NUTRIOSE® soluble fibre supplementation as an effective dietary strategy to improve glycaemic response

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Abstract

Purpose: Postprandial glycaemic control is critical for diabetes prevention and management. Various dietary strategies have been explored to modulate postprandial glycaemia, with the inclusion of innovative fibres showing promising benefits in reducing daily glycaemic load and improving overall glycaemic control.

Methods: In this study, we conducted an 8-week, randomised, controlled, parallel-arm trial involving 78 healthy adults living in Singapore (average age: 39.9 ± 10.6 years; 48 men and 30 women), who were divided into two subgroups: the overweight group (OG) and the genetic risk group (GRG).

Results: Daily supplementation with 40 g of NUTRIOSE® soluble fibre significantly improved 24 h glucose homeostasis under free-living conditions. Compared to the postprandial glycaemic response after breakfast and lunch, NUTRIOSE® supplementation for 8 weeks resulted in a more substantial improvement in the glycaemic response after dinner. Furthermore, one day of NUTRIOSE® supplementation led to improved glucose management in both participant subgroups, with more significant improvements observed in the OG group compared to the GRG. However, a reduction in appetite following NUTRIOSE® supplementation was mostly observed in the GRG. Additionally, NUTRIOSE® consumption led to a decrease in PBFtrunk% and an increase in FFMtrunk in female participants in the GRG.

Conclusion: Incorporating innovative fibres can serve as an effective dietary strategy to lower daily glycaemic load and enhance glycaemic control, offering significant public health benefits and encouraging the consumption of resistant dextrin. However, further investigation into the role of chrono-nutrition is warranted, as it plays a key role in understanding glucose homeostasis throughout the day.

Keywords: resistant dextrin; innovative fibres; continuous glucose monitoring; glycaemic response; glucose homeostasis; satiety

Introduction

In recent decades, the prevalence of type 2 diabetes (T2D) and its associated risk factors has surged across Asia, with Singapore being no exception [1, 2]. Improved economic prosperity, technological advancements and enhanced food production, availability and safety have contributed to a lifestyle characterized by decreased physical activity and a diet higher in carbohydrates, sugars, and fats, but lower in fibre [3]. These factors collectively contribute to the rising incidence of diabetes in the region.

Dietary fibre offers numerous health benefits, such as reducing the risks of cardiovascular disease (CVD), T2D, stroke, obesity, and gastrointestinal diseases [4]. However, dietary fibre intake often falls below recommended levels [5, 6]. Although traditional Asian diets are typically rich in plant-based foods, meeting daily fibre recommendations has become increasingly difficult due to food processing, refinement, and a shift towards a more Westernized diet. In Singapore, the Health Promotion Board (HPB) recommends a daily dietary fibre intake of 20 g for women and 26 g for men, yet the average daily intake among Singaporeans is only 13 g [7]. While increasing the consumption of fruits and vegetables remains the primary strategy to promote fibre intake, an alternative is to supplement a daily diet with dietary fibre, particularly soluble fibre, to improve health.

Resistant dextrin is a non-viscous soluble fibre [8]. Unlike other viscous soluble fibres that form gel-like texture, which limits their use in foods, resistant dextrin can be easily incorporated into various foods and beverages and is well-tolerated [9, 10]. Several studies have examined the effects of resistant dextrin supplementation, particularly its role in appetite regulation [11]. By influencing orexigenic hormones such as ghrelin, resistant dextrin can help suppress hunger and increase satiety [12, 13]. This appetite suppression can lead to reduced daily energy intake and increased weight loss [14, 15]. Moreover, resistant dextrin has been shown to reduce insulin resistance in both healthy individuals and those with T2D,

contributing to improved blood glucose control [16, 17]. In term of cardio-protective effects, resistant dextrin has been found to lower blood cholesterol levels and reduce inflammation biomarkers [17, 18]. Recent evidence suggests that resistant dextrin also exhibits prebiotic properties and modulates gastrointestinal ecology [19, 20]. The colonic fermentation of resistant dextrin increases the concentrations of faecal enzymes in humans [21] and promotes the production of short-chain fatty acids (SCFAs), such as acetic, propionic, and butyric acids, which can affect glucose homeostasis in the body [22].

