

**Comparison of the effects of rapeseed oil, olive oil and hydrogenated plant oil  
on postprandial lipids and fatty acid oxidation**

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**Abbreviations:**

BMI: body-mass-index  
HDL: high density lipoprotein  
LDL: low density lipoprotein  
PPAR: peroxisome proliferator activated receptor  
TG: triacylglycerols  
VLDL: very low density lipoprotein

**Key words:** rapeseed oil, olive oil; fatty acid oxidation, <sup>13</sup>C, PPAR

**Abstract**

Olive and rapeseed oil are both considered beneficial for lipid metabolism because of their high content of monounsaturated fatty acids. We compared postprandial lipid metabolism after test meals with these plant oils including hydrogenated plant oil as a reference.

**Subjects and Methods:** Twenty normolipidemic subjects (age:  $25.1 \pm 4.9$  year,  $M \pm SEM$ , body mass index  $21.5 \pm 0.6$  kg/m<sup>2</sup>) underwent three one week diet periods in a cross over study design with consumption of muffins containing either olive, rapeseed or hydrogenated oil. On the seventh day of each diet week subjects consumed a test meal with 0.5 g of the corresponding oil and 0.5 mg <sup>13</sup>C labelled linoleic acid per kg body weight after a 12 h fast. Blood was sampled before the meal and in hourly intervals thereafter. Breath was collected at baseline and in half hourly intervals. After 8 hours sampling was stopped.

**Results:** Fasted plasma phospholipid fatty acids showed significantly higher oleic acid after olive oil ( $12.3 \pm 0.3$  %), significantly higher  $\alpha$ -linolenic acid after rapeseed oil ( $0.32 \pm 0.03$  %) and significantly higher palmitic acid after hydrogenated oil ( $30.3 \pm 0.3$  %), although stool fat indicated that hydrogenated oil was not adequately absorbed. Fasted plasma triacylglycerol and cholesterol (total, LDL, VLDL and HDL) were not different between the diets. Postprandial increase of triacylglycerol was similar with olive and rapeseed oil, but lower after hydrogenated oil reflecting its poor absorption. Cumulative recovery of <sup>13</sup>C after 8 h was  $10.7 \pm 0.6$  % after olive oil,  $11.6 \pm 0.6$  % after hydrogenated oil and  $13.1 \pm 0.8$  % after rapeseed oil, indicating significantly greater linoleic oxidation after rapeseed oil.

**Conclusion:** Postprandial lipid metabolism is similar with rapeseed and olive oils. Rapeseed oil enhances linoleic acid oxidation, possibly mediated by an interaction of n-3 fatty acids with peroxisome proliferator activated receptors.

## Introduction

Monounsaturated and polyunsaturated fatty acids have favourable effects on plasma lipid and lipoprotein concentrations regarding atherogenic risk, particularly in hypercholesterolemic and in hypertriglyceridemic subjects <sup>1</sup>. Meta analyses of the results of numerous studies resulted in regression equations, which enable a prediction of the cholesterol lowering effect of individual fatty acid classes or even individual fatty acids <sup>2;3</sup>. Based on these calculations rapeseed oil and olive oil are recommended as dietary oils because they contain high amounts of monounsaturated fatty acids and have beneficial effects on the lipoprotein profile in normo- and hyperlipidemic subjects. Olive oil is a major component of the Mediterranean diet, which is associated with low incidence of cardiovascular disease, and rapeseed oil induced beneficial effects on cardiovascular disease risk in the Lyon Diet Heart study <sup>4;5</sup>. Lichtenstein et al reported similar effects on total and LDL cholesterol of diets providing mainly corn oil, olive oil and canola oil, with the highest total cholesterol decline (12%) obtained after 32 days on the canola oil diet <sup>6</sup>. With similar results found also in other studies, diets high in monounsaturated fatty acids are recommended for the reduction of cardiovascular risk <sup>7-9</sup>.

A relationship between triacylglycerol (TG) concentrations both in the fasted state and postprandially and the risk for cardiovascular disease is increasingly recognized. The atherogenic effect differs between large and small TG rich particles, which is mediated by the contribution of chylomicron remnants to the more atherogenic small, dense LDL particles and the formation of foam cells <sup>10</sup>. Moreover, postprandial lipemia is associated with decreased plasminogen activator type 1 activity and increased coagulant factor VII activity <sup>10</sup>.

Since little is known on the relative effects of rapeseed and olive oil on postprandial lipoprotein metabolism, we performed a crossover study to elucidate those effects as

well as the a possible influence on lipid oxidation. We also included hydrogenated plant oil diet as a saturated fat reference group.

