Postprandial triglyceride response in normolipidemic, hyperlipidemic and obese subjects – the influence of polydextrose, a non-digestible carbohydrate

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Abstract

Background: Three independent trials were conducted to evaluate postprandial triglyceride (TG) responses in subjects with different lipid metabolism. The effect of polydextrose (PDX), a soluble non-digestible carbohydrate, on postprandial response was also studied using practically relevant, high fat meal interventions.

Methods: A total of 19 normolipidemic (average BMI 24.1 kg/m²), 21 overweight/hyperlipidemic (average BMI 29.6 kg/m²) and 18 obese/non-diabetic subjects (average BMI 33.6 kg/m²) were included in the study. On two separate occasions all subjects ate two high-fat meals (4293 kJ, 36% from fat), one with PDX (either 12.5 g or 15 g) and one without PDX during placebo-controlled, double-blind, crossover and randomized trials. To obtain the triglyceride measurements venous blood samples were taken before the consumption of the test meal and five times afterwards, up to 6 h post-test meal. The triglyceride responses were modeled using a mixed-effects linear model.

Results: The key variables that explain the variation of the postprandial triglyceride response in the different subject groups were: baseline triglyceride concentration, time point, and PDX vs. placebo treatment (p < 0.05). The maximum postprandial TG concentration was more pronounced in hyperlipidemic group compared to normolipidemic (p < 0.001) or obese groups (p < 0.01). The modeled TG response analysis showed that irrespective of the study population PDX supplementation was one of the factors significantly reducing triglyceride response compared to the placebo treatment (p < 0.05).

Conclusions: Subjects with elevated fasting triglyceride levels display exaggerated and prolonged postprandial triglyceride responses. PDX, a soluble non-digestible carbohydrate, may offer a dietary concept for reducing the postprandial triglyceride response after the consumption of a meal containing a high concentration of fat.

Keywords: Postprandial triglycerides, Hyperlipidemia, Hypertriglyceridemia, Obesity, Dietary fiber
are processes that have both been presented as mechanisms which give rise to an increase in the risk of CVD due to increased levels of circulatory triglyceride [6]. PPL is also linked to increased oxidative stress, a contributor to endothelial dysfunction [7].

The effects of dietary, physiological, genetic and pathological influences on postprandial lipid metabolism have been reviewed by Lopez-Miranda et al. [3]. Insulin is an important regulator of lipid metabolism and so insulin resistance within the peripheral tissues is a link between metabolic syndrome and dyslipidemia [8]. Impaired postprandial triglyceride clearance is typically associated with the accumulation of visceral adipose tissue [9,10], increased waist circumference [11], obesity [5,12], metabolic syndrome [13], type 2 diabetes [14] as well as elevated levels of triglyceride during the fasting state [9,15]. In addition, various physiological factors and lifestyle conditions are known to increase PPL, such as age [16], male gender [17,18], some gene variants [3], menopause [19], use of alcohol [20] and a low level of physical activity [21]. Females demonstrate a less intense postprandial lipid response than men due to their increased clearance capacity enabling them to remove triglycerides from their circulatory system more quickly. Tolerance of fatty meals typically decreases with age but it may also be associated with weight gain or the menopausal phase. Conversely, increased physical exercise such as running or cycling, or even walking, can significantly reduce fasting triglyceride levels and the postprandial triglyceride response [22].

The main nutritional factor influencing postprandial lipemia is the amount of fat present in a meal. Other components such as the presence of digestible carbohydrates, fibers and alcohol can mediate an effect but the details are as yet unclear [3]. The role of digestible and indigestible carbohydrates on postprandial lipid metabolism is reviewed by Lairon et al. [23] and the involvement of dietary fiber in the prevention of metabolic syndrome components by Galisteo et al. [24]. Although an increased intake of certain dietary fibers demonstrated a tendency to decrease postprandial lipid response [25], a controversy still exists surrounding the role of different dietary fibers on lipid response [26]. Generally, there are indications that various soluble fibers reduce the levels of fasting LDL without affecting the concentration of fasting triglyceride [27]. A reduced postprandial lipolysis due to the concomitant ingestion of fibers in a meal can be explained by various mechanisms. For instance, fibers that form viscous solutions, those that generate aggregates which contain lipids, and those that directly inhibit lipase activity [23] will all impact on the rate of lipolysis. Studies have shown that a diet enriched with oat bran is able to reduce fat absorption demonstrated by a measured increase in faecal fat associated with such diets [28].