A previous U.S. population study [23] found that a family history of diabetes is a significant risk factor for T2D, probably due to shared genetic and environmental factors [24, 25]. This indicates that the risk of developing T2D increases with the number of family members affected by the condition. Similarly, overweight people are nearly 2.4 times more likely to develop T2D compared to those of healthy weight [26]. However, evidence from clinical trials on the effects of resistant dextrin on blood glucose, lipid homeostasis, energy balance, and body composition, particularly among Asian participants at increased risk for T2D, is still insufficient to make specific dietary recommendations. Furthermore, Asians are phenotypically distinct from Caucasians, as they are more susceptible to T2D, even with a lower body mass index (BMI) [27]. This study aims to investigate whether daily supplementation with resistant dextrin over 8 weeks can improve glycaemic control and insulin sensitivity in Asians at increased risk for T2D, such as those who are overweight or have direct family members with T2D.

Methods

This randomised control trial was conducted in the Clinical Nutrition Research Centre (CNRC), Singapore, and registered at ClinicalTrials.gov as NCT06554002. All procedures involving human subjects were approved by the National Healthcare Group Domain Specific Review Board, Singapore (DSRB 2017/00558). Written informed consent was obtained from

all eligible participants prior to the commencement of the trial and the research procedures and trial protocols were followed in accordance with the good clinical practice (GCP) guidelines [28] and with the ethical standards in concordance to the Declaration of Helsinki, 1983.

Study population

A total of 114 participants were screened from the public through advertisements online, in newspapers, flyers, posters, and personal communications. Participants underwent an initial screening and various measurements to determine their eligibility for the study. These measurements included (1) anthropometry [e.g., height, weight, waist circumference (WC), and hip circumference (HC)], (2) habitual dietary intake, (3) habitual physical activity levels, (4) the Three-Factor Eating Questionnaire, (5) blood pressure (BP), and (6) fasting blood glucose (FBG).

Ninety-eight participants were considered eligible because they met the following inclusion criteria: healthy Chinese males or females (aged 21-60 years); not pregnant; not diagnosed with any medical conditions; not consuming any fibre supplements or any prescription medication likely to affect energy metabolism, blood glucose, insulin, or lipids; not allergic or intolerant to any of the test foods; and not intentionally restricting food intake. Participants also had to fall into one of the following categories: genetic risk group (GRG) - BMI 21 - 25 kg/m^2 with a first-degree family history of T2D, or overweight group (OG) - BMI 23 - 30 kg/m^2, with a BMI of 23 used as the cut-off point to define overweight in the Chinese population [29, 30].

Study design and protocol

This study was conducted using a randomised, controlled, parallel-arm design with 2 arms: control and treatment. After an overnight fast, participants who expressed interests and met all

inclusion criteria attended a screening session, where informed consent was obtained along with anthropometric measurements. Participants were first divided by gender and then further categorized according to the inclusion criteria into either GRG or OG. Participants within each group were then block-randomised into either the control or treatment group. Of the 98 eligible participants, 78 completed the study (control: n = 38 and treatment: n = 40).

Participants were required to follow the entire study protocol, which included visits to the lab at three separate time points: the baseline test session, the mid-intervention visit, and the final test session. Both the baseline and final test sessions involved 3-day visits, while the mid-intervention visit was completed in one day. Twice daily (once after breakfast and once after lunch) for 8 weeks, participants in the control group consumed a flavoured beverage powder with 3 g glucose, while those in the treatment group consumed a flavoured beverage powder with 20 g NUTRIOSE® FB06 (ROQUETTE Frères). Both beverage sachets were provided in identical packaging and labelled with numbers for blinding purposes. The control sachet contained the same quantity of available carbohydrates as the resistant dextrin sachet. The study timeline and measurements are illustrated in Figure S1.

