Dietary fat and carbohydrate quality have independent effects on postprandial glucose and lipid responses

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Running title: Glycemic index, fat saturation, and postprandial metabolism

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Abstract

Purpose The magnitude of postprandial lipemia is influenced not only by the amount but also the type of fat and carbohydrate consumed. The aim of this study was to evaluate differences in postprandial glucose and lipids responses after a mixed meal containing low or high glycemic index (GI) carbohydrate and three different types of fat varying in the degree of saturation in healthy subjects.

Methods A randomized, controlled, single-blinded crossover study was conducted in 20 healthy Chinese men. Subjects consumed in random order 6 experimental isocaloric meals that differed in carbohydrate and fat quality, and contained 40 g of either saturated fat (SFA, butter), monounsaturated fat (MUFA, olive oil) or polyunsaturated fat (PUFA, grape seed oil), and 50 g of either low GI (basmati rice) or high GI (jasmine rice) carbohydrate. Glucose, insulin, c-peptide, triglycerides (TG) and non-esterified fatty acids (NEFA) were measured over 4 h.

Results For all substrates evaluated, there were no significant interactions between fat and carbohydrate. The incremental area under the curve (iAUC) for TG was significantly lower after the SFA and PUFA meals compared with the MUFA meal, irrespective of GI. No significant difference was found for NEFA iAUC in all treatments. Glucose, insulin and c-peptide iAUCs were significantly lower after ingestion of low GI than high GI meals, independent of the type of fat.

Conclusions A carbohydrate-rich meal (of either low or high GI) that contains butter or grapeseed oil results in lower postprandial TG concentrations relative to olive oil in healthy Chinese males. Glucose, insulin, and c-peptide responses, however, are directly dependent on the GI of the meal and not on the degree of saturation of dietary fat.

The trial was registered at clinicaltrials.gov as NCT02585427.

Key words: dietary fat, carbohydrate, postprandial response, mixed meal, rice
Introduction

Asia has gained the unenviable reputation as being the epicenter of Type 2 diabetes (T2D) [1, 2]. Diet, in part, has been considered a major contributor in the etiology of diabetes. Most Asian diets are characterized by the consumption of white rice, a rich source of carbohydrate of high glycemic index (GI). The GI is a measure of how rapidly blood glucose is elevated when a given amount of carbohydrate is consumed [3]. High GI diets have been implicated in the increased risk of T2D and cardiovascular disease [4-7]. Although Asian diets are predominantly rice-based, rice is usually accompanied by a variety of different types of fat, vegetables, and protein. Asian diets are rich in carbohydrate (up to 70% of energy), relatively low in fat and protein, and also low in omega-3 fatty acids, which have been implicated in hypertriglyceridemia.

The increased focus on carbohydrate metabolism and the identification of ways to improve glycemic control in Asians has neglected the potential impact of carbohydrate quality (low or high GI) on lipid metabolism. Triglyceride (TG) concentrations in the postprandial period, i.e. after consuming a meal, are emerging as a significant independent risk factor for atherosclerosis, and may be a stronger predictor of cardiovascular disease and a contributor to insulin resistance compared with fasting TG levels [8-13]. This is not entirely surprising, given that, in contemporary settings, humans spend the majority of their time in the postprandial rather than the fasting state. Although the quantitative effects of dietary fat and carbohydrate on postprandial TG metabolism have been extensively studied, comparatively less is known about the importance of fat and carbohydrate quality. Most [14-16] but not all [17] studies found that dietary saturated fatty acids (SFA) result in lower postprandial lipemia (i.e. lower postprandial TG concentrations) than monounsaturated (MUFA) or polyunsaturated (PUFA) fatty acids. These published studies used saturated fat in the form of butter [14, 17], stearic acid [15], and milk fat [16]. Also, early studies found that ingestion of meals containing glucose and fat resulted in lower serum TG concentrations compared to ingestion of sucrose or fructose and fat [18-20], suggesting that the type of carbohydrate consumed is also important in determining postprandial TG responses. Subsequent studies revealed that both the amount [21, 22] and the type of
fat [14, 17, 23] and carbohydrate [18, 24, 25] can influence postprandial TG metabolism. The GI of dietary carbohydrate [3] is a well-established determinant of postprandial glucose responses, and so is the type of dietary fat [26, 27] which is believed to reduce postprandial glycemic excursions by delaying gastric emptying and by enhancing the secretion of incretins. The degree of saturation of dietary fat can also influence glucose and insulin responses to carbohydrate-containing high fat meals [28].

