11), insulin resistance was calculated by HOMA-IR (homeostasis model assessment – insulin resistance) and β-cell function by HOMA-bcell (homeostasis model assessment – β-cell function) [18]:

$$\text{HOMA-IR} = (\text{glucose (mmol/l)} \times \text{insulin (}\mu\text{U/ml)}) / 22.5 \quad (1)$$

$$\text{HOMA-bcell} = (20 \times \text{insulin (}\mu\text{U/ml)}) / (\text{glucose (mmol/l)} - 3.5) \quad (2)$$

Assessment of pp Lipid Metabolism

After a minimum of 12 h fasting, oral metabolic tolerance test (OMTT) was performed to assess pp lipid metabolism. A catheter was placed into an antecubital vein to allow frequent blood sampling: fasting, 0.5 h, 1 h, 2 h, 3 h, 4 h, 5 h, 6 h, 7 h, 8 h and 9 h after ingestion of 500 ml of a fluid test meal, which contained the following ingredients: 4,221 kJ energy, 30 g of protein (11.9% of energy), 75 g of carbohydrate (29.6% of energy; 93% saccharose and 7% lactose), 58 g of fat (51.6% of energy; 65% saturated and 35% unsaturated fatty acids), 10 g of alcohol (6.9% of energy), 600 mg cholesterol and 30,000 IU retinylpalmitate. During examination, subjects were allowed to drink water; food intake or smoking were not allowed. Physical activity was standardised. Pp TG, glucose and insulin levels were used to calculate the AUC.

Characterisation of pp TG Response

TG_{ppmax} was used to characterise pp TG response. Applying a cut-off of 260 mg/dl [2], 64 subjects were divided into TG normal-responders

(TG_{ppmax} < 260 mg/dl) and 36 subjects into TG high-responders (TG_{ppmax} ≥ 260 mg/dl) (fig. 1).

Taking into account differences in the volume of distribution, data were adjusted for 'ingested fat / FFM (kg)'. FFM was assumed as volume of distribution of ingested fat because TG_{ppmax} was reached after 3.4 ± 1.5 h and fat is stored in adipose tissue with temporal delay [19]. It has been shown that after 4 h only one quarter of ingested fat is incorporated into

fat mass [20]. Alternative adjustments of TG_{ppmax} for 'ingested fat / weight (kg)' and 'ingested fat / BSA (cm²)' were performed in order to compare different ways to characterise pp TG response. TG-AUC (adjusted for 'ingested fat / weight (kg)') was also considered as a parameter of pp TG response. Over a period of 9 h, which reflects TG-AUC, body weight (FM and FFM) was assumed as the volume of distribution of ingested fat.

Table 1. Comparison of age, body composition and anthropometric variables between TG normal- (TG_{ppmax} < 260 mg/dl) and high-responders (TG_{ppmax} ≥ 260 mg/dl) (adjusted for 'ingested fat / FFM (kg)'; means ± SE)

	Normal-responders (n = 64)	High-responders (n = 36)
Age, years	60.2 ± 0.7	60.5 ± 0.9
Weight, kg	85.9 ± 1.2	89.6 ± 1.6
Height, m	1.77 ± 0.01	1.78 ± 0.01
BMI, kg/m ²	27.3 ± 0.4	28.4 ± 0.6
Body fat, %	27.2 ± 0.9	30.6 ± 1.2*
FFM, kg	61.9 ± 0.2	61.8 ± 0.3
Waist circumference, cm	99.1 ± 1.2	103.4 ± 1.7*

*p < 0.05; difference between TG normal- and high-responders; ANCOVA.

Statistics

All analyses were performed using SPSS for Windows (Statistical Package for the Social Sciences, version 13.0). For normally distributed parameters, descriptive statistics were given as mean ± standard deviation or standard error (SE) and otherwise (for alcohol intake, fasting TG levels, TG_{ppmax}, TG-AUC, fasting insulin levels, insulin-AUC, HOMA-IR and HOMA-bcell) as mean (95% confidence interval).