## Subjects and Methods

The study was designed as a randomised crossover study, in which all subjects consumed low fat basal diets to which muffins were added for one week each based on either olive oil, rapeseed oil or hydrogenated plant oil (table 1). The subjects were instructed to eat 4 muffins per day. At the last day of each dietary period a fat tolerance test was done, including 0.5 g per kg of body weight of the test fat that had been supplied with the muffin. Diet periods were performed directly one after the other without washout periods on habitual diets. The study protocol was reviewed by the Ethics Committee of the Landesärztekammer (Board of Physicians) of Bavaria, and all subjects gave written informed consent prior to enrolment.

Subjects of both sexes were included if they met the inclusion criteria: age  $\geq 18$  years, apparently healthy, normal body weight with body-mass index (BMI) between 18 and 25 kg/m<sup>2</sup>, no intake of lipid lowering drugs during the last three months, fasted plasma TG between 70 and 250 mg/dl, and fasted plasma cholesterol <200 mg/dl. A total of 20 subjects were recruited, and all underwent all three dietary periods. During each of the dietary periods subjects kept 3 day dietary records, which were evaluated using the Bundeslebensmittelschlüssel 2.1 and the evaluation software PRODI (Wissenschaftliche Verlagsgesellschaft. GmbH, Stuttgart, Germany). At the last day of each diet period (day 7), subjects were admitted to the hospital and a basal blood sample was taken after a 12 hour fasting period. The blood sample was divided and EDTA or lithium heparine, respectively, were added as anticoagulants to two aliquots. Plasma samples were frozen for later analysis. Basal breath samples were taken by having the subjects exhale into a breath bag and subsequent transfer of an aliquot of the exhaled air into an evacuated glass tube (Labco Limited, Buckinghamshire, UK). After the basal samples, subjects consumed the test muffins within 10 minutes. Muffins were prepared from flour, sugar, egg white, fat free curd cheese

and the test fat and contained per 100 g some 383 kcal, 41 g carbohydrate (44 % of energy), 10 g protein (11 % of energy) and 19 g fat (45 % of energy). The amount of muffins consumed was adjusted to ensure that each subject consumed 0.5 g fat per kg body weight, thus consequently the energy intake varied from subject to subject, but a constant relationship of macronutrient intakes was ensured.

With the test meal the subjects consumed 0.5 mg uniformly  $^{13}\text{C}$  labelled linoleic acid per kg body weight. Unesterified  $^{13}\text{C}$  linoleic acid was given as a droplet on a sugar cube and was consumed after about half of the muffins were ingested. With the test meal 200 ml of water were consumed, but additional liquid intake was not allowed for the next 2 hours, while afterwards water and unsweetened tea could be consumed ad libitum. Food intake was only allowed after the end of the sampling period. Indirect calorimetry was performed about two hours after the meal, with the subject in lying position and a ventilated hood above the subjects head. Using the deltatrac II (Hoyer, Bremen, Germany)  $\text{CO}_2$  production and oxygen consumption were measured and together with the estimated nitrogen excretion total energy expenditure was calculated.

Blood samples were taken in hourly intervals and breath samples in 30 min intervals until 8 hours after the test meal following the procedures as described for the basal samples.

### **Laboratory analyses**

Plasma lipids were analysed enzymatically using an auto analyser and corresponding reagents from Boehringer (Mannheim, Germany). While total cholesterol and TG were analysed directly, VLDL, HDL and LDL cholesterol were analysed after precipitation of other lipoproteins with tungstic phosphoric acid or dextrane sulphate. Glucose, insulin aspartate-amino-transferase, alanine-amino-transferase,  $\gamma$ -glutamyl-

transferase and bilirubine were determined by hospital routine methods from lithium heparine plasma.

From the fasted plasma samples phospholipid fatty acid composition was determined, as described previously <sup>11</sup>. Briefly, lipids were extracted from 250 µl plasma into hexane/isopropanol, phospholipids were isolated by thin layer chromatography, fatty acid methyl esters were synthesised under acidic conditions and quantified by capillary gas chromatography, using a BPX 70 column (60 m length, 0.32 mm inner diameter, SGE, Darmstadt, Germany).

<sup>13</sup>C content of the breath samples was determined by gas isotope ratio mass spectrometry after chromatographic purification of CO<sub>2</sub> <sup>12</sup>.

### **Calculations and statistics**

Areas under the curve of postprandial glucose and TG increase were calculated by averaging neighbouring values and multiplication of the averages by the time differences, yielding mg\*h/dl.

The exhaled amount of tracer per time was calculated by multiplication of the amount of CO<sub>2</sub> produced per time and the increase in <sup>13</sup>C content of CO<sub>2</sub>. Total recovery was obtained by numerical integration of the <sup>13</sup>CO<sub>2</sub> production over time <sup>12</sup>. Results are given as percentage of the dose applied, to eliminate the influence of variations of the tracer dose.