Polydextrose (PDX), a non-digestible carbohydrate was used to test the efficacy of this fiber to reduce the triglyceride response. PDX is a highly branched, randomly bonded glucose polymer [29]. Due to its complex structure and profile of glycosidic bonds mammalian digestive enzymes are unable to digest PDX. Instead it is slowly fermented during its passage through the colon by colonic microbes producing short-chain fatty acids (SCFAs) [30,31]. PDX is a well-tolerated [32], low-caloric [29] fiber that can be easily incorporated into various food applications to reduce energy content and replace sugar and fat [33]. It has been recognized as soluble fiber, and human clinical, animal and in vitro studies have demonstrated several physiological effects associated with these features [34]. There are also indications that PDX can increase fasting HDL [35] and decrease postprandial triglyceride [36] concentrations.

Due to the importance of non-fasting triglyceride as a risk marker for cardiovascular diseases there is a need to understand the role of different nutrients which affect triglyceride metabolic regulation. However, there are very few studies which directly compare subjects with different lipid metabolism. This study examines postprandial lipid responses in normolipidemic, obese and hyperlipidemic subjects. The postprandial challenge model used in this study may provide a practical tool to test the efficacy of different nutrients to lower the triglyceride response.

Methods
Participants
The study was conducted in three research centers located in southern and central Finland. The study protocol of the normolipidemic subjects was approved by the Sports Institute of Finland (4.12.2004/Polydextrose study), the protocol for obese subjects by the Research Ethics Committee from the Hospital District of Northern Savo, Finland (2007/123) and the hyperlipidemic protocol by the Ethical Committee, Intermunicipal Hospital District of Southwest Finland (29.8.2006/346). The study was conducted according to the guidelines laid down in the Declaration of Helsinki. The purpose of the study was explained to participants who gave their written informed consent before being included in the study.

This study consists of three study populations: normolipidemic young adults, mildly hyperlipidemic overweight adults and obese non-diabetic adults. The main inclusion criteria for the normolipidemic subjects were total cholesterol < 5.0 mmol/l, triglyceride < 1.5 mmol/l and BMI < 30 kg/m². The main inclusion criteria for the hyperlipidemic overweight subjects were fasting triglyceride 1.5-2.5 mmol/l. The main inclusion criteria for the obese non-diabetic subjects were BMI 30–37 kg/m². Exclusion criteria for all the groups were: use of lipid lowering medication, antiobesity drugs, dietary supplements with high fiber content, pregnancy, cardiovascular
conditions and metabolic diseases. A structured interview on previous and current diseases, current use of medica-
tion and alcohol and tobacco consumption was carried out during a screening visit to clarify the health status of
the subjects and their suitability for the study. In addition,
body weight and height were measured and fasting blood
samples were taken at this time.

Study design
All the studies were conducted as randomized, double-blind
placebo-controlled, cross-over trials. Each study consisted of
two periods (postprandial interventions) and a wash-out
period of approximately 10 days between interventions. A
standard high fat, hamburger meal was used as a postpran-
dial lipidemia model as described by Ahotupa et al. [37].

Body weight and height were determined during the
screening visit. All subjects were requested to keep their
lifestyles and body weight consistent during the study.
Subjects were also advised to avoid strenuous exercise and
not to drink alcohol for 24 h before the test days and
to avoid fat-rich foods on the day prior to the trial.
Before the first postprandial test day subjects recorded
everything they ate after 3 p.m. and they were encouraged
to eat similarly during the day before the second postpran-
dial test day. This was to standardize the diet on the day
before the postprandial study. Use of nicotine containing
products (maximal use: 10 cigarettes or equivalent daily)
were noted from 24 hours after study commenced.
The normolipidemic and hyperlipidemic groups of
subjects arrived at the trial center the morning after an
overnight fast of 10–12 hours. Three hours before the
high-fat test meal they were fed a light breakfast con-
taining 1 sandwich with ham and cheese, and a glass of
juice (total 2.9 g fat, 738 kJ). The obese group of subjects
did not receive breakfast. Instead they were fed a high-
fat test meal immediately after their overnight fast. The
difference between the fasting and non-fasting baseline
will be discussed later. All subjects ate a high-fat test
meal (4293 kJ, 36% from fat). A drink was also provided
which may or may not have included the test product, i.
e. it was administered in a randomized order. The inter-
vention meals were served at 10 a.m. and the last blood
samples were taken at 4.30 p.m. Participants were given
20 minutes to consume the test meal and they were not
allowed to consume any additional food during the 6-
hour testing period, only drinking water was permitted.
The subjects spent the trial days in the laboratory sitting
and reading and all physical exercise was forbidden.
Venous blood samples were collected twice before and
five times after the study meal.