TG-, glucose- and insulin-AUC were calculated by trapezoidal rule. Analysis of covariance (ANCOVA (Bonferroni's post-hoc test)) was performed to compare TG normal- and high-responders. Relationships between two variables were tested by Pearson's correlation coefficient. Partial correlations were used to adjust for covariates. Multiple stepwise regression analysis was used to describe the relationship between one dependent and multiple independent variables. Parameters which showed no normal distribution were log-transformed for correlation and regression analysis. Chi-square test was applied to analyse differences in the frequency distribution of categorical variables. All tests were two-sided with a probability of 5%.

Table 2. Correlations between cardiometabolic risk factors and log TG_{ppmax} and log TG-AUC' in 100 men and accordingly 89 men for HOMA-IR and HOMA-bcell

	log TG _{ppmax} ^a	log TG _{ppmax} adjusted for 'ingested fat/FFM (kg)' ^b	log TG _{ppmax} adjusted for 'ingested fat/weight (kg)' ^c	log TG _{ppmax} adjusted for 'ingested fat/BSA(cm ²)' ^d	log TG-AUC adjusted for 'ingested fat/weight (kg)' ^c
BMI, kg/m ²	0.15	0.24*	0.16	0.19	0.26*
Body fat, %	0.29**	0.28**	0.30**	0.31**	0.35***
Waist circumference, cm	0.22*	0.33**	0.31**	0.32**	0.37***
log TG, mg/dl	0.89***	0.89***	0.89***	0.89***	0.93***
Cholesterol, mg/dl	0.20*	0.18	0.21*	0.21*	0.22*
LDL-cholesterol, mg/dl	0.15	0.15	0.15	0.15	0.15
HDL-cholesterol, mg/dl	-0.42***	-0.43***	-0.41***	-0.42***	-0.46***
Glucose, mg/dl	0.28**	0.29**	0.27**	0.28**	0.30**
Glucose-AUC, mg/dl × 540 min ⁻¹	0.40***	0.40***	0.39***	0.40***	0.45***
log Insulin, μU/ml	0.32**	0.38***	0.32**	0.35***	0.33**
log Insulin-AUC, μU/ml × 540 min ⁻¹	0.43***	0.46***	0.44***	0.45***	0.48***
log HOMA-IR, mmol/l × μU/ml	0.24*	0.29**	0.27*	0.27*	0.34**
log HOMA-bcell, μU/ml / mmol/l	-0.24*	-0.30**	-0.27*	-0.28**	-0.30**
RR _{sys} , mm Hg	0.17	0.21*	0.16	0.18	0.18
RR _{dias} , mm Hg	0.09	0.12	0.07	0.09	0.06

RR_{dias} = diastolic blood pressure; RR_{sys} = systolic blood pressure.

*p < 0.05; **p < 0.01; ***p < 0.001.

^aPearson's correlation coefficient.

^bPartial correlation (adjusted for 'ingested fat / FFM (kg)').

^cPartial correlation (adjusted for 'ingested fat / weight (kg)').

^dPartial correlation (adjusted for 'ingested fat / BSA (cm²)').

Table 3. Comparison of energy and nutrient intake between TG normal- (TG_{ppmax} < 260 mg/dl) and high-responders (TG_{ppmax} ≥ 260 mg/dl) (adjusted for ‘ingested fat / FFM (kg)’; means ± SE or means (95% confidence interval))