The interacting effects of co-ingesting different types of fat and carbohydrate on postprandial TG and glucose metabolism are not clear. A better understanding of this interaction has important clinical implications, because in real life, humans consume mixed meals containing different types of carbohydrate and fat. The purpose of this study was to compare the effect of ingesting isocaloric mixed meals containing a moderate amount of fat (40 g) and available carbohydrate (50 g), on postprandial TG, non-esterified fatty acids (NEFA), glucose, insulin, and c-peptide, in healthy subjects. Fat was provided in the form of three common sources varying in the degree of saturation: olive oil (n-9; MUFA), grapeseed oil (n-6; PUFA), or dairy fat in the form of butter (SFA). Rice was chosen as the carbohydrate source, as it is the staple dietary carbohydrate of many Asian populations. In particular, Basmati rice (low GI= 55) and Jasmine rice (high GI= 91) [29] are the two most commonly consumed types of rice in Southeast Asia.

Methods

Subjects

Twenty-three Chinese men were recruited in this study by means of advertisements, flyers, and personal communication (word-of-mouth); 3 subjects dropped out during the study (one due to discomfort with cannulation and two due to inability to commit to study schedule), whereas 20 of them (age: 22-39 years; BMI: 18.3-28.6 kg/m²) completed all study procedures (Table 1). All participants were healthy and none had a family history of either type 2 diabetes or cardiovascular
disease. None of the subjects smoked or used tobacco products, consumed special diets, or took medications known to alter metabolism. All subjects had normal fasting blood glucose concentrations and no history of food intolerance. The present study was conducted according to the guidelines of the Declaration of Helsinki, and all procedures were approved by the Domain-Specific Review Board of National Healthcare Group, Singapore. Written informed consent was obtained from all subjects before participation.

**Study design**

This was a randomized, crossover, single-blinded study with six experimental trials separated by a 1-week washout period to minimize carryover effects. On the evening before each study day (~1830h), participants consumed a standardized evening meal consisting of rice, chicken, and a non-alcoholic beverage. Participants were then asked to refrain from consuming any food except water until they reported to the laboratory the next morning. Participants were also asked to refrain from any vigorous physical activity the day before the study.

For each trial, participants arrived at the Clinical Nutrition Research Centre (CNRC) at 0830 hours in the morning. After a 10-min rest, baseline capillary blood samples were taken 5 min apart (-5 and 0 min) by finger-prick by using the Accu-Chek lancing device (Abbott). Thereafter, an indwelling cannula was inserted into a forearm vein by a registered nurse and a baseline blood sample was obtained (time = 0). Subsequently, participants consumed the test meal at a comfortable pace (12 min on average), and then rated their overall liking of the test meal on a visual analogue scale (VAS). Finger-prick capillary and venous blood samples were collected at 15, 30, 45, 60, 90, 120, 150, 180, 210 and 240 min following the start of the meal. Serum was isolated and stored until further analysis.

Participants remained seated throughout the postprandial period.

**Test meals**

Subjects consumed, in random order, six isocaloric rice-based meals. All meals contained only 50 g of available carbohydrate cooked with 40 g of dietary fat in a rice cooker. The test meals consisted of
either low GI rice (basmati rice, 233 kcal, 59.4 g carbohydrate, 6.3 g protein, and 0.47 g fat) or high
GI rice (jasmine rice, 222 kcal, 50.2 g carbohydrate, 4.52 g protein, and 0.32 g fat), cooked with either
48 g of unsalted dairy fat in the form of butter (24 g SFA, 8.3 g MUFA, 7.7 g PUFA, rich in SFA), or
44 g of refined olive oil (6 g SFA, 31 g MUFA, 3 g PUFA, rich in MUFA), or 40 g of refined
grapeseed oil (4 g SFA, 7.6 g MUFA, 28.4 g PUFA, rich in PUFA). The rice and dietary fat were
bought from a local supermarket, and the calorie and nutrient contents of the test meals were
calculated on the basis of the weight of the components and the dietary information provided by the
manufacturer. The test foods were freshly prepared in the morning of the test days.