Meal plan

Participants were provided with a standardized dinner on Day 1 and three standardized meals on Day 2 for both the baseline and final test sessions (Fig. S1). Table S1 shows the meal plan for the standardized dinner. The meal plan for Day 2 was designed based on the average energy intake recommended by the HPB, Singapore. An example of the Day 2 meal plan is shown in Table S2 for females and Table S3 for males. Portion sizes were adjusted based on the energy requirements recommended for each gender (2200 kcal/d for males and 1800 kcal/d for females). The macronutrient proportions in the meals (60% carbohydrates, 25% fats, and 15% protein) were the same for both males and females.

Glucose analysis

156 The iPro™2 Continuous Glucose Monitoring System (CGMS) (iPro™2 Professional CGM,
157 Medtronic MiniMed, Northbridge, CA, USA) was used to measure glucose levels during this
158 study. The CGMS sensor was inserted on Day 1 at 4:00 pm and removed on Day 3 at 10:00
159 am for both the baseline and final test sessions (Fig. S1). The collected data was processed
160 using the Medtronic Diabetes CareLink iPro online software (https://carelink.minimed.eu).
161 The reported data represented interstitial glucose readings recorded every 5 min for up to 42
162 h. Participants were instructed to calibrate the CGMS sensor against finger-prick blood
163 glucose measurements four times a day (before each meal and before sleep) using the
164 OneTouch Ultra®2 blood glucose metre (LifeScan, Inc., Milpitas, CA, USA).

165 *Satiety analysis*

166 A 100-mm Visual Analog Scale (VAS) [31] was used to access participants' hunger and
167 satiety on Day 2 of both the baseline and final test sessions. The VAS measured subjective
168 satiety at baseline and at subsequent 30-min intervals. The appetite rating questionnaire
169 included questions on hunger, fullness, desire to eat, and the amount of food one could eat.
170 Scores were recorded on the 100-mm VAS, with the low end anchored by the most negative
171 or lowest intensity feelings (e.g., not at all hungry, nothing at all), and the high end anchored
172 by opposing terms (e.g., extremely hungry, a large amount). Participants were instructed to
173 read each question carefully and indicate, with a single marking on the line, the option that
174 best reflected their feelings at that moment.

175 *Blood analysis*

176 Venous blood samples were collected in BD Vacutainer® containing spray dried K2EDTA on
177 Day 3 of both baseline and final test sessions. The tubes were centrifuged at 1500 × g for 10
178 min at 4 °C, and stored at -80 °C immediately until further analysis was required.

179 *Body composition analysis*

DEXA (QDR 4500A, fan-beam densitometer, Hologic, Waltham, MA, USA) was used to measure whole-body composition, including fat mass, lean body mass, bone mineral density and content. DEXA measurements were taken on Day 3 of both baseline and final test sessions. To ensure accuracy, participants were instructed to change into a standard robe provided and remove any jewellery. Percentage body fat (PBF%) was calculated and provided by the manufacture's software. Due to the radiation exposure, DEXA measurement was not taken during the mid-intervention visit.

Bioelectrical impedance analysis (BIA) measurements were carried out using an 8-electrode BIA device (Tanita BC-418, Tokyo, Japan) on Day 3 of both baseline and final test sessions, as well as during the mid-intervention visit. Participants were instructed to remove all footwear and socks, and step onto the BIA platform barefooted. The electrodes connected to the foot pads sent a low electrical current through the body, and PBF% was calculated and displayed based on prediction equations provided by the manufacture.

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 23. All data are expressed as means ± standard deviation (SD). One-way ANOVA was used for between-group comparisons. A two-sided $p < 0.05$ was considered statistically significant in all cases. The normality of the data distribution was assessed using the Shapiro-Wilk test, with a *p*-value < 0.05 indicating a statistically significant deviation from the normal distribution. Data were analysed for the entire cohort as well as for the GRG and OG subgroups separately. Gender was controlled for in the regression models, which accounted for the clinical measures of participants in both the treatment and control groups. Area under the curve (AUC) values for hunger and satiety were calculated using the trapezoid rule [32].