	Normal-responders (n = 40)	High-responders (n = 21)
Energy intake, kcal/day	2468 ± 51.9	2573 ± 71.7
Protein intake		
g/day	88.5 ± 3.0	96.3 ± 4.1
% energy intake	14.6 ± 0.4	15.2 ± 0.6
Fat intake		
g/day	100.4 ± 3.3	107.5 ± 4.5
% energy intake	36.4 ± 0.8	37.6 ± 1.1
Carbohydrate intake		
g/day	267.8 ± 7.6	267.2 ± 10.6
% energy intake	44.2 ± 0.9	42.4 ± 1.3
Alcohol intake		
g/day	15.8 (10.9–20.7)	17.7 (10.7–24.8)
% energy intake	4.8 (3.4–6.2)	4.8 (2.8–6.8)
Cholesterol intake, mg/day	346.1 ± 18.8	372.5 ± 25.9
SFA		
g/day	37.8 ± 1.7	38.5 ± 2.3
% energy intake	14.0 ± 0.5	13.8 ± 0.7
MUFA		
g/day	30.9 ± 1.4	34.0 ± 1.9
% energy intake	11.5 ± 0.4	12.2 ± 0.6
PUFA		
g/day	12.1 ± 0.7	14.8 ± 0.9*
% energy intake	4.5 ± 0.2	5.2 ± 0.3
Dietary fibre, g/day	26.5 ± 1.3	23.8 ± 1.7

MUFA = Monounsaturated fatty acids; PUFA = polyunsaturated fatty acids; SFA = saturated fatty acids.

*p < 0.05; differences between TG normal- and high-responders; ANCOVA.

Results

The study population consisted of 100 men aged between 44 and 68 years (60.3 ± 5.6 years), who were on average overweight (BMI 27.7 ± 3.8 kg/m²). 24% were normal weight (BMI 20–24.9 kg/m²), 57% overweight (BMI 25–29.9 kg/m²) and 19% obese (BMI ≥ 30 kg/m²). The subjects had a mean body fat of 28.4 ± 7.2%, a FFM of 61.9 ± 8.0 kg and a waist circumference of 100.6 ± 11.5 cm.

Influence of Overweight, Abdominal Obesity and Body Composition on pp TG Response

After adjusting for ‘ingested fat / FFM (kg)’, TG high-responders had a higher % body fat and a higher waist circumference when compared with TG normal-responders. Age, height, body weight, BMI and FFM showed no differences between the groups (table 1). BMI, % body fat and waist circumference were positively associated with pp TG peak values after adjusting for ‘ingested fat / FFM (kg)’ (table 2).

Influence of Dietary Habits on pp TG Response

Comparison of energy and nutrient intake between TG normal- and high-responders is shown in table 3. After adjusting for ‘ingested fat / FFM (kg)’ TG high-responders had a higher intake of polyunsaturated fatty acids (PUFA, g/day) when compared with TG normal-responders. Furthermore, the intake of dietary fibre was inversely correlated with pp TG peak values after adjusting for ‘ingested fat / FFM (kg)’ (r = –0.31; p < 0.05).

Comparison of pp and Fasting TG Levels for Assessment of Cardiometabolic Risk

Fasting TG levels were highly correlated with both parameters of pp TG response: TG_{ppmax} (r = 0.89; p < 0.001) and TG-AUC (r = 0.93; p < 0.001) (fig. 2).

Comparison of cardiometabolic risk between TG normal- and high-responders is shown in table 4. When compared with TG normal-responders, high-responders showed higher fasting TG, total- and LDL-cholesterol levels as well as a higher glucose-AUC, insulin-AUC, HOMA-IR and systolic blood pressure after adjusting for ‘ingested fat / FFM (kg)’. Furthermore, TG high-responders had lower HDL-cholesterol levels and a lower HOMA-bcell index when compared with TG normal-responders. The prevalence of the metabolic syndrome (according to NCEP-ATPIII criteria [21]) was significantly higher in TG high- vs. normal-responders (41.7 vs. 15.6%; p < 0.01).

After adjusting for ‘ingested fat / FFM (kg)’ and fasting TG levels, only LDL-cholesterol levels remained significantly different between TG normal- and high-responders (TG normal-responders 128.9 ± 4.0 mg/dl vs. high-responders 151.5 ± 5.7 mg/dl; p < 0.01).