Sample analysis

Blood glucose concentration was measured by using the HemoCue glucose 201 analyzer (Hemo-Cue
Glucose 201 RT), with intra- and inter- assay CVs <2%. Serum insulin and c-peptide concentrations
were determined by commercially available electrochemiluminescence immunoassays using the
Cobas e411 analyzer (Roche Diagnostics), with intra- and inter- assay CVs < 3%. Serum TG was
measured by using the Cobas e311 analyzer (Roche Diagnostics), with intra- and inter- assay CVs
<2%. Serum NEFA was measured by using a commercially available enzymatic colorimetric assay
(Wako Chemicals, Japan), with intra- and inter- assay CVs < 3%.

Statistical analysis

We estimated that a sample size of 19 subjects would allow us to detect a difference of 25% in
postprandial triglyceride iAUC (our main outcome measure) between experimental meals, at α = 0.05
with a power of 80% (type II error, β = 0.2). Differences in the concentrations of TG, glucose, insulin,
c-peptide and NEFA were evaluated by using repeated measures three-factor ANOVA, with main
effects for rice type (low GI vs high GI), fat type (butter vs olive vs grapeseed), and time, as well as
their interactions. The incremental areas under the curve (iAUC) were calculated as summary
measures by using the trapezoidal rule, and data were analyzed by repeated measures two-factor
ANOVA (rice type x fat type). Data are presented as means ± SEM, unless otherwise stated. A P-
value< 0.05 was considered statistically significant. Statistical analysis was performed by using SPSS software version 16 (SPSS Inc.).

Results

The participants consumed the test meals without any problems; overall liking did not differ among test meals. There were no significant differences in fasting concentrations of glucose, insulin, c-peptide, TG and NEFA among the six experimental days.

In the analysis of TG, NEFA, glucose, insulin, and c-peptide concentrations over time, we found no significant 3-way interactions (rice type x fat type x time) and no significant 2-way rice type x fat type interactions. However, for all metabolites, the 2-way time x rice type and time x fat type interactions were statistically significant. Similarly, when iAUCs were used in the analysis as summary measures of the whole time courses, we found no significant 2-way rice type x fat type interactions. These results indicate that postprandial metabolite responses are affected by the type of fat and carbohydrate ingested in an independent manner, rather than interactive, i.e. the effects of fat are not modified by the carbohydrate type, and vice versa. For this reason, and to facilitate presentation, pooled significant results are shown in the Figures to facilitate presentation of the independent effects of carbohydrate and fat.

For plasma TG, there were significant rice type x time (P<0.01) and fat type x time (P<0.01) interactions. TG iAUC differed significantly according to the type of fat ingested (P<0.01) but not carbohydrate (P=0.22), and was greater after the olive oil meal than after the grapeseed oil or butter meals (Table 2 and Figure 1). There were no significant rice type x time (P=0.07) and fat type x time (P=0.14) interactions for plasma NEFA response. Postprandial NEFA concentrations decreased after meal ingestion, as a result of meal-induced suppression of adipose tissue lipolysis, and returned to baseline by the end of the observation period, without any significant differences between meals (Table 2). There were significant rice type x time (all P<0.01) interactions for plasma glucose, insulin and c-peptide but no significant fat type x time (all P>0.05) interactions. Glucose, insulin, and c-
peptide iAUCs were significantly different between the low GI and high GI rice meals, but not different according to the type of fat ingested (Table 2 and Figures 3-5). Postprandial blood glucose, serum insulin and serum c-peptide responses were significantly greater after ingestion of the high GI rice than after the low GI rice, independent of the type of co-ingested fat.