Composition of the study meal
The study meal consisted of a standard hamburger, french-
fries and carbonated drink. The energy and nutrient content
of the study meal is presented in Table 1. The experimen-
tal meals contained either 12.5 g (normolipidemic group)
or 15 g (hyperlipidemic and obese groups) of PDX
(Litesse™ Ultra™, DuPont) added to the carbonated drink
(400 mL). The drink without PDX acted as the placebo.
The PDX dose was lower for the (lighter) normolipidemic
subjects than obese and hyperlipidemic subjects, however
the amount of PDX per kg of weight across all three sub-
ject groups was equal (p > 0.05). The addition of PDX
(4 kJ/g) to the drink increased the energy content of the
meal compared to the placebo meal by 50 kJ in the nor-
molipidemic group and 60 kJ in the obese and hyperlipid-
emic groups. A blinded sensory evaluation ensured that
PDX did not change the appearance or taste of the drink.
Two hundred mL of water was served to the participants
two and four hours after the meal.

Blood sampling and analysis
Fasting blood samples were taken during the initial
screening and used to analyze lipid profile, plasma glucose
and serum insulin concentrations. The venous blood sam-
ple was taken at 4.30 p.m. and serum insulin concentra-
tions were analysed by enzymatic - colorimetric
assay using an automatic analyser (Roche/Hitachi
MODULAR ANALYTICS, Roche Diagnostics GmbH,
D-68298 Mannheim, Germany) and commercial reagents
(Cholesterol CHOD-PAP Cat. NO. 11875540, HDL-
Cholesterol Cat. No. 04713214, LDL-Cholesterol Cat.
No. 03038777, Triglycerides GPO-PAP Cat. No. 11730711,
Roche Diagnostics GmbH, Mannheim, Germany). The
plasma-LDL concentrations were calculated using the
Friedewald formula. Plasma-glucose concentrations were
analysed by the hexokinase method using citrate-fluoride
plasma and serum insulin concentrations to create an
immunoluminometric assay which was evaluated using an
Immulite 2000 Analyzer (Thermo Fisher Scientific Inc.).

Statistical analyses
The triglyceride response data were modeled using a lin-
ear mixed effects model with fixed effects for treatment,
time point, and their interaction well as covariates for
baseline, gender, age and BMI. The model also had
nested random effects according to the subgroup struc-
ture of the data: intercept terms for study and subject
within study. Prior to modeling, the data were transformed using logarithmic transform in order to obtain good model fit to data.

The 6-hour incremental area under curve (iAUC; ignoring the area below fasting level), peak concentration (C\text{max}) and time to reach peak concentration (T\text{max}) comparisons were performed either using one-way ANOVA followed by pairwise comparisons using Tukey’s HSD test or Kruskal-Wallis test followed by pairwise comparisons using Mann-Whitney U test and Holm-Bonferroni p-value correction.

All the statistics calculations were performed using R software (R Core Team, R Foundation for Statistical Computing, version 3.1.2 [38] and nlme library, version 3.1-118, [39]).

Results
Characteristics of the study participants
Twenty eight males and thirty females participated in the studies. The obese group experienced one subject drop-out due to personal reasons. All other subjects successfully completed the combination of two postprandial test days. The baseline characteristics of participants are summarized in Table 2. The hyperlipidemic group had more males than females (14/7) and in the obese group the ratio was vice versa (5/13). As expected, results for the normolipidemic group demonstrated that plasma glucose, triglyceride, total cholesterol and LDL concentrations were significantly lower and HDL higher compared to the obese and the hyperlipidemic groups. The mean age of the normolipidemic subjects (range 20–25 years) was significantly lower than in the other groups (range 21–58 years). On the other hand, obese subjects showed significantly lower plasma glucose and total cholesterol and also higher HDL concentrations than the hyperlipidemic subjects. Both obese and hyperlipidemic subjects were only mildly hyperlipidemic.

