# Incremental Area Under Response Curve More Accurately Describes the Triglyceride Response to an Oral Fat Load in Both Healthy and Type 2 Diabetic Subjects

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**Elevation of postprandial triacylglycerol (TG)-rich plasma lipoproteins is considered potentially atherogenic. Type 2 diabetic patients have exaggerated postprandial TG compared with healthy subjects. Postprandial TG responses to oral fat loads are usually studied as the area under the TG curve. No consensus exists regarding the method of choice when calculating the TG response area. We evaluated the correlation between fasting TG and postprandial TG responses calculated by the trapezoid rule as total area under the curve (AUC) and incremental area under the curve (iAUC). Furthermore, we compared the AUC and iAUC to a 3-point calculation method. Ten healthy subjects and 47 type 2 diabetic patients ingested test meals consisting of an energy-free soup plus 80 g fat and 50 g carbohydrate. TG responses were measured in total plasma, in a chylomicron (CM)-rich fraction and in a CM-poor fraction. In healthy subjects the AUC, but not iAUC, correlated positively to fasting TG. In type 2 diabetic patients a strong correlation was found between fasting TG and AUC, whereas weak associations were found to the iAUCs. The iAUC was strongly correlated to the postprandial TG rise in both groups. The 3-point areas differed significantly from the trapezoid measurements in both healthy and type 2 diabetic subjects. In conclusion, in both healthy and type 2 diabetic subjects total AUC is highly correlated to fasting TG, whereas iAUC more accurately describes the TG response to an oral fat load. The 3-point test seems less suitable for the determination of postprandial response in both healthy and type 2 diabetic subjects.**



THE TYPICAL western diet is fat rich, ie, dietary fat constitutes greater than 30% of total energy. The fat-rich meals cause postprandial lipemia 18 hours per day, and this response is related to atherosclerosis[1-3] and cardiovascular disease.[4,5] Most studies of postprandial triacylglycerol (TG) in humans cover at least 8 hours. No consensus exists about the method to use to calculate postprandial TG responses and different methods are often used in various studies,[6-8] eg, calculations are done both as the area under the curve (AUC) and the incremental area under the curve (iAUC).[9] The fasting TG concentrations and the total TG-AUC have been shown to correlate strongly.[8,10] Furthermore, the postprandial peak TG after an oral fat load seems to reflect the fasting TG, whereas TG concentrations at late time points may better discriminate for the presence of cardiovascular disease.[11] There is, however, some discrepancy regarding the most descriptive time points of the postprandial TG.[12,13] Special interest has been directed to the postprandial chylomicrons (CMs). Thus, accumulating evidence suggests a causal role in the development of atherosclerosis[14-16] and coronary artery disease (CAD).[17]

The TG responses of type 2 diabetic patients are exaggerated as compared with healthy subjects[8] and generally cosegregate with the dyslipidemia of insulin resistance/type 2 diabetes.[18] Guerci et al[19] found good correspondence between calculations of postprandial TG responses to an oral fat load by a simplified 3-point test, using blood samples from 3 time points only, and the conventional trapezoid rule, using more frequent blood sampling, in healthy and type 2 diabetic subjects.

The study of postprandial lipemia is a growing field; however, no systematic evaluation of the method of choice to calculate the TG responses has been performed. The 3-point test may reduce the workload in postprandial studies. So far, however, the method has been tested only in 10 type 2 diabetic patients[19] and needs further evaluation.

In the present study we wanted to investigate the correlation between the fasting TG and postprandial TG responses to an oral fat load, calculated as total AUC and iAUC, in both healthy and type 2 diabetic subjects. In addition, we wanted to evaluate the 3-point test in the same subjects.

## MATERIALS AND METHODS

Forty-seven type 2 diabetic patients, recruited from our outpatient clinic,[20,21] and 10 healthy medical students[22] participated in studies concerning postprandial lipemia.

The type 2 diabetic patients had an average (mean ± SD) age of 65 ± 5 years, were overweight with a mean body mass index (BMI) of 28.2 ± 5.7 kg/m$^2$, and had a mean hemoglobin $A_{1c}$ ($HbA_{1c}$) of 7.2% ± 0.9 % (normal range, 3.5% to 5.5%). Fasting TG was 1.5 ± 0.7 mmol/L (range, 0.4 to 4.2 mmol/L). All patients been diagnosed with diabetes for at least 1 year. None was treated with insulin or lipid-lowering drugs.

The average age of the healthy subjects was 23 ± 2 years; they were lean with a mean BMI of 21.7 ± 1.8 kg/m$^2$, and had a mean fasting blood glucose concentration of 4.5 ± 0.5 mmol/L. Fasting TG was 1.0 ± 0.3 mmol/L (range, 0.6 to 1.6 mmol/L). None was treated with medicine.