Results

Of the 98 participants, 20 withdrew or were terminated from the study for various reasons: failure to meet study requirements and/or commit to study schedule (n = 17), unexpected allergic reactions (n = 1), and unexpected pregnancy (n = 2). This left 78 participants who successfully completed the entire study (Fig. 1). Table 1 shows the basic characteristics of the study population, including differences between females and males. At baseline, there were no significant differences between participants in the control and treatment groups in terms of mean age, BMI, PBF%, FBG, and lipid panels. Males, but not females, exhibited borderline high concentrations of low-density lipoprotein cholesterol (LDL-C), but total cholesterol (TC) and triglycerides (TG) were well below the reported normal levels of 5.2 and 1.7 mmol/L, respectively [33].

Out of the 78 participants, 66 completed both arms of the CGMS measurements, with 33 in the control group and 33 in the treatment group. Before the intervention (at week 0), Figure 2a shows no significant differences in fasting glucose concentrations between the control and treatment groups. However, directly following the intake of NUTRIOSE® supplementation, participants in the treatment group showed a reduced postprandial glucose response compared to those in the control group. Specifically, small, significant differences were observed at certain time points after lunch (575 and 580 min) and dinner (740, 745, and 750 min). As shown in Table 2, the NUTRIOSE® supplementation resulted in a lower 24 h glucose iAUC (965 ± 639 vs 1121 ± 751 mmol/L), and a decreased postprandial glucose iAUC after breakfast (156 ± 113 vs 189 ± 173 mmol/L), lunch (275 ± 200 vs 323 ± 249 mmol/L), and dinner (384 ± 240 vs 509 ± 342 mmol/L). However, these differences did not reach statistical significance.

Similar results were observed at the final test session (at week 8), as shown in Figure 2b and Table 3. Small, significant differences were observed at time points 670 and 675 min before dinner between the control and treatment groups. Table 3 shows that participants consuming

NUTRIOSE® supplementation for 8 weeks spent more time in the normal range of TIR (4.5 – 7%, $p = 0.014$) and less time in the higher range (TIR > 7%, $p = 0.042$) compared to those in the control groups. Table 2 and 3 show no gender effect on the glucose response as measured by the CGMS system, except for dinner iAUC in females and TIR 4.5 – 7% in males.

When participants from different groups, i.e. GRG and OG, were analyzed separately, Table 4 and Figure 3a, 3c, and 3e show no significant differences between the control and treatment groups in the GRG at week 0. Figure 3b, 3d, and 3f show that participants in the OG exhibited a reduced glucose change in the treatment group compared to the control group, although these differences did not reach statistical significance, except for dinner iAUC, as shown in Table 4. However, significant differences were observed in the effects of 8-week NUTRIOSE® supplementation on glucose management between the GRG and OG participants. In the GRG, no differences were observed in the glucose control parameters (Fig. 4a, 4c, 4e, and Table 5). In contrast, in the OG, nearly all glycaemic outcomes showed significant differences between control and treatment groups after 8 weeks (Fig. 4b, 4d, 4f, and Table 5). These differences were evident over the 24 h assessment period and were also reflected in most per-meal parameters.

Figure 5 shows that at week 0, significant differences ($p < 0.05$) were observed in appetite between the control and treatment groups for participants in the GRG. Compared to control, the NUTRIOSE® supplementation resulted in reduced hunger at 90, 150, 180, and 240 min (Fig. 5a), increased satiety at 180, 210, and 240 min (Fig. 5b), reduced desire to eat at 60, 180, 210, and 240 min (Fig. 5c), and reduced prospective amount to eat at 180 and 210 min (Fig. 5d). However, there were no significant differences between the control and treatment groups for measurements taken at the final test session (at week 8) for the participants in the GRG (Fig. 6). The AUC for rated hunger, satiety, desire to eat and prospective amount to eat are shown in Figure 7. Before the intervention (at week 0), significant differences were observed

255 in appetite expressed as AUC between the control and treatment groups for participants in the
256 GRG. Compared to control, the treatment resulted in reduced hunger after breakfast (p =
257 0.008) and over 8 h (p = 0.048, Fig. 7a), increased satiety after breakfast (p = 0.054, Fig. 7b),
258 reduced desire to eat after breakfast (p = 0.010) and over 8 h (p = 0.049, Fig. 7c), and reduced
259 prospective amount to eat after breakfast (p = 0.043) and over 8 h (p = 0.050, Figure 7d).
260 There were no significant differences between the control and treatment groups after the
261 intervention (at week 8) for the participants in the GRG (Fig. 7). Interestingly, there were no
262 significant differences in appetite ratings between the control and treatment groups when
263 comparing participants in the OG before (at week 0) and after (at week 8) the intervention
264 (Fig. 8 and 9).