All results are given as means and standard errors of the mean (SEM). Statistical calculations were performed with the Software package for social sciences (SPSS, Chicago, IL, USA, Version 12.0). Repeated measures ANOVA was used for comparisons between dietary regimes, after Kolmogorov-Smirnov tests had shown that data for all parameters seemed to be normally distributed. P-values below 0.05 were considered statistically significant.

## Results

From April 2003 until Dec 2003, 11 female and 9 male subjects were recruited who participated in all the 3 dietary regimes. Their age at enrolment was  $25.1 \pm 4.9$  years ( $M \pm SEM$ ), body weight  $65.4 \pm 10.9$  kg, height  $174.0 \pm 2.2$  cm and BMI  $21.5 \pm 0.6$  kg/m<sup>2</sup>. At the initial screening test total cholesterol was  $168.6 \pm 4.8$  mg/dl and TG  $92.6 \pm 7.7$  mg/dl. Analysing the plasma samples from the first tests with hydrogenated oil revealed that the post test meal increase of plasma TG was unrealistically low, raising severe doubts regarding a near complete absorption of this test fat. Thus in the remaining test periods from all subjects stool samples were collected and the fat content determined. Stool fat content during the intake of the muffins prepared from hydrogenated plant oil was  $8.2 \pm 0.8$  %, which was significantly higher than with olive oil ( $4.0 \pm 0.5$  %) or rapeseed oil ( $2.6 \pm 0.2$  %), respectively. Therefore, although the results obtained after hydrogenated plant oil intake are presented here, differences to the other diet groups can not be attributed only to different metabolic effects of absorbed fatty acids but must be considered to largely depend also on lower fat absorption. The dietary records kept during each period revealed that there were no differences in the intake of the macronutrients (fat, carbohydrates, protein) and total energy intake between the periods, but depending on the test fat the contribution of individual fatty acids to total energy intake differed significantly (Table 2). Thus polyunsaturated fatty acid intakes were higher during the rapeseed week, monounsaturated fatty acid intakes higher during the olive oil week and saturated fat intakes higher during the hydrogenated fat week. This was reflected in the fatty acid composition of plasma phospholipids, with significantly higher palmitic acid under hardened fat, higher oleic acid while the subjects were consuming olive oil and higher  $\alpha$ -linolenic acid, when they took rapeseed oil (Table 3). The differences in linoleic acid intake introduced a non significant trend towards a higher percentage during rapeseed oil consumption.



The other biochemical and safety parameters tested did not differ between diet periods (Tables 3 and 4). Total cholesterol or TG did not change with the study diets. The test meals resulted in increasing postprandial plasma TG, which were virtually identical between olive oil and rapeseed oil, whereas hydrogenated fat showed a much smaller response which was ascribed to a lower fat absorption compared to the other oils (Fig. 1). While TG increased, NEFA showed a significant decrease compared to the fasted values at the time 1 h, 2 h, 3 h and 4 h with all types of test meal, and with rapeseed and olive oil also at 5 hours (Fig. 1). The postprandial increase of TG was accompanied by a decrease in total cholesterol, HDL-cholesterol and LDL-cholesterol, while VLDL-cholesterol showed significantly increased values after olive oil and rapeseed oil meals one, two and three hours after the meal (Fig. 2). Glucose showed significantly elevated values at 1 hour after the rapeseed and the olive oil meals (Fig. 3). Insulin concentrations were significantly increased above basal values at 1, 2 and 3 hours after all meal intakes, independent of the dietary fat, and below baseline from 6 h onwards with all diets (Fig. 3).

The integrated increase of the plasma TG during the first 4 hours after the meal was not different between the rapeseed and the olive oil diets ( $113.5 \pm 24.5$  mg/dl\*h vs.  $115.0 \pm 20.9$  mg/dl\*h), while it was only  $2.0 \pm 10.9$  mg/dl\*h with hydrogenated oil, again related to its low absorption. About 5 hours after the meal intake values had returned close to baseline values.

While the maximal increase in glucose levels after the meal was lower with the hydrogenated fat, the area under the concentration curve for glucose from the meal until 4 hours afterwards was very similar between all diets (olive oil:  $16.4 \pm 8.5$  mg/dl\*h, hydrogenated fat:  $17.0 \pm 9.8$  mg/dl\*h, rapeseed oil:  $17.5 \pm 8.6$  mg/dl\*h).