Postprandial triglyceride responses in different study groups
Postprandial changes in plasma triglyceride levels among different study groups are shown in Figure 1. The corresponding kinetic parameters: iAUC, C\text{max} and T\text{max} are shown in Table 3. TG C\text{max} in normolipidemic group was significantly lower compared to hyperlipidemic (p < 0.001) or obese (p < 0.05) groups (1.8 ± 0.5, 2.48 ± 1.3 and 3.32 ± 1.47 mmol/l, respectively). TG C\text{max} was also significantly higher in hyperlipidemic group compared to obese group (p < 0.01). Postprandial TG T\text{max} occurred significantly earlier in normolipidemic (179 min) group compared to obese (252 min) (p < 0.01) or hyperlipidemic (271 min).

Table 1 The energy and nutrient content of the study meal

|                | Weight (g)/Volume (ml) | Energy (kJ) | Protein (g) | Carbohydrates (g) | Fat (g) | Fiber (g) | Salt (g) |
|----------------|------------------------|-------------|-------------|-------------------|---------|-----------|----------|
| Hamburger      | 219                    | 2071        | 27          | 40 (incl. 8 g sugar) | 25 (SFA 10 g) | 3         | 2.3      |
| French Fries   | 114                    | 1423        | 5           | 42 (incl. 1 g sugar) | 17 (SFA 3 g) | 4         | 0.4      |
| Carbonated drink | 450                  | 799         | <0.1        | 42                | <0.1    | 0         | 0        |
| Total          | 783                    | 4293        | 32          | 124               | 42      | 7         | 2.7      |

SFA, saturated fatty acid.

Table 2 The demographic and clinical characteristics of the study participants

| Characteristic          | Normolipidemic | Obese  | Hyperlipidemic |
|-------------------------|----------------|--------|----------------|
| Men/Women*              | 9/10           | 5/13   | 14/7           |
| Age (years)             | 22.1 (20–25)   | 42.0 (26–53)*** | 42.0 (21–58)*** |
| Weight (kg)             | 71.9 (60–88)   | 94.8 (73–114)*** | 88.6 (67–112)*** |
| BMI (kg/m\text{2})      | 24.1 (20–28)   | 33.6 (30–37)*** | 29.6 (21–37)***### |
| Glucose (mmol/L)        | 4.53 ± 0.52    | 5.65 ± 0.58*** | 6.66 ± 1.66***# |
| Insulin (mU/L)          | 10.26 ± 4.89   | 9.75 ± 6.81  | 11.29 ± 7.22    |
| TG (mmol/L)             | 1.02 ± 0.32    | 1.71 ± 0.96** | 1.62 ± 0.62***  |
| Total cholesterol (mmol/L) | 4.24 ± 0.84   | 5.28 ± 1.19** | 6.32 ± 1.54***# |
| LDL-cholesterol (mmol/L) | 2.45 ± 0.54   | 3.12 ± 0.96*  | 3.34 ± 0.83***  |
| HDL-cholesterol (mmol/L) | 2.33 ± 0.20   | 1.43 ± 0.51*** | 1.05 ± 0.30*### |

Values are expressed as ratios*, mean values with their ranges (min-max) or standard deviations (±SD).

BMI, body mass index; TG, triglyceride; LDL, low density lipoprotein; HDL, high density lipoprotein.

Student’s t-test was used to test statistical differences between the study groups.

*p < 0.05, **p < 0.01, ***p < 0.001, compared to normolipidemic.

#p < 0.05, ##p < 0.001 compared to obese.
Normolipidemic subjects showed lower iAUC compared to the obese (p < 0.01) or hyperlipidemic (p < 0.001) subjects.

The effect of PDX on the iAUC in different study groups is shown in Figure 2. Due to the high biological variation of plasma triglyceride responses no significant differences in iAUC between placebo and PDX treatments were recorded. The effect of PDX on the individual triglyceride responses are shown in Figure 3. The curves represent the difference between the placebo response and PDX response for each individual.

Factors affecting postprandial triglyceride responses
The baseline triglyceride value, PDX treatment, and time-point were all statistically significant (p < 0.05) factors explaining the differences in the postprandial triglyceride responses in the linear mixed-effects model. Irrespective of the study subpopulation, PDX supplementation resulted in lower values for the triglyceride response than the placebo treatment demonstrated by a statistically significant difference (p < 0.05). Notably the strongest decrease in response was observed in the normolipidemic group.