The studies were approved by the local ethical committee of Aarhus County.

All studies were performed according to our standardized experimental set-up as described previously.[22] The participants ingested high-

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**Table 1. TG Results From Healthy Subjects (20 test meals) and Type 2 Diabetic Patients (82 test meals)**

| | Mean ± SD | |
|---|---|---|
| | Healthy Subjects | Type 2 Diabetic Subjects |
| Plasma TG (mmol/L) | | |
| Fasting | 1.00 ± 0.30 | 1.50 ± 0.73 |
| Max. | 1.89 ± 0.64 | 2.34 ± 1.57 |
| CM-rich TG (mmol/L) | | |
| Fasting | 0.14 ± 0.16 | 0.36 ± 0.34 |
| Max. | 0.44 ± 0.29 | 1.63 ± 1.13 |
| CM-poor TG (mmol/L) | | |
| Fasting | 0.76 ± 0.21 | 1.00 ± 0.52 |
| Max. | 0.90 ± 0.28 | 1.20 ± 0.53 |

NOTE. Max. values are postprandial.

carbohydrate food delivered by a dietitian during the 24 hours before the oral fat tests. The amounts of food corresponded to the individual energy requirements estimated from the Harris-Benedict equation with adjustments for physical activity.[23] Physical activity was standardized and minimized on the study mornings, and participants were transported to the experimental setting by bus and immediately seated at arrival at 7 AM. Subsequently, a catheter was placed in an antecubital vein and the participant rested for 30 minutes. Basal blood samples were drawn and at time 0, and the test meal was ingested within 10 minutes. During the following 8 hours, blood samples were drawn 1 or 2 hours apart. Plasma was immediately separated by centrifugation and kept frozen at −20°C.

The healthy subjects[22] and one group of type 2 diabetic patients[20] were tested on 2 or 4 separate occasions at least 1 week apart. The remaining type 2 diabetic patients were tested only once.[21] Altogether, we attained 20 oral fat loading tests from the healthy subjects and 82 test meals from the type 2 diabetic patients.

The test meal consisted of a calorie-free soup, which was enriched by 80 g of fat and taken with 50 g carbohydrate as white bread. Furthermore, thin slices of raw leek were added the test meal to mask the taste, and it was served with 250-mL tap water. The energy content was 4,200 kJ.

For separation of lipoproteins, the plasma samples were subjected to a single ultracentrifugation step, dividing the sample into a CM-rich and a CM-poor fraction. Four milliliters of plasma was overlayered with 2 mL of a solution with a density of 1.006 g/mL in a Quick-seal tube (No. 344619 Beckman Coulter, Fullerton, CA) and centrifuged in a Sw 50.3 Ti fixed-angle rotor (Beckman Instruments, Palo Alto, CA) for 30 minutes at 26,000 × *g*. Tubes were sliced in a Beckman slicer 4 mL from the bottom and the CM-rich supernatants (Svedberg flotation units [Sf] value > 1,000) were removed and brought to a final volume of 4 mL with saline. The infranatant, ie, the CM-poor fraction, contained the plasma proteins and remaining lipoproteins, and thus the TG concentration may be allocated to the CMs in the CM-rich fraction and to the very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), and CM remnants, respectively, in the CM-poor fraction. TG was determined in plasma and in both fractions by standard enzymatic colorimetric assays using commercial kits (Waco Chemicals, Neuss, Germany, and Boehringer Mannheim, Mannheim, Germany).

The results are expressed as mean ± SD from individually analyzed results from all participants (Table 1). The TG response data are given as the AUC and as the iAUC (area above baseline), calculated by the conventional trapezoid rule from data during 8 hours.[9] Furthermore, the data were analyzed by a 3-point rule as described by Guerci et al.[19] This method assumes that response areas can be calculated by data from 3 time points: t = 0 h, t = 4 h, and t = 8 h. TG responses were analyzed by linear regression analysis with response areas as dependent and fasting plasma TG as independent variables (Table 2). Furthermore, TG responses were analyzed with response areas as dependent and TG rises from baseline as independent variables. The correlation is described as the squared correlation coefficient ($r^2$).[24] The $r^2$ gives the fraction of the total variation, which is determined by the actual dependent variable (SigmaStat for Windows Version 2.03, SPSS Inc, Chicago, IL). Corresponding areas from the trapezoid and 3-point rules, respectively, were compared by paired Student's *t* test or Wilcoxon's signed-rank test, and correlation was tested by Pearson product moment correlation (Tables 3 and 4). *P* values less than .05 were considered statistically significant.