The exhalation of labelled  $^{13}\text{C}$  linoleic acid integrated into the test meal showed similar curves between rapeseed and olive oil, with a tendency towards higher recoveries

at all time points with rapeseed oil (Fig. 4). After hydrogenated fat higher recoveries were seen in the initial phase, while these tended to be lower towards the end of the observation period. Integrated recovery after 8 hours was  $10.67 \pm 0.58$  % of dose after the olive oil meal,  $11.56 \pm 0.60$  % after the hydrogenated oil and  $13.12 \pm 0.82$  % after the rapeseed meal ( $p < 0.005$  vs. olive and  $p < 0.04$  vs. hydrogenated). The higher recovery after rapeseed oil intake was due to higher  $^{13}\text{C}$  enrichment in breath  $\text{CO}_2$  and not due to higher  $\text{CO}_2$  production, which tended to be even slightly lower after the rapeseed oil meal ( $185 \pm 7$  ml/min) compared to olive oil meal ( $189 \pm 6$  ml/min). From the indirect calorimetry measurements respiratory quotients were calculated, which were significantly different ( $p = 0.028$  repeated measures ANOVA, RQ after olive oil:  $0.83 \pm 0.02$ , RQ after hydrogenated oil:  $0.87 \pm 0.01$ , RQ after rapeseed oil:  $0.82 \pm 0.02$ ). Paired t-tests compared to hydrogenated fat yielded p-values of 0.051 for olive oil and 0.015 for rapeseed oil, whereas there was no difference between olive and rapeseed oils ( $p = 0.725$ ).

The areas under the curve for plasma TG and breath  $^{13}\text{CO}_2$  recovery did not show a significant correlation during any of the test diets.

## Discussion

This study demonstrates similar effects of olive oil and rapeseed oil on postprandial lipid metabolism. Both olive oil and rapeseed oil have been included in various studies on lipid metabolism indicating their beneficial effects on cholesterol and LDL cholesterol concentrations. This effect was not obvious in our study, presumably due to the short study duration.

A comparison with the hydrogenated fat is invalidated by its lower absorption, which was clearly demonstrated by the higher stool fat content, even though this does not allow quantification of the absorption rate. The one week diet periods are considered to serve as a priming of gastrointestinal and body pools with the fatty acids dominating the corresponding test oil. This was achieved, as observed from the fatty acid percentages in the plasma phospholipids analysed from the fasting sample on the seventh day on the corresponding diet which were clearly reflecting the different diets. Oleic acid was significantly higher after olive oil than after the other dietary interventions, while  $\alpha$ -linolenic acid was increased after rapeseed oil intake. Despite the incomplete absorption, palmitic acid, which contributed more than 80 % to the hydrogenated fat was increased after its intake. Although linoleic acid is clearly higher in rapeseed than in olive oil, the difference was not large enough to obtain significantly higher plasma values. The long chain polyunsaturated derivatives of linoleic and  $\alpha$ -linolenic acid, which influence eicosanoid metabolism, have not changed significantly with the one week dietary intervention, although there was a tendency towards higher eicosapentaenoic acid with the higher  $\alpha$ -linolenic acid intake from rapeseed oil. This agrees with the study of Finnegan et al., who compared the effects of  $\alpha$ -linolenic acid and fish oil intake in a longer term study and found clear changes of the long-chain derivatives eicosapentaenoic and docosahexaenoic acids with fish oil but not with  $\alpha$ -linolenic acid, indicating a limited  $\alpha$ -linolenic acid conversion.

The dietary intake of the subjects studied were similar to usual dietary habits in the adult German population, and there were no differences during the 3 test weeks.

Subjects complaint during the hydrogenated oil period on some abdominal discomfort, but none of the participants considered termination of the study.

The postprandial plasma lipids showed the time course expected from the literature, taking into account incomplete absorption of the hydrogenated fat. There were also very similar curves for postprandial glucose and insulin after the plant oil meals, but a much different curve was observed for the hydrogenated fat, with a lower postprandial maximal increase of glucose and a delayed decrease. This is most readily explained by a delayed absorption of glucose, since the ingredients of the test meal were baked into a muffin.

Given that differences in metabolic effects between saturated and unsaturated dietary fat are largely known and considering the low absorption of the hydrogenated fat, the comparison between olive and rapeseed oils is of greatest interest in this study. Fasted and postprandial lipid concentrations were not significantly different between these two oils. The significantly higher oxidation of  $^{13}\text{C}$  labelled linoleic acid with the rapeseed oil diet is of interest. The observed tracer recoveries are comparable to values reported by Delany and Windhauser, who found  $16.1 \pm 6.6$  % of dose recovery over 9 hours for carboxyl or methyl  $^{13}\text{C}$ -labelled linoleic acid <sup>14</sup>. The slightly lower recoveries in our study can be explained by the shorter duration of sampling. Also, energy intake may have been different because it has not been reported by Delany and Windhauser<sup>14</sup>. In an other study  $14.0 \pm 4.1$  % ( $M \pm SD$ ) of the dose were recovered in healthy men after application of 1- $^{13}\text{C}$ linoleic acid incorporated into TG during 8 hours <sup>15</sup>. The tendency towards lower values in our study might be due the provision of a meal with carbohydrates, whereas Bretillon et al supplied only lipids (tracer plus olive oil) <sup>15</sup>.