265 Table 6 shows that after 8-week NUTRIOSE® supplementation, significant reductions in WC
266 and HC were observed in the treatment group compared to the control group for those in
267 GRG. Additionally, female participants in GRG showed a significant increase in trunk FFM
268 and a significant decrease in trunk PBF% in the treatment group compared to the control
269 group. However, for participants in OG (Table 7), only females showed a significant decrease
270 in HC in the treatment group compared to the control group following 8 weeks of
271 NUTRIOSE® supplementation.

272 **Discussion**

273 The current study highlights significant improvements in 24-h glucose homeostasis with
274 twice-daily supplementation of 20 g of NUTRIOSE® soluble fibre, amounting to a total of 40
275 g/d, in Asian participants. These improvements were observed under free-living conditions,
276 without restrictions on energy intake and with minimal changes in body composition. This
277 validates the use of resistant dextrin supplementation as an effective strategy for glycaemic
278 management in Asians.

A previous clinical trial indicated that 12 weeks of resistant dextrin supplementation helped reduce insulin resistance and improved key factors of metabolic syndrome in overweight Chinese men [16]. Similarly, a daily dose of 10 g of resistant dextrin was shown to improve insulin resistance and inflammation in Iranian women with T2D [17]. Although our study did not show more pronounced long-term effects on blood glucose management, the short-term impact of NUTRIOSE® supplementation on blood glucose levels may be due to its slight increase in the viscosity of chyme, which slows carbohydrate digestion and reduces enzyme-substrate interactions [34, 35]. Notably, NUTRIOSE® supplementation appears to have a more pronounced effect on improving postprandial glycaemic response after dinner. In the evening, there is a greater relative reduction in glucose parameters, including iAUC, maximum value, range, and variability, compared to earlier in the day. This aligns with previous studies showing greater improvements in glucose control later in the day [36, 37]. The relationship between diet and circadian rhythm, influenced by factors such as meal timing and nutrient composition, is referred to as chrono-nutrition. Recent study has highlighted the link between biological rhythms, chrono-nutrition, and gut microbiota, showing how this interplay affects various metabolic outcomes [38]. According to our findings, the effects of resistant dextrin supplementation are likely to be most pronounced during the evening, a time when insulin sensitivity tends to be lower and colonic fermentation leads to the accumulation of metabolites, including SCFAs, after a day of fermentation [39]. It has been demonstrated that SCFAs produced by gut microbiota can influence insulin sensitivity and energy balance by mediating GPR43 [40].

In the sub-analysis of GRG and OG groups, we found positive effects on glycaemic responses in the OG group. However, no such effects were observed in participants at genetic risk of T2D (GRG group). While further long-term interventions are necessary, adopting a healthy lifestyle with a fibre-rich diet may aid in managing blood glucose levels [41]. It is possible

304 that the duration of our trial was insufficient to detect a significant improvement in blood
305 glucose response for participants with genetic risks.

306 Previous study shown that 28 days of supplementation with resistant dextrin could modify
307 satiety in healthy adults [11]. However, the impact was only observed in normal-weight
308 adults, not in those who were overweight. This difference may be due to the relatively low
309 dose of 14 g/day, which might not be sufficient to alter satiety in the overweight adults. In the
310 current study, we increased the dose to 40 g/day and found that there was no difference
311 between the control and treatment groups at both week 0 and 8. Similarly, a reduction in
312 appetite following resistant dextrin supplementation was noted in participants in the GRG but
313 not in the OG. This suggests that the higher increase in postprandial blood glucose in the
314 GRG may lead to increased feelings of satiety. These findings align with the glucostatic
315 theory proposed by Jean Mayer more than 50 years ago, which posits that fluctuations in
316 blood glucose levels play a critical role in regulating appetite and food intake [42]. Another
317 possible reason is that the microbial community in individuals with higher body weight might
318 have a different capacity to utilize non-digestible carbohydrate [43].