**Discussion**

The present study evaluated the effects of various mixed meals on postprandial glucose and lipid responses in healthy Chinese male subjects. All meals were isocaloric and had the same macronutrient composition, and differed only in the GI of their carbohydrate and the degree of saturation of their fat. Our results showed that postprandial TG responses are affected by the saturation of dietary fat, with olive oil (MUFA) resulting in greater postprandial TG concentrations as compared to dairy fat butter (SFA) and grapeseed oil (PUFA); this effect was independent of the GI of the meal. Blood glucose, insulin and c-peptide concentrations were lower after the low GI meals compared to the high GI meals and this was independent of the type of dietary fat. Despite the greater insulin concentrations after the high GI than low GI meals, NEFA concentration responses were similar, indicating that acutely after consuming a high GI meal (i.e. in the postprandial period), adipose tissue exhibits some degree of insulin resistance which is compensated for by more circulating insulin.

Postprandial TG concentrations are a significant independent risk factor for cardiovascular disease [8-11, 30]. The magnitude of the postprandial TG response is directly dependent on the amount of fat ingested [22]. The addition of high GI carbohydrate to a fat-rich meal, notably rice or rice-based cuisines that are typical of the Asian diet, amplifies the postprandial TG response compared with a meal that contains only fat. The type of carbohydrate also affects postprandial lipemia, as serum TG concentrations were found to be lower after ingestion of meals containing glucose and fat than those containing sucrose, fructose and fat [18]. Thus, the effects of carbohydrate on postprandial lipemia may depend on the glycemic index of the dietary carbohydrate. In our study, we did not find any significant differences between low and high GI rice meals on postprandial TG concentrations.
However, even if the glycemic index of the dietary carbohydrate does not affect the magnitude of postprandial lipemia, it may affect certain other aspects of postprandial TG metabolism. The postprandial TG response was significantly greater after the olive oil-containing meal than after the grapeseed oil- or butter-containing meals, which indicates that dietary MUFA increase the magnitude of postprandial lipemia as compared to isocaloric amounts of SFA and PUFA. Changes in fat digestion, absorption, intestinal TG re-synthesis and secretion may all affect postprandial TG responses after consumption of meals with different fatty acid composition [14]. Our study cannot directly assess the mechanism for the differences in postprandial TG responses among the different fat-containing meals, but several possibilities can be put forth and others can be excluded. For example, the size of chylomicrons formed in vitro is greater in the presence of MUFA (oleic acid) than SFA (palmitic acid) [32], which may affect subsequent intravascular metabolism and TG clearance. On the other hand, since the absorption efficiency of linoleic acid is similar to that of oleic acid [33], it is unlikely that differences in absorption rate are responsible for the observed differences in postprandial TG responses among meals. Generally, n-6 PUFA-rich meals lead to lower [16, 34, 35] or similar [14, 15] postprandial lipemia compared with MUFA-rich meals in healthy subjects. Purcell et al [35] demonstrated that a high-linoleic acid sunflower oil meal (PUFA) induced a lower postprandial TG response than a high-oleic acid sunflower oil meal (MUFA) in healthy men, in agreement with our results. Also, most studies [14-16] found that olive oil leads to greater postprandial TG responses compared to butter, milk fat, or safflower oil, which is consistent with our findings. However, Thomsen et al [17] reported the opposite (postprandial TG response after olive oil was lower than after butter). The amount and nature of dietary fat, composition of the test meals and subject’s fasting TG concentrations were different among these studies, which could partly explain the discrepant results. A previous study conducted in Japan by Higashi et al [16] found that olive oil ingestion increased postprandial TG concentrations compared to milk fat and safflower oil ingestion, which is consistent with our findings. This study, like ours, was conducted in Asian healthy males. Ethnic differences between Asians and Europeans in lipid and lipoprotein metabolism could thus be another source of variability among studies.
In a previous study, we reported that the glycemic response to Jasmine rice (high GI) was attenuated when the rice was co-ingested with ground nuts oil, with little or no change in the insulin response [36]. This observation prompted us to question whether different dietary fatty acids have similar effects on postprandial substrate metabolism when co-ingested with either high GI or low GI rice-based meals. Contrary to our hypothesis, in our present study we found that different types of dietary fat did not differentially affect glucose and insulin responses to meal ingestion. Nevertheless, when compared to the magnitude of postprandial glycemia after ingestion of carbohydrate-only low GI or high GI rice meals, studied previously [29], the addition of fat attenuated postprandial glycemia, as expected [36], but the magnitude of the reduction was approximately twice as great when fat was co-ingested with high GI rice than with low GI rice (~30% and ~15% reduction), and independent of the type of dietary fat. On the other hand, co-ingestion of fat with carbohydrate (low or high GI) led to similar insulinemic responses as carbohydrate alone [29]. The decreased glucose response to carbohydrate ingestion in the presence of fat could be the result of delayed rate of gastric emptying, leading to reduced rate of carbohydrate absorption [37, 38], or improved sensitivity to circulating insulin in the postprandial period. Glucose, insulin and c-peptide concentration responses to the low GI rice meals were significantly lower than the corresponding responses to the high GI rice meals, as expected. In our study, we found that co-ingestion of different types of dietary fatty acids does not modify the responses to low or high GI carbohydrate. This observation is in line with most [14, 17, 39, 40] but not all previous studies. For example, Gatti et al.[41] reported that postprandial glycemic response was reduced after the addition of olive oil and corn oil, but not butter; and Pedersen et al [42] reported that PUFA-rich meals lowered insulin (but not glucose) responses compared to MUFA-rich meals. The variability in these findings may be related to subject characteristics, the duration of the postprandial period, or the type of fat and carbohydrate in the test meals. Generally, however, the weight of evidence suggests that substitution of MUFA/PUFA for SFA will not improve postprandial glycemia and insulinemia. The increase in c-peptide concentration is in line with the greater insulin concentration after the high GI meals, and likely reflects augmented insulin secretion. Rasmussen et al observed that co-ingesting butter with carbohydrate increased insulin and lowered glucose responses.
compared to olive oil in type 2 diabetes [43]. However, this effect was not evident in healthy subjects [17], which implies results could vary by diabetic status and, by extrapolation, by baseline level of insulin sensitivity and pancreatic function. This possibility needs to be investigated further.