The similar postprandial cholesterolemia and triglyceridemia leads us to assume that olive and rapeseed oil did not differ markedly in absorption and in effects on lipoprotein metabolism. The similar postprandial TG concentrations indicate a similar availability of the tracer linoleic acid for intracellular disposal. Intracellular metabolism of fatty acids is also influenced by glucose and insulin levels, but there were no significant differences in their plasma concentrations between the two test meals. Although fasted phospholipid percentages for linoleic acid were not different between the diets it would be conceivable that the higher linoleic acid content in rapeseed oil might have caused significantly greater tracer dilution, but this would be expected to decrease rather than increase the portion of tracer oxidised and recovered in the breath, because the tracer amount was identical in all tests. Furthermore, respiratory quotients did not significantly differ between the olive oil and the rapeseed oil meals, which indicates that the relative proportions of lipids and glucose oxidised did not differ between the meals.

Fatty acid metabolism can be influenced by fatty acids via nuclear receptors, mainly peroxisome proliferator activated receptors (PPAR), but also a variety of other transcription factors<sup>16;17</sup>. Carnitine palmitoyltransferase catalysing the entry of long chain fatty acids into the mitochondria is considered the rate limiting step in mitochondrial fatty acid  $\beta$ -oxidation, and its mRNA expression is influenced by PPAR $\alpha$ <sup>18</sup>. In cultured rat hepatocytes, the addition of oleic, linoleic and  $\alpha$ -linolenic acid shows only minor differences with respect to the PPAR $\alpha$  mediated mRNA expression of AcylCoA oxidase (peroxisomal fatty acid oxidation), while eicosapentaenoic acid induces an about twofold increase of the enzyme<sup>19</sup>. Thus it is tempting to speculate that the non significant increase in eicosapentaenoic acid during the rapeseed oil diet might have led to an increased fatty acid oxidation via PPAR $\alpha$  activation, which was reflected in the increased tracer oxidation, while hardly decreasing the respiratory quotient.

In conclusion, the results obtained indicate a very similar beneficial influence of rapeseed and olive oils on postprandial lipid metabolism. However, rapeseed oil with its higher  $\alpha$ -linolenic acid content may have additional beneficial effects on enhancing fat oxidation.

**Acknowledgement**

The authors gratefully acknowledge partial financial support of this study by Union zur Förderung von Oel- und Proteinpflanzen e. V, Berlin, Germany.

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Table 1: Fatty acid composition (%-wt/wt) of the tested oils (own analyses)

fatty acid	olive oil	rapeseed oil	hydrogenated plant oil
C14:0	-	0.1	1.4
C16:0	11.0	4.9	81.1
C16:1	0.8	0.2	0.1
C18:0	3.5	1.8	5.0
C18:1	78.2	61.4	9.7
C18:2n-6	5.1	20.0	2.2
C18:3n-3	0.6	8.4	-
C20:0	0.4	0.6	0.3
C20:1	0.2	1.3	-

Table 2: Dietary intake of energy, macronutrients and major fatty acid classes during the corresponding periods (M $\pm$ SEM)

	olive oil	rapeseed oil	hydrogenated plant oil
energy	2016 $\pm$ 120	2031 $\pm$ 116	1973 $\pm$ 147
fat	25.4 $\pm$ 1.1	32.6 $\pm$ 5.9	30.6 $\pm$ 2.8
carbohydrates	60.5 $\pm$ 1.1	55.9 $\pm$ 3.0	58.4 $\pm$ 3.5
protein	12.3 $\pm$ 0.8	12.5 $\pm$ 1.0	14.7 $\pm$ 1.8
saturated fat*	5.0 $\pm$ 0.4	5.0 $\pm$ 0.8	21.7 $\pm$ 2.3
monounsaturated fat*	15.0 $\pm$ 0.6	12.4 $\pm$ 0.8	5.0 $\pm$ 0.6
polyunsaturated fat*	3.4 $\pm$ 0.2	8.0 $\pm$ 0.4	2.7 $\pm$ 0.4
ALA*	0.35 $\pm$ 0.03	1.81 $\pm$ 0.14	0.33 $\pm$ 0.08

\* significantly different from the other dietary interventions

Table 3: Fasted plasma values (M±SEM) of phospholipid fatty acids (%-wt/wt), lipids and lipoproteins (mg/dl), non esterified fatty acids (mmol/l,) glucose (mg/dl) and insulin (U/l) of the subjects after 6 days on the three diet periods (n=20)