319 Due to fibre's ability to decrease appetite, and thus food intake, and to increase satiety [44],
320 dietary fibre intake has been reported to be associated with a favourable body weight and
321 body composition [45, 46]. In this study, we did not observe major changes in the participants
322 following resistant dextrin supplementation under free-living conditions. However, initial
323 modifications in body composition were detected, including a decrease in PBFtrunk% and an
324 increase in FFMtrunk. These effects were particularly noticeable in females in GRG group. A
325 previous study [47] found that dietary fibre intake was associated with skeletal muscle mass,
326 body fat mass, and muscle-to-fat ratio in women with T2D, but not in men. Dietary fibre
327 intake has also been linked to lower inflammatory marker levels and improved insulin
328 sensitivity, which can reduce visceral fat and inflammation, ultimately contributing to better

muscle mass retention [48]. In addition, fibre influences the gut microbiome, promoting the growth of bacteria that produce SCFAs, which inhibit inflammatory molecules and regulate immune responses, protecting against muscle atrophy. SCFAs also enhance energy metabolism, glucose homeostasis, and the oxidation of fatty acids in skeletal muscle, further supporting lean body mass preservation. Through these mechanisms, dietary fibre intake helps prevent muscle atrophy by reducing fat accumulation, improving insulin sensitivity, and promoting anti-inflammatory effects, as seen in its positive correlation with FFMtrunk in females. These results, when combined, suggest that while resistant dextrin plays an essential role in appetite regulation and may contribute to weight management, other factors probably influence its effectiveness in achieving significant changes in body weight and composition. Future research may benefit from exploring these dynamics further, particularly in specific populations or with more controlled dietary interventions.

Our study has several notable strengths. One key advantage is the use of CGMS to measure blood glucose responses. This technology allows us to gather comprehensive and real-time data throughout the day, providing a nuanced understanding of the participants' glucose fluctuations. Such detailed monitoring enhances the reliability of our findings and allows for a more thorough analysis of the impact of resistant dextrin supplementation on blood glucose levels. Furthermore, the control group is isocaloric, ensuring that any observed effects are attributable to resistant dextrin supplementation rather than simple changes in calorie intake.

Despite these strengths, there are several limitations that warrant consideration. One significant limitation is that the diet was not rigorously controlled throughout the entirety of the study. While we provided standardized meals during participant visits and offered nutritional guidance, the reliance on self-selected diets during the study period introduced variability that could have influenced the results. Participants may have made food choices based on personal preferences or convenience, potentially affecting the consistency of their

overall dietary intake and, consequently, their metabolic responses. Furthermore, while including participants from both the GRG and the OG was beneficial for broadening the population scope and enhancing the generalizability of our findings, it may also have contributed to increased variability among the study participants. This diversity, while enriching the dataset, could potentially reduce the statistical power of the study, making it more challenging to detect significant differences or trends. Future research could benefit from more stringent dietary controls and a focus on specific populations to further elucidate the efficacy of resistant dextrin supplementation.

Conclusions

In conclusion, despite the free-living conditions with no other intervention apart from resistant dextrin supplementation, our study has identified detectable and clinically relevant differences. The inclusion of daily 40 g of resistant dextrin supplementation has shown a significant and positive impact on glucose homeostasis, particularly in the short and long-term management of post-prandial glycaemia. Given the escalating prevalence of T2D, the potential role of dextrin in diabetes and pre-diabetes management warrants further application and investigation. Our findings indicate that daily supplementation with resistant dextrin effectively reduces postprandial glycaemia in the Asian population. As elevated blood glucose is closely linked to the etiology of diabetes, the inclusion of resistant dextrin could serve as a valuable dietary intervention in this region. Moreover, there is potential for enhanced effects with increased concentration or prolonged use of this product under free-living conditions. Importantly, changes in body composition are not essential prerequisites for achieving blood glucose homeostasis. Our study demonstrates that daily intake of 40 g of resistant dextrin enables free-living individuals, consuming ad libitum food, to effectively reduce their postprandial blood glucose levels. This represents a significant nutritional insight with broad public health implications.