There are some limitations in our study. First, a 4 h postprandial test period in this study may not be sufficient as the TG concentration did not return to baseline for all 6 treatments. Thus the difference between treatments after 4 h was not known. Second, although the volunteers are described as 'healthy', three of the volunteers were estimated to be insulin resistant using HOMA-IR (ratio > 2.5).

Third, this study only recruited healthy Chinese male participants so we cannot be certain our results are representable for other populations.

In conclusion, the present study showed that co-ingestion of olive oil with either low or high GI carbohydrate induces greater postprandial TG responses than co-ingestion of butter and grapeseed oil, without differences in glucose, insulin and c-peptide concentrations in healthy Chinese male subjects. Ingestion of low GI rice with all types of fat induced significantly lower glucose and insulin responses compared to high GI rice, which indicates that the predominant effects of GI on postprandial glycemia and insulinemia are not modified in any way by co-ingestion of various types of fat. Future studies should address the mechanisms that underlie these observations.
Acknowledgments

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Conflict of Interest

None of the authors has any conflict of interest to declare.
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Figure 1: Postprandial serum triglyceride responses between butter, olive and grapeseed treatment group regardless of the type of rice. Data were analysed using three-factor repeated measures ANOVA (time * fat type * rice type) for comparison among the 6 treatments. There were no significant interactions for rice type * fat type (P=0.917) and rice type * fat type * time (P=0.701), but there were significant interactions for rice type * time (P<0.01) and fat type * time (P<0.01). All values are mean ± SEM, total n = 20.