	Olive oil	Rapeseed oil	Hydrogenated fat
C16:0	27.8±0.3	27.6±0.4	30.3±0.3*
C18:0	12.1±1.0	11.1±0.2	10.9±0.2
C18:1	12.3±0.3*	10.5±0.2	9.8±0.3
C18:2n-6	17.3±0.5	18.4±0.4	17.9±0.6
C18:3n-3	0.14±0.02	0.32±0.03*	0.15±0.01
C20:4n-6	10.2 ±0.4	10.4 ±0.4	9.8±0.3
C20:5n-3	0.8±0.1	0.9±0.1	0.8±0.1
C22:6n-3	3.7±0.2	3.9±0.2	3.9±0.2
TG	87±7	90±9	97±14
CHOL	161±4	158±5	163±5
LDL	94±4	92±4	96±4
HDL	54±3	53±3	53±2
NEFA	0.69±0.07	0.68±0.07	0.64±0.06
Glucose	95±2	97±2	92±2
Insulin	6.3±0.8	7.8±1.0	7.4±0.8

\* significantly different from the other dietary interventions

Table 4: Safety parameters (M $\pm$ SEM) after 6 days on the three test diets, no significant differences

	olive oil	rapeseed oil	hydrogenated plant oil
total bilirubin	0.59 $\pm$ 0.09	0.64 $\pm$ 0.09	0.59 $\pm$ 0.11
GOT	24.2 $\pm$ 1.6	23.2 $\pm$ 1.2	24.8 $\pm$ 1.4
GPT	17.0 $\pm$ 1.8	16.4 $\pm$ 1.7	17.9 $\pm$ 2.0
GGT	18.2 $\pm$ 1.5	18.8 $\pm$ 1.7	19.3 $\pm$ 1.7
AP	62.4 $\pm$ 3.4	62.5 $\pm$ 3.3	64.4 $\pm$ 3.0

Figure 1: Postprandial change ( $M \pm SEM$ ) of plasma TG and nonesterified fatty acids (NEFA) after intake of a test meal containing either olive, rapeseed or hydrogenated oil (\*significantly different from basal value)