Discussion
This study assessed triglyceride responses in subjects with both normal and compromised lipid types of metabolism. Results demonstrate that postprandial triglyceride responses were clearly elevated in those subjects with the highest baseline blood triglyceride concentrations. However, screening for hypertriglyceridemia which was based on fasting triglyceride levels is not able to highlight a definitive group of subjects with increased risk of excessive PPL. The variation in fasting triglyceride concentration was high amongst all the subjects and many the hyperlipidemic subpopulation exhibited only mild hyperlipidemia during the intervention (1.62 ± 0.62 mmol/l) despite being identified as hyperlipidemic according to the screenings data (1.5–2.5 mmol/l). Even though it was difficult to categorize hyperlipidemic subjects, the findings of

| Variable          | Normolipidemic | Obese     | Hyperlipidemic |
|-------------------|----------------|-----------|----------------|
| iAUC (mmol/l × min) | 135.0 ± 86     | 245.8 ± 147* | 328.7 ± 190***## |
| $C_{\text{max}}$ (mmol/l) | 1.8 ± 0.5      | 2.48 ± 1.3* | 3.32 ± 1.47****## |
| $T_{\text{max}}$ (min)  | 179.1 ± 102    | 251.7 ± 72.9** | 271.4 ± 70.4*** |

Values are shown as means ± standard deviations.
(iAUC), incremental area under curve; ($C_{\text{max}}$), maximum concentration; ($T_{\text{max}}$) time to achieve maximum concentration.
*p < 0.05, **p < 0.01, ***p < 0.001, compared to normolipidemic.
##p < 0.01 compared to obese.
this study are consistent with earlier findings which have shown that subjects with fasting hypertriglyceridemia display exaggerated and prolonged triglyceride responses [11,40]. In hyperlipidemic subjects the peak value was reached an average of 4 h 30 min after the test meal. In obese subjects this peak was reached slightly earlier (4 h 10 min), whereas for normolipidemic subjects the peak was reached by 3 h. The elevated triglyceride response in hyperlipidemic subjects that continued into the late post-prandial phase (5–8 h) is associated with an increased risk of CVD [4].

Obesity is known to be an independent risk factor for cardiovascular diseases and it is linked to hyperlipidemia [41], however not all obese subjects in this study showed an excessive triglyceride response. Further, the statistical models showed that BMI did not significantly affect the triglyceride response. Generally, the magnitude of the triglyceride response of the obese group sits in between the normolipidemic and the hyperlipidemic groups. This emphasizes the importance to study the differences in metabolically healthy and abnormal obese subjects [42].

This study did not use the postprandial fat tolerance test to evaluate cardiovascular risk factors in patients [1] but focused on the typical meal induced triglyceride response in different target populations. However, the studies on the different target groups were performed in different years, environments and pre-breakfast status which presented some methodological limitations. More specifically, the normolipidemic subjects were mainly young, healthy subjects with a limited age range possibly explaining this group’s more limited variation in triglyceride response as compared to that for the hyperlipidemic and obese subjects. Moreover, studies with hyperlipidemic and obese subjects were bi-centric whereas normolipidemic study was single centric. Also, the unbalanced distribution of males to females in the obese and hyperlipidemic groups was a disadvantage to the study, but conversely the statistical analysis demonstrated that gender was not a factor that significantly affected triglyceride responses. Also, for this study the obese subjects were fasting in the true sense of its definition (10–12 h before the test meal) whereas the hyperlipidemic and normolipidemic subjects had a light breakfast 3 hours before the test meal. However, based on the study by Sundvall et al. [43] we propose that this light breakfast (2.9 g fat, 738 kJ) did not significantly alter the subsequent post-prandial effect caused by the fatty, high caloric test meal (42 g of fat and 4293 kJ). Although these methodological differences within the studies make it somewhat difficult to draw definite conclusions of the differences between the study groups this study provides information for a real-life postprandial lipid challenge among different populations. Moreover the effect of polydextrose was measured in a practical fiber dose and in a realistic meal context.

Although most epidemiological studies have shown that high-fasting triglyceride levels are a CVD risk factor [44,45], postprandial triglyceride responses have recently gained more attention as an independent risk factor for the development of atherosclerosis [46-48]. Hokanson and Austin [44] have determined that 1 mmol/L higher-fasting triglyceride is associated with a 14% increased risk of CVD after adjustment with other risk factors. The importance of addressing triglyceride responses, and dietary ways of reducing these responses, is underlined by the fact that
approximately two thirds of cardiovascular events have been estimated to persist in spite of effective statin treatment [8]. Statins have been described as not as effective in lowering blood triglyceride as they are for lowering LDL.