After adjusting for ‘ingested fat / FFM (kg)’, pp TG peak values were positively associated with fasting TG and glucose levels, glucose-AUC, fasting insulin levels, insulin-AUC, HOMA-IR as well as systolic blood pressure and inversely associated with HDL-cholesterol as well as HOMA-bcell index (table 2).

Using a multiple stepwise regression analysis with cardiometabolic risk factors as dependent variables and fasting TG levels, pp TG peak values, ‘ingested fat / FFM (kg)’ and TG-AUC as independent variables, fasting TG levels explained the highest proportion of variance in 12 of 14 examined cardiometabolic risk factors (BMI, waist circumference, % body fat, systolic blood pressure, total cholesterol, HDL-cholesterol, fasting glucose levels, glucose-AUC, log HOMA-IR, log HOMA-bcell, log insulin, log insulin-AUC).

Characterisation of pp TG Response

The association between cardiometabolic risk factors and different opportunities to normalise pp TG response is shown in table 2. TG-AUC (adjusted for ‘ingested fat / weight (kg)’) showed the highest correlation coefficients in 10 of 15 examined cardiometabolic risk factors. Comparing different ways to normalise pp TG peak values (adjustments for ‘ingested fat /

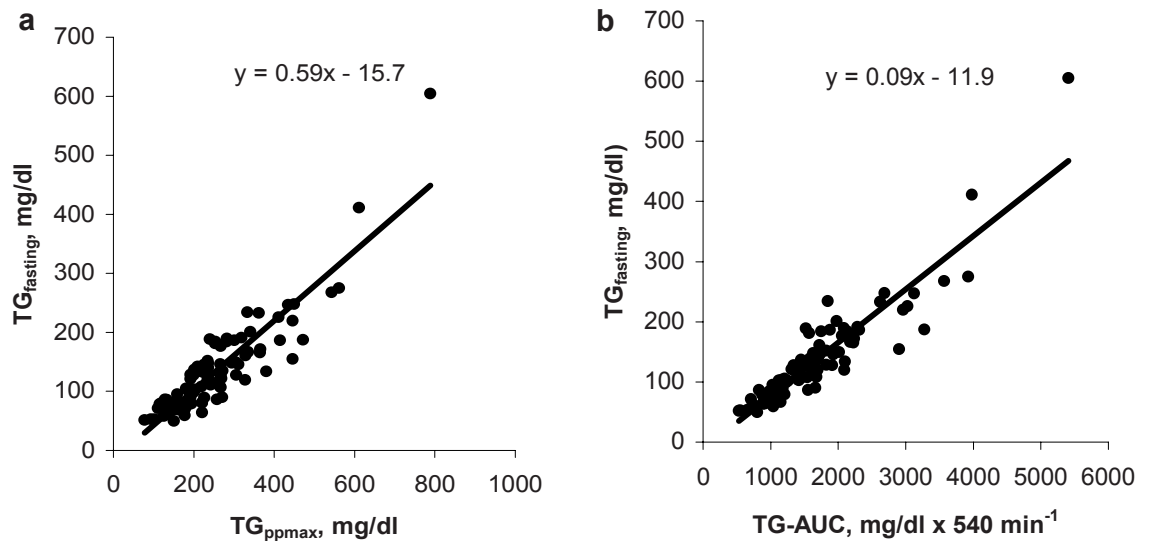


Fig. 2. Association between fasting triglyceride levels ($TG_{fasting}$) and **a** TG_{ppmax} as well as **b** $TG-AUC$ in 100 men.