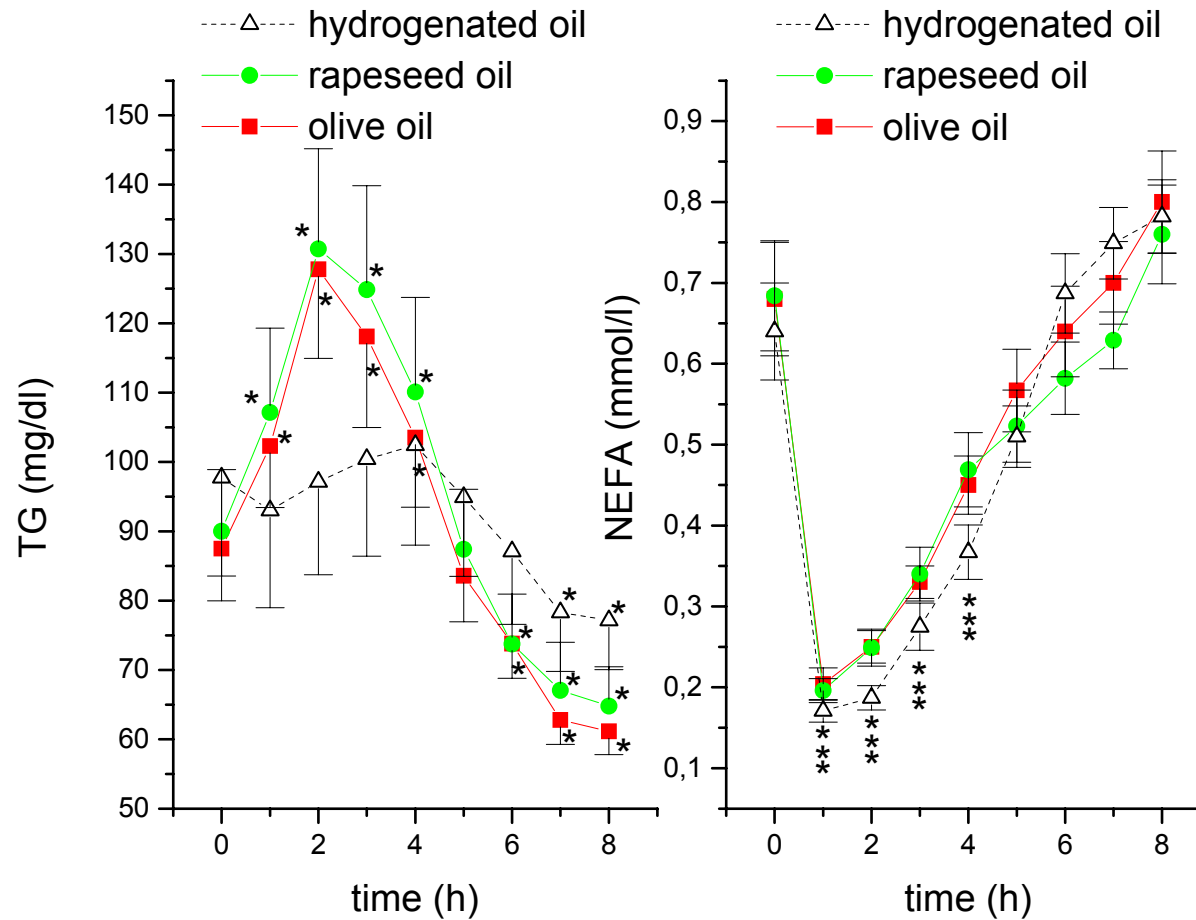


Figure 2: Postprandial change of VLDL-cholesterol, LDL-cholesterol, HDL-cholesterol and total cholesterol after intake of a test meal containing either olive, rapeseed or hydrogenated oil (S $\pm$ SEM)

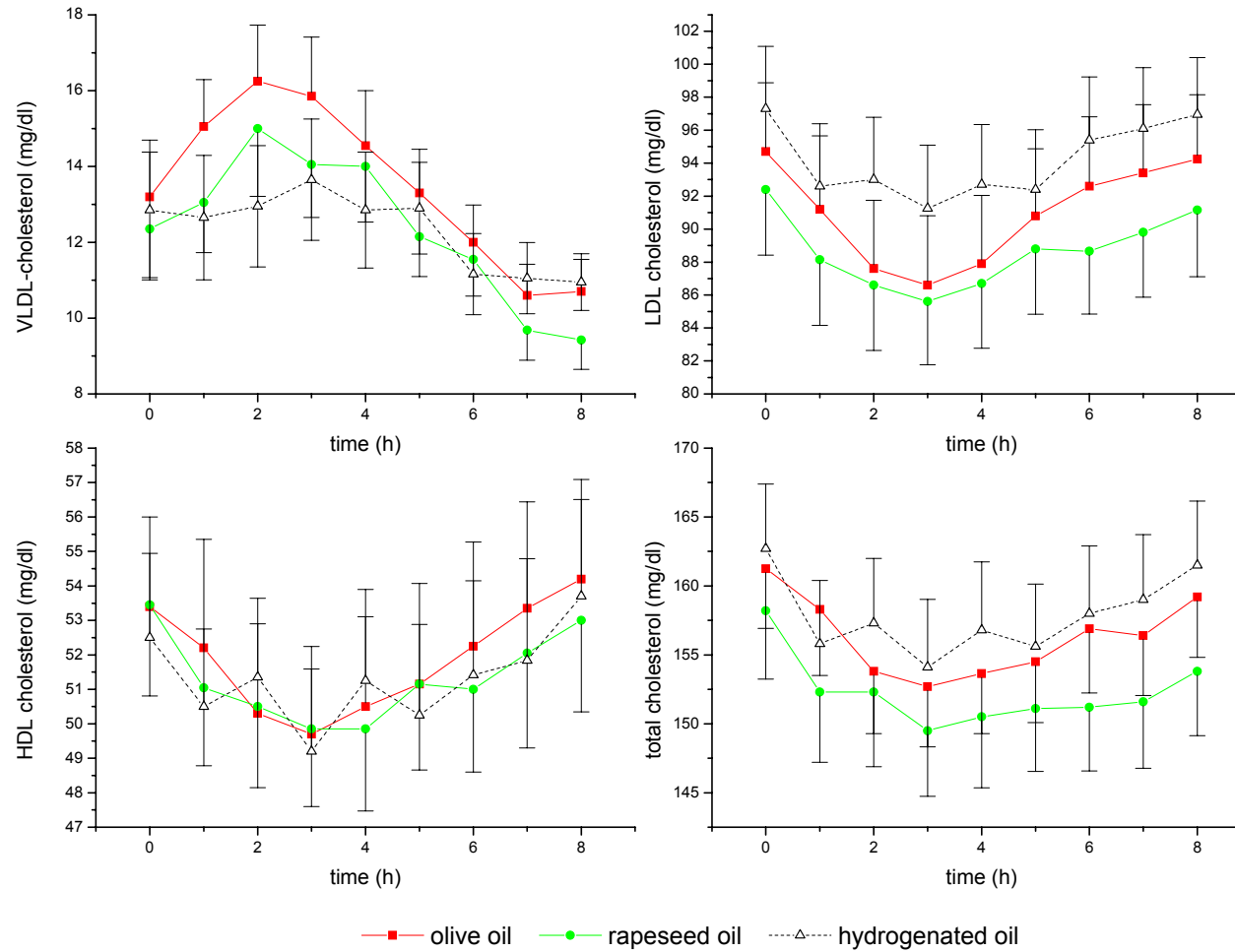


Figure 3: Postprandial change ( $S \pm SEM$ ) of plasma insulin and glucose after intake of a test meal containing either olive oil or rapeseed oil or hydrogenated fat (\*significantly different from basal value)