## RESULTS

Basic plasma TG results from healthy subjects (20 test meals) and type 2 diabetic patients (82 test meals) are shown in Table 1.

### *Healthy Subjects*

TG-AUC was positively correlated to fasting TG in total plasma and the CM-poor fraction, by both calculation methods (Table 2), explaining between 67% and 83 % of the total variation. The fasting TG (Table 2) only very modestly influenced the AUC response in the CM-rich fractions. The iAUC did not correlate with fasting TG by either calculation method (Table 2). The iAUC was strongly correlated to the rise in TG ($r^2 = 0.87$, $P < .001$), ie, 87 % of the total variation was explained by the rise in TG. AUC had a weaker correlation ($r^2 = 0.47$, $P < .001$).

Comparisons of the traditional trapezoid rule and the 3-point rule showed a high correlation between the 2 methods in total plasma and the 2 fractions (Table 3). However, the results were

**Table 2. Linear Regression Results From Healthy Subjects (20 test meals) and Type 2 Diabetic Patients (82 test meals)**

| | Plasma TG | | CM-rich TG | | CM-poor TG | |
|---|---|---|---|---|---|---|
| | Total AUC | iAUC | Total AUC | iAUC | Total AUC | iAUC |
| Healthy subjects | | | | | | |
| Trapezoid rule | 0.70 (<.001) | 0.09 (.24) | 0.02 (.57) | 0.003 (.81) | 0.76 (<.001) | 0.04 (.43) |
| 3-point rule | 0.67 (<.001) | 0.06 (.31) | 0.26 (.02) | 0.14 (.11) | 0.83 (<.001) | 0.005 (.76) |
| Type 2 diabetic subjects | | | | | | |
| Trapezoid rule | 0.78 (<.001) | 0.20 (<.001) | 0.56 (<.001) | 0.18 (<.001) | 0.89 (<.001) | 0.07 (.02) |
| 3-point rule | 0.72 (<.001) | 0.16 (<.001) | 0.55 (<.001) | 0.18 (<.001) | 0.85 (<.001) | 0.05 (.04) |

NOTE. In the model, individual total AUC and iAUC are the dependent variables. The corresponding fasting TG values are the independent variables. Results are given as coefficient of determination ($r^2$) and corresponding *P* value in parenthesis.

**Table 3. TG Response Areas in Plasma, and in the CM-Rich and CM-Poor Fractions to Oral Fat Loads in Healthy Subjects (20 test meals)**

| | Mean ± SD | | *P* Value (paired comparison) | Pearson's Correlation Coefficient |
|---|---|---|---|---|
| | Trapezoid Rule | 3-Point Rule | | |
| Plasma TG (mmol/L · 480 min) | | | | |
| Total AUC | 647.0 ± 218.6 | 616.1 ± 212.7 | .07 | 0.95 ($P < .001$) |
| iAUC | 171.1 ± 121.5 | 142.1 ± 119.9 | .07 | 0.84 ($P < .001$) |
| CM-rich TG (mmol/L · 480 min) | | | | |
| Total AUC | 230.8 ± 125.4 | 110.9 ± 64.9 | <.001 | 0.40 ($P = .08$) |
| iAUC | 61.7 ± 51.7 | 118.8 ± 70.4 | <.001 | 0.88 ($P < .001$) |
| CM-poor TG (mmol/L · 480 min) | | | | |
| Total AUC | 363.7 ± 122.9 | 349.3 ± 109.7 | .03 | 0.97 ($P < .001$) |
| iAUC | 18.1 ± 47.7 | 9.9 ± 26.9 | .01 | 0.76 ($P < .001$) |

NOTE. Corresponding areas from the trapezoid and 3-point rules, respectively, compared by paired Student's *t* test or Wilcoxon's signed-rank test, and correlation tested by Pearson product moment correlation.

statistically different, both calculated as AUC and as iAUC (Table 2).

*Type 2 Diabetic Subjects*

TG-AUC was positively correlated to fasting TG by both calculation methods, explaining between 55% and 89 % of the total variation (Table 2). The iAUC correlated statistically significantly to fasting TG by both calculation methods (Table 2); however, it only explained between 5% and 20 % of the variation. The iAUC was strongly correlated to the rise in TG ($r^2 = 0.90$, $P < .001$), whereas AUC had a weaker correlation ($r^2 = 0.64$, $P < .001$).

Comparisons of the traditional trapezoid rule and the 3-point rule showed a high correlation between the 2 methods in total plasma and the 2 fractions (Table 4). The results from total plasma and the CM-rich fractions were statistically different, both calculated as AUC and iAUC (Table 2).