Table 4. Comparison of cardiometabolic risk factors between TG normal- ($TG_{ppmax} < 260$ mg/dl) and high-responders ($TG_{ppmax} \geq 260$ mg/dl) (adjusted for 'ingested fat / FFM (kg)'; means \pm SE or means (95% confidence interval))

	Normal-responders (n = 64)	High-responders (n = 36)
Triglycerides, mg/dl	98.0 (82.8–113.2)	192.0 (171.6–212.3)***
Cholesterol, mg/dl	213.1 \pm 5.3	232.0 \pm 7.0*
LDL-cholesterol, mg/dl	130.8 \pm 3.7	148.2 \pm 4.9**
HDL-cholesterol, mg/dl	56.6 \pm 1.7	47.6 \pm 2.2**
Glucose, mg/dl	101.2 \pm 1.9	106.1 \pm 2.5
Glucose-AUC, mg/dl \times 540 min ⁻¹	498.2 \pm 10.9	558.1 \pm 14.6**
Insulin, μ U/ml	14.3 (12.1–16.4)	16.2 (13.2–19.2)
Insulin-AUC, μ U/ml \times 540 min ⁻¹	181.0 (147.5–214.4)	249.6 (205.0–294.2)*
HOMA-IR, mmol/l \times μ U/ml ^a	3.0 (2.5–3.5)	4.0 (3.3–4.7)*
HOMA-bcell, μ U/ml / mmol/l	8.2 (6.9–9.6)	5.0 (3.1–6.9)**
RR _{sys} , mm Hg	126.0 \pm 2.1	134.1 \pm 2.8*
RR _{dias} , mm Hg	79.3 \pm 1.4	81.9 \pm 1.8

RR_{dias} = diastolic blood pressure; RR_{sys} = systolic blood pressure.

*p < 0.05; **p < 0.01; ***p < 0.001; differences between TG normal- and high-responders; ANCOVA.

^aComparison between 59 TG normal- and 30 high-responders.

FFM (kg)', 'ingested fat / weight (kg)' and 'ingested fat / BSA (cm²)', pp TG peak values adjusted for 'ingested fat / FFM (kg)' showed the highest correlation coefficients in 10 of 15 examined cardiometabolic risk factors.

Discussion

Influence of Overweight, Abdominal Obesity and Body Composition on pp TG Response

In agreement with previous studies, BMI and % body fat were positively associated with pp TG response (table 2). Lewis et al. [7] found a higher pp TG response in overweight when compared with normal-weight subjects. This positive association between BMI as well as FM and pp TG response could be

due to a hypercaloric diet inducing a higher synthesis of TGs in the liver. Other studies focused on the importance of abdominal FM regarding pp TG response [22, 23]. Waist circumference was significantly higher in TG high- than in TG normal-responders (table 1) and was positively associated with pp TG response (table 2). This might be explained by insulin resistance, higher lipolysis in visceral FM, increased influx of free fatty acids via portal vein to the liver and an overproduction of TG-rich VLDL in abdominal/visceral obesity.

Influence of Dietary Habits on pp TG Response

In agreement with other studies, the intake of dietary fibre was inversely correlated to pp TG response [5, 24]. This might be due to a decreased absorption of fat, an increased clearance of chylomicrons, an elevated absorption of chylomicron remnants

or a decreased secretion of VLDL by the liver as well as a higher catabolism of VLDL induced by dietary fibre [5].

TG high-responders had a higher intake of PUFA when compared with normal-responders (table 3). Unfortunately a differentiation of PUFA intake into n-3 fatty acids was impossible from our data. Contrary to our results, other authors found an inverse relationship between the intake of n-3 fatty acids (contained e.g. in fish oils) and pp TG response [6, 12, 25]. This might be due to the inhibition of hepatic VLDL TG synthesis and to the improvement of VLDL catabolism by n-3 fatty acids [26].

Other studies found more associations between dietary habits and pp TG response: an inverse relationship between energy intake and pp TG response [12] as well as positive associations between pp TG response and the intake of protein [13], fat, carbohydrates [27] and alcohol [14] as well as cholesterol and SFA intake [10]. These associations could potentially not be obtained in this study because of 37% underreporting.

Comparison of pp and Fasting TG Levels for Assessment of Cardiometabolic Risk

Fasting TG levels were positively associated with TG_{ppmax} and TG-AUC (fig. 2). Considering this, most of the subjects with an elevated pp TG response also had elevated fasting TG levels and vice versa. However, an elevated fasting TG level did not necessarily predict an elevated pp TG response. Verifying this, 4% of the subjects only had elevated fasting TG levels, and 13% only had elevated pp TG levels.