Acknowledgements

The authors greatly acknowledge the financial support and resistant dextrin provided by Roquette Frères.

Conflict of interest

Clementine Thabuis, Laetitia Guérin-Deremaux and Caroline Perreau are employees of Roquette Frères. They were not involved in the study execution, analysis, or data interpretation.

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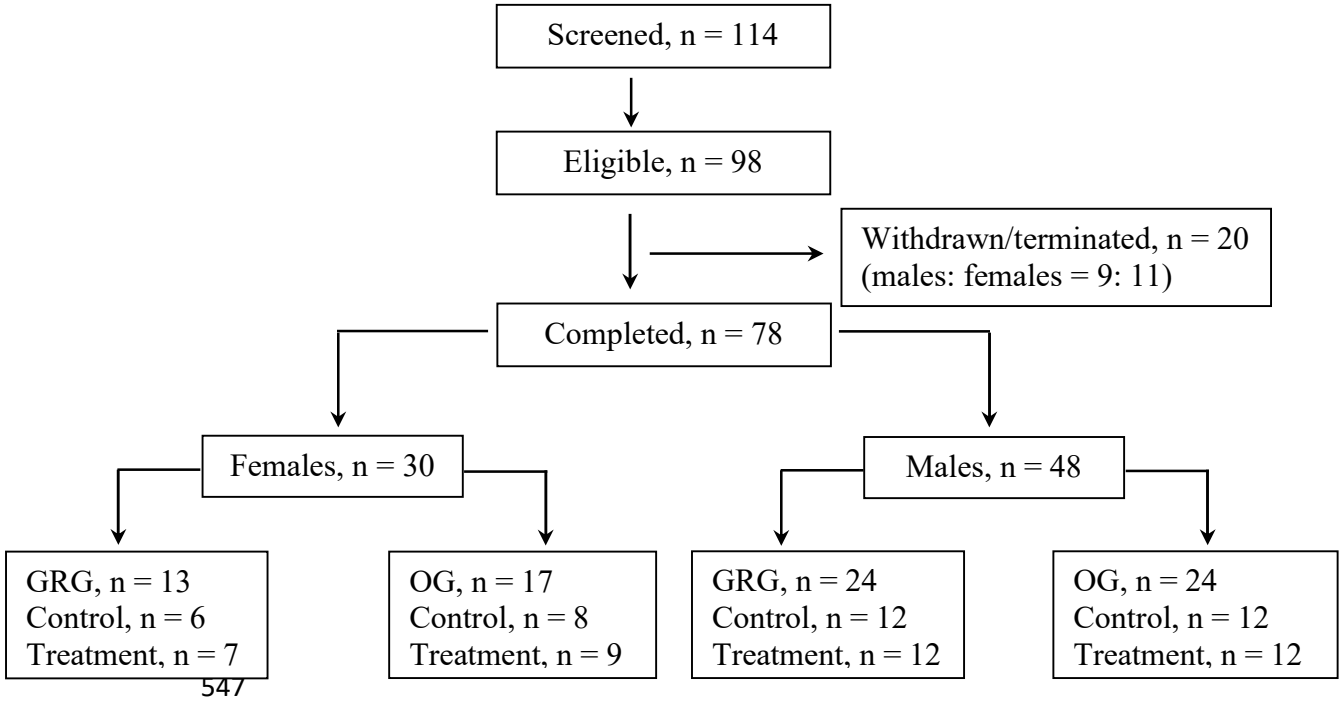


Figure 1. Participant flow diagram. Participants in GRG had direct family members with T2D and BMI between 21 - 25 kg/m^2, participants in OG had a BMI between 23 - 30 kg/m^2.