## DISCUSSION

Our results strongly suggest that postprandial TG responses to an oral fat load should be calculated as the iAUC rather than the AUC in both healthy subjects and type 2 diabetic patients. Furthermore, the results of a simplified 3-point test were not equal to a more frequently sampled test in either healthy subjects or type 2 diabetic patients.

Our experiments were performed in a highly controlled setting, which minimized the variation of fasting TG,[20-22] and the participants were given the test meals with similar compositions.

Several investigators have demonstrated an association between fasting TG and postprandial TG rise in nondiabetic subjects,[25-27] which may be exaggerated by obesity.[28] In the present study, we found that fasting TG in lean healthy subjects is strongly associated to total AUC but not to iAUC of TG (Table 2). Furthermore, the iAUC was strongly associated with the rise in postprandial TG. Thus, in healthy subjects, the iAUC describes better the postprandial TG response than the total AUC, which seems to be a descriptive factor, related to basal TG.

Lewis et al[8] found in type 2 diabetic patients that fasting TG and TG-iAUC had a strong correlation ($r^2 = 0.77$, $P = .0001$), which was more pronounced in patients with "high fasting TG" (1.7 to 4.7 mmol/L). We found much weaker associations ($r^2 =$ 0.07 to 0.20) between fasting TG and iAUCs in total plasma and the CM-rich and CM-poor fractions, respectively (Table 2). The fasting TG in the study by Lewis et al and in our type 2 diabetic patients was apparently quite similar. Concomitant intake of fat and carbohydrate may exaggerate postprandial lipid responses.[29] Furthermore, the addition of fructose to even a small fat load accentuated the postprandial TG responses in total plasma, a CM-rich, and a CM-poor fraction.[30] The test meals used in our studies and by Lewis et al did not differ

**Table 4. TG Response Areas in Plasma, and in the CM-Rich and the CM-Poor Fractions to Oral Fat Loads in Type 2 Diabetic Patients (82 test meals)**

| | Mean ± SD | | | | *P* Value (paired comparison) | Pearson's Correlation Coefficient |
|---|---|---|---|---|---|---|
| | Trapezoid Rule | | 3-Point Rule | | | |
| Plasma TG (mmol/L · 480 min) | | | | | | |
| Total AUC | 873.7 | 527.8 | 852.3 | 544.4 | <.01 | 0.98 ($P < .001$) |
| iAUC | 169.4 | 262.5 | 154.8 | 293.8 | .03 | 0.93 ($P < .001$ |
| CM-rich TG (mmol/L · 480 min) | | | | | | |
| Total AUC | 440.9 | 317.5 | 413.7 | 319.4 | <.01 | 0.96 ($P = .08$) |
| IAUC | 274.5 | 212.3 | 247.9 | 216.1 | <.01 | 0.91 ($P < .001$) |
| CM-poor TG (mmol/L · 480 min) | | | | | | |
| Total AUC | 479.5 | 235.7 | 474.7 | 231.6 | .95 | 0.98 ($P < .001$) |
| iAUC | 27.5 | 40.3 | 28.4 | 39.8 | .44 | 0.87 ($P < .001$) |

NOTE. Corresponding areas from the trapezoid and 3-point rules, respectively, compared by paired Student's *t* test or Wilcoxon's signed-rank test, and correlation tested by Pearson product moment correlation.

significantly in the amount of carbohydrate, although in the latter study, fruit was used as a carbohydrate source.[8] In the present study, we found that in type 2 diabetic patients total AUC is strongly associated to fasting TG, whereas iAUC has a much weaker association (Table 2). Furthermore, the iAUC was strongly associated with the rise in postprandial TG. Thus, as in healthy subjects, the iAUC seems to describe the TG response to foods better than does the total AUC.

The study of postprandial lipemia is very time-consuming and labor-intensive. Guerci et al therefore tested a protocol[19] using blood samples from only 3 time points during 8 postprandial hours. They found good associations between 3-point data and results from the AUCs calculated from TGs taken at several time points. However, the study was performed on only a few postprandial fat tests,[19] which enhances the risk of a statistical type 2 error. We found a high degree of correlation between the 3-point and usual trapezoid calculations (Tables 3 and 4). However, the differences were statistically different in total plasma and the 2 fractions in healthy subjects (Table 3), as were the differences in total plasma and the CM-rich fraction in the type 2 diabetic patients (Table 4). Thus, the results from the 3-point rule and the more frequently sampled trapezoid rule were not similar in healthy subjects or in type 2 diabetic patients.

In conclusion, in both healthy and type 2 diabetic subjects, total AUC is highly correlated to fasting TG, whereas iAUC more accurately describes the TG response to an oral fat load. The 3-point test seems less suitable for the determination of postprandial response in both healthy and type 2 diabetic subjects. Further studies are needed to prove the inappropriateness of the 3-point method.

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