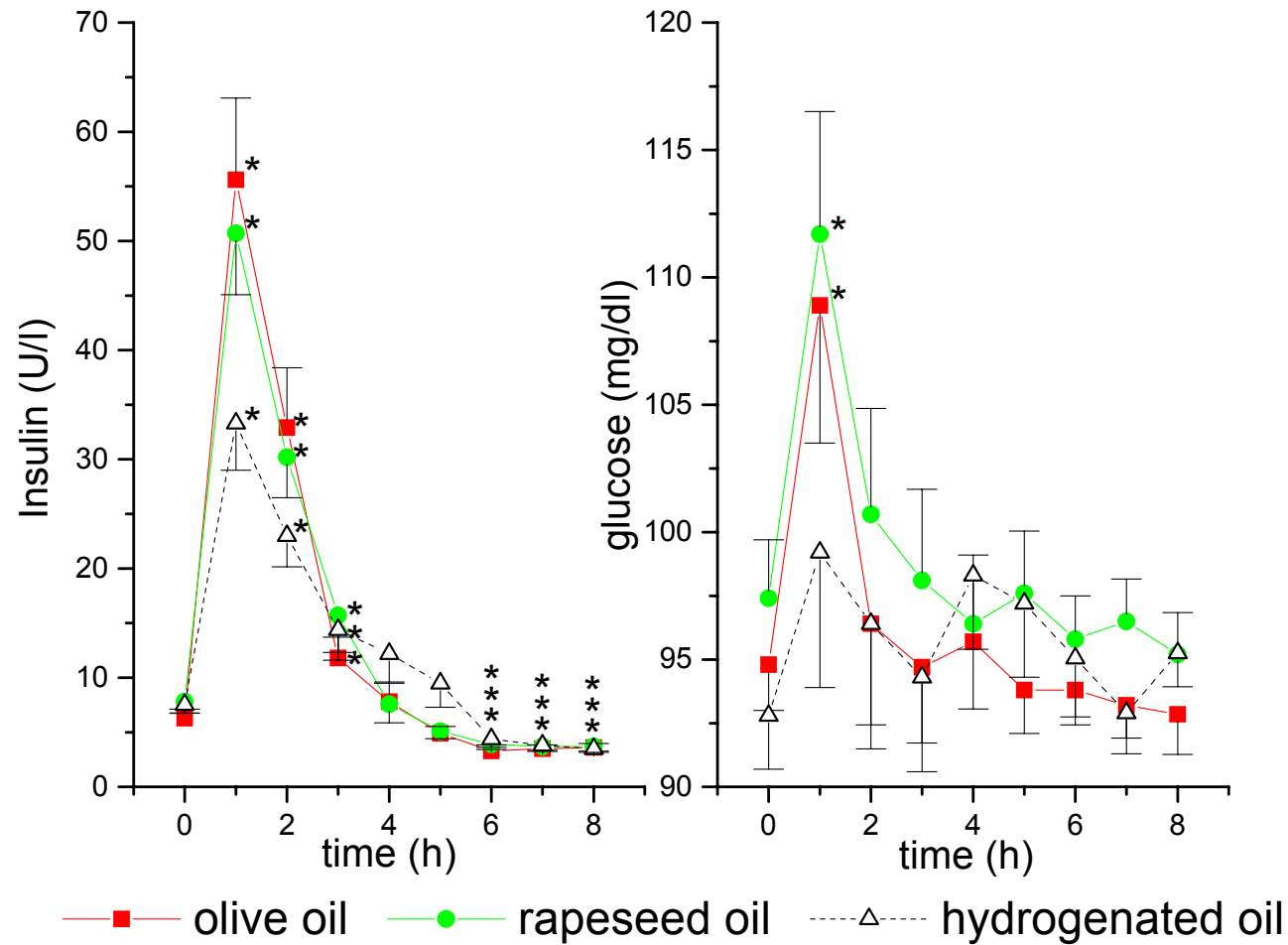




Figure 4: Appearance of  $^{13}\text{CO}_2$  in breath after oral intake of  $^{13}\text{C}$  labelled linoleic acid together with test meals containing either rapeseed, olive or hydrogenated oil ( $M \pm \text{SEM}$ , \* significantly different according to repeated measures ANOVA)