Due to the large inter-person and daily variations in PPL the mixed-effects linear model was used to clarify the factors affecting the PPL response. The model considered the data as one population with nested substructure based on groups and individual subjects. It showed that PDX supplementation was one of the factors responsible for decreasing significantly PPL. However, the effect of PDX supplementation on PPL was not significant if the TG response was analysed only within the study groups (Figure 2).

Other proposed means for decreasing postprandial triglyceride responses include adding n-3 fatty acids, certain types of proteins or adding carbohydrates to the diet [23,49,50]. The present study demonstrated that supplementing a high fat meal with PDX can decrease postprandial triglyceride concentrations. This effect was most pronounced in the study’s normolipidemic group. Previous studies have also shown that the addition of a dietary fiber, especially derived from oat bran, can reduce postprandial lipemic responses especially in normal-weight subjects [23,25,36]. Further, a study by Shimomura et al. [36] demonstrated that PDX in combination with lactitol and calcium was shown to reduce the rate of increase in plasma triglyceride concentrations after the ingestion of chocolate. As well as dietary fiber and other dietary components, there are further factors that affect postprandial lipidemia, such as exercise and smoking [3]. For this study we limited the impact of these further factors on triglyceride responses. For example exercise habits [21] were controlled the day before the study and alcohol consumption was prohibited due to its negative effects on postprandial lipidemia [20], whilst in moderation it may have beneficial effects on low grade inflammatory conditions.

Regulation of lipid metabolism by dietary fibers is typically thought to be associated with the physicochemical properties of soluble fibers, such as viscosity [51]. However, this alone does not explain the different outcomes of the postprandial lipid studies with dietary fibers [26]. In this study, the low-viscous PDX reduced the postprandial triglyceride response induced by a high fat meal. The effect of PDX on the individual triglyceride responses in Figure 3 indicates that PDX has an effect on the kinetics of TG absorption. Although PDX seems to decrease the overall TG response it may even increase the TG absorption shortly after the meal. Although the mechanism is yet unclear, dietary fibers are known to modify lipid digestion and absorption [26]. In addition, the colonic fermentation of non-digestible carbohydrates and generation of subsequent metabolites may modulate lipid metabolism systemically or locally within the intestine. PDX is partially
fermented in the colon and its fermentation metabolites have been shown in vitro to regulate the major genes (e.g. PPARα, PCG-1α, Lipin 1) involved with energy metabolism [52]. In a recent study by Olli et al. [53] PDX increased postprandial GLP-1 response which may partially explain the reduced triglyceride absorption of the PDX treatment.

Conclusions
Postprandial triglyceride responses were most clearly exaggerated and prolonged in subjects with the highest baseline triglyceride concentrations. However, identifying the subjects with increased risk for excessive PPL is challenging due to the high intra-individual variation in the baseline triglyceride concentrations. The modelled triglyceride response of the combined study groups showed that in addition to the baseline triglyceride concentration also PDX treatment influenced the postprandial triglyceride response. Notably the decrease in postprandial TG with PDX supplementation was strongest in normolipidemic subjects. The decreased triglyceride response achieved by supplementing a high fat meal with PDX, may offer a practically relevant means of reducing atherosclerotic risk factors.

Abbreviations
BMI: Body mass index; CAD: Coronary artery disease; Cmax: Peak concentration; CVD: Cardiovascular disease; HDL: High density lipoprotein; iAUC: Incremental area under the curve; LDL: Low density lipoprotein; PDX: Polydextrose; PPL: Postprandial lipaemia; SCFA: Short chain fatty acid; SD: Standard deviation; SFA: Saturated fatty acid; TC: Total cholesterol; TG: Triglyceride; Tpeak: Time to reach peak concentration; T2D: Type 2 diabetes.

Competing interests
This work was funded by and polydextrose provided by DuPont Nutrition and Health. KT, KO, JS and NR are or have been employed by a manufacturer of polydextrose (DuPont, formerly Danisco). The authors declare no other competing interests regarding this study.

Authors’ contributions
All authors contributed to the preparation of the manuscript and the interpretation of the results. KT, NR and TV co-designed the study. In addition TV planned the study protocols and data collection, KT was responsible of the coordination of studies and manuscript preparation. MA supervised the plasma analysis and contributed to data interpretation. EA contributed to the data management and statistical analysis. In addition JS contributed to the content revision and the language of the manuscript. All authors have read and approved the final manuscript.

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