Figure 2: Postprandial blood glucose responses between low GI and high GI treatment group regardless of the type of fat. Data were analysed using three-factor repeated measures ANOVA (time * fat type * rice type) for comparison among the 6 treatments. There were no significant interactions for rice type * fat type (P=0.211), rice type * fat type * time (P=0.792), and fat type * time (P=0.120), but there was a significant interaction for rice type * time (P<0.01). All values are mean ± SEM, total n = 20.
Figure 3: Postprandial serum insulin responses between low GI and high GI treatment group regardless of the type of fat. Data were analysed using three-factor repeated measures ANOVA (time * fat type * rice type) for comparison among the 6 treatments. There were no significant interactions for rice type * fat type (P=0.964), rice type * fat type * time (P=0.671), and fat type * time (P=0.112), but there was a significant interaction for rice type * time (P<0.01). All values are mean ± SEM, total n = 20.

Figure 4: Postprandial serum C-peptide responses between low GI and high GI treatment group regardless of the type of fat. Data were analysed using three-factor repeated measures ANOVA (time * fat type * rice type) for comparison among the 6 treatments. There were no significant interactions for rice type * fat type (P=0.941), rice type * fat type * time (P=0.558), and fat type * time (P=0.210), but there was a significant interaction for rice type * time (P<0.01). All values are mean ± SEM, total n = 20.
Table 1: Anthropometric and fasting metabolic characteristics of the study subjects

<table>
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<tr>
<th>Characteristic</th>
<th>Mean ± SD</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age (yrs)</td>
<td>26.8 ± 5.9</td>
<td>22 - 39</td>
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<tr>
<td>Weight (kg)</td>
<td>68.8 ± 9.9</td>
<td>50.4 - 90.5</td>
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<td>Height (m)</td>
<td>1.73 ± 0.05</td>
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<td>Body mass index (kg/m²)</td>
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<tr>
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<td>Hip circumference (cm)</td>
<td>94.6 ± 6.8</td>
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<td>Systolic blood pressure (mmHg)</td>
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<tr>
<td>Diastolic blood pressure (mmHg)</td>
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<td>Fasting triglyceride[^] (mmol/L)</td>
<td>1.04 ± 0.46</td>
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<td>Fasting glucose[^] (mmol/L)</td>
<td>4.49 ± 0.28</td>
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<tr>
<td>Fasting insulin[^] (µU/ml)</td>
<td>9.28 ± 4.89</td>
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<td>HOMA-IR score[^]</td>
<td>1.86 ± 0.97</td>
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[^] HOMA-IR: homeostasis model assessment of insulin resistance. Values represent the average of the six study visits.
**Table 2:** Incremental areas under the curve (iAUC) for 240 minutes after ingestion of isocaloric mixed meals containing carbohydrate of different glycemic index type (low GI basmati rice vs high GI jasmine rice) and fat of different saturation (butter vs olive oil vs grapeseed oil)

<table>
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<th>Carbohydrate Type (C)</th>
<th>Fat Type (F)</th>
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<tr>
<td>Basmati rice</td>
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<tr>
<td>Jasmine rice</td>
<td>Grapeseed oil</td>
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<tr>
<td>Triglyceride (mmol*min/L)</td>
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<td>0.22</td>
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<tr>
<td></td>
<td>103±15</td>
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<td>NEFA (mmol*min/L)</td>
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<tr>
<td></td>
<td>-20±6</td>
<td>0.92</td>
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<tr>
<td></td>
<td>-20±6</td>
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<tr>
<td>Glucose (mmol*min/L)</td>
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<tr>
<td></td>
<td>193±16a</td>
<td>0.02</td>
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<tr>
<td></td>
<td>221±12b</td>
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<td>Insulin (μU*min/ml)</td>
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<tr>
<td></td>
<td>3411±328a</td>
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<tr>
<td></td>
<td>4560±394b</td>
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<td>C-peptide (ng*min/ml)</td>
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<tr>
<td></td>
<td>405±35a</td>
<td>&lt;0.001</td>
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<td>525±38b</td>
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NEFA: non-esterified fatty acid. Data were analyzed by repeated measures 2-factor ANOVA. Values with different superscripts are significantly different from each other (P<0.05).