In agreement with previous studies, pp TG response was associated with a higher prevalence of the metabolic syndrome and elevated cardiometabolic risk factors (table 2). TG high-responders had higher LDL-cholesterol levels when compared with TG normal-responders (table 4; see also [8]). HDL-cholesterol levels were inversely associated with pp TG response [table 2; see also [28]], whereas fasting glucose and insulin levels, HOMA-IR as well as glucose- and insulin-AUC showed a positive association with pp TG response (table 2; see also [22]). A higher systolic blood pressure was also related to a higher pp TG response (tables 2, 4; see also [29]).

After adjusting for 'ingested fat / FFM (kg)' and fasting TG levels, the differences in cardiometabolic risk factors between TG normal- and high-responders disappeared; only fasting LDL-cholesterol levels remained higher in TG high-responders when compared with normal-responders. Therefore, fasting TG levels had a higher impact to assess cardiometabolic risk than pp TG response. Verifying this, multiple stepwise regression analysis showed a higher explanation of variance in 12 cardiometabolic risk factors by fasting TG levels when compared with pp TG peak values and TG-AUC. This result is contrary to examinations in younger normal-weight men [2] and might be explained by differences in FM.

To get further insight, a longitudinal assessment of subjects with elevated pp TG levels with regard to the incidence of the metabolic syndrome is necessary. Furthermore, a discrimina-

tion of plasma TGs could be of interest. In a study by Carstensen et al. [30], type 2 diabetic males with prior myocardial infarction had higher responses of plasma TGs and retinyl-palmitate-labeled lipoproteins of intestinal origin when compared with a matched group without prior myocardial infarction. However, further analyses of the lipid fractions are impossible in our study, the data are not available.

Another interesting topic is the assessment of the genetic background. As our study is a sub-cohort analysis of the MICK, such analyses have already been published. For example, the study by Rubin et al. [31] dealt with the common functional exon polymorphism in the microsomal TG transfer protein gene, which was associated with type 2 diabetes, impaired glucose metabolism and insulin levels.

Characterisation of pp TG Response

Adjusting for 'ingested fat / FFM (kg)', pp TG peak values showed the highest correlations with 10 of 15 cardiometabolic risk factors when compared with the other possibilities of normalisation ('ingested fat / weight (kg)' as well as 'ingested fat / BSA (cm²)') (table 2). Other arguments in favour of adjusting pp TG peak values for 'ingested fat / FFM (kg)' were:

- i) Characterisation of pp TG response on the basis of TG_{ppmax} could lead to misinterpretation because the volume of distribution of ingested fat is not considered. A higher pp TG response would be expected in subjects with a low volume of distribution.
- ii) Adjusting TG_{ppmax} for 'ingested fat / weight (kg)' might be inappropriate because weight is not the initial volume of distribution of ingested fat [19, 20].
- iii) BSA is related to metabolic mass, so it is an indirect parameter of FFM. BSA can be calculated by body height and weight, but in an individual case this is imprecise.

TG-AUC (adjusted for 'ingested fat / weight (kg)') had the highest correlations with 10 of 15 cardiometabolic risk factors compared with the other parameters of pp TG response (table 2). Therefore, TG-AUC might be a more precise parameter to characterise pp TG response when compared with TG_{ppmax} . In contrast to TG_{ppmax} TG-AUC does not only reflect the rise/absorption and accumulation of TGs but also the elimination of TGs in plasma.

Conclusion

Elevated BMI, waist circumference as well as % body fat and a low intake of dietary fibre are associated with a higher pp TG response. When compared with pp TG peak values, TG-AUC is preferred for cardiometabolic risk assessment. There is a positive association between pp TG response and cardiometabolic risk. In contrast to studies in younger normal-weight subjects, fasting TG levels exceeded pp TG response in cardiometabolic risk assessment.

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