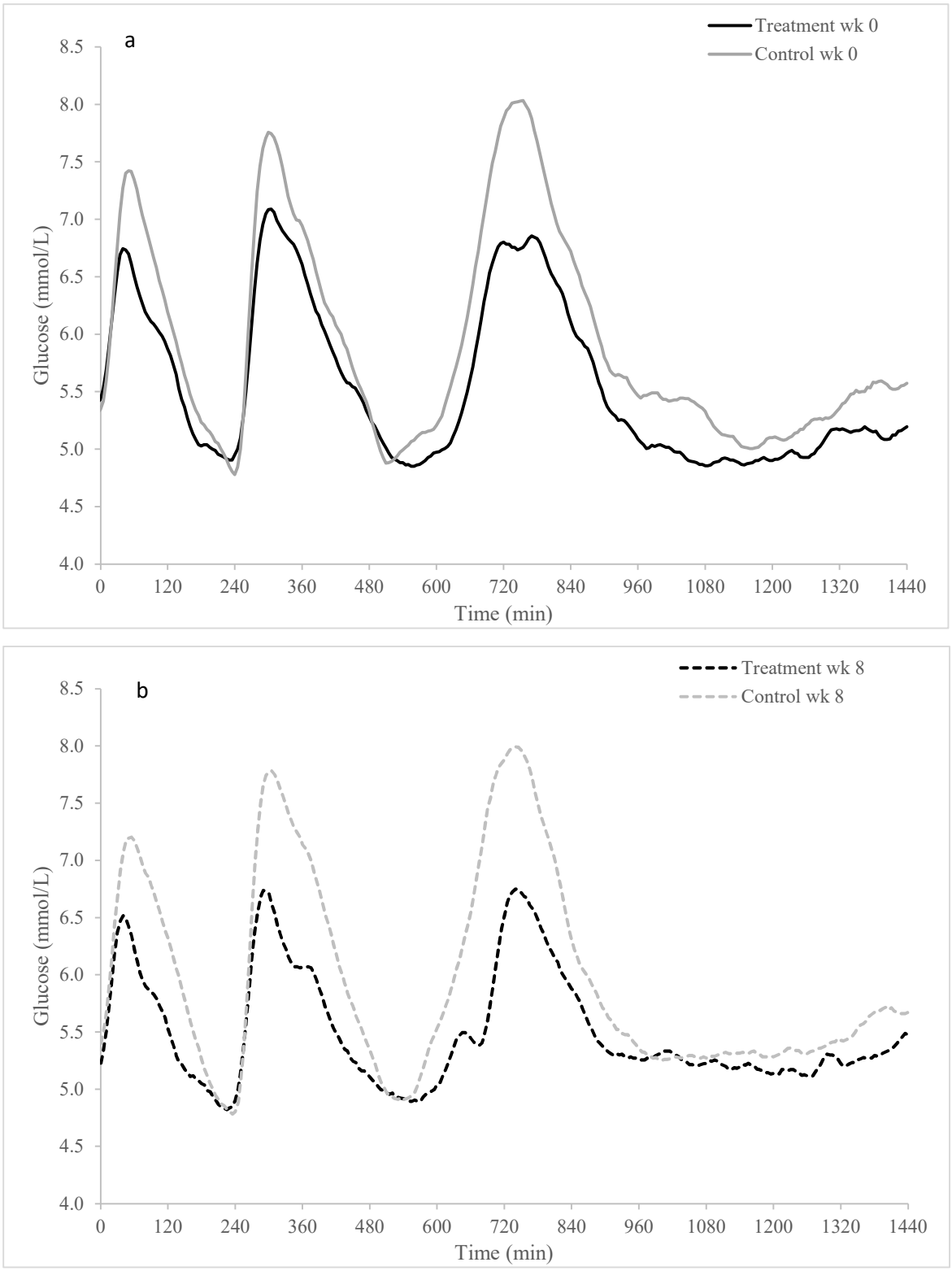


Figure 2. Mean glucose concentration over 24 h for all participants in the control (n = 33) and treatment (n = 33) group (a) before (at week 0) and (b) after (at week 8) intervention as measured with CGMS. Breakfast was consumed at 0 min, lunch was consumed at 240 min and dinner was consumed around 540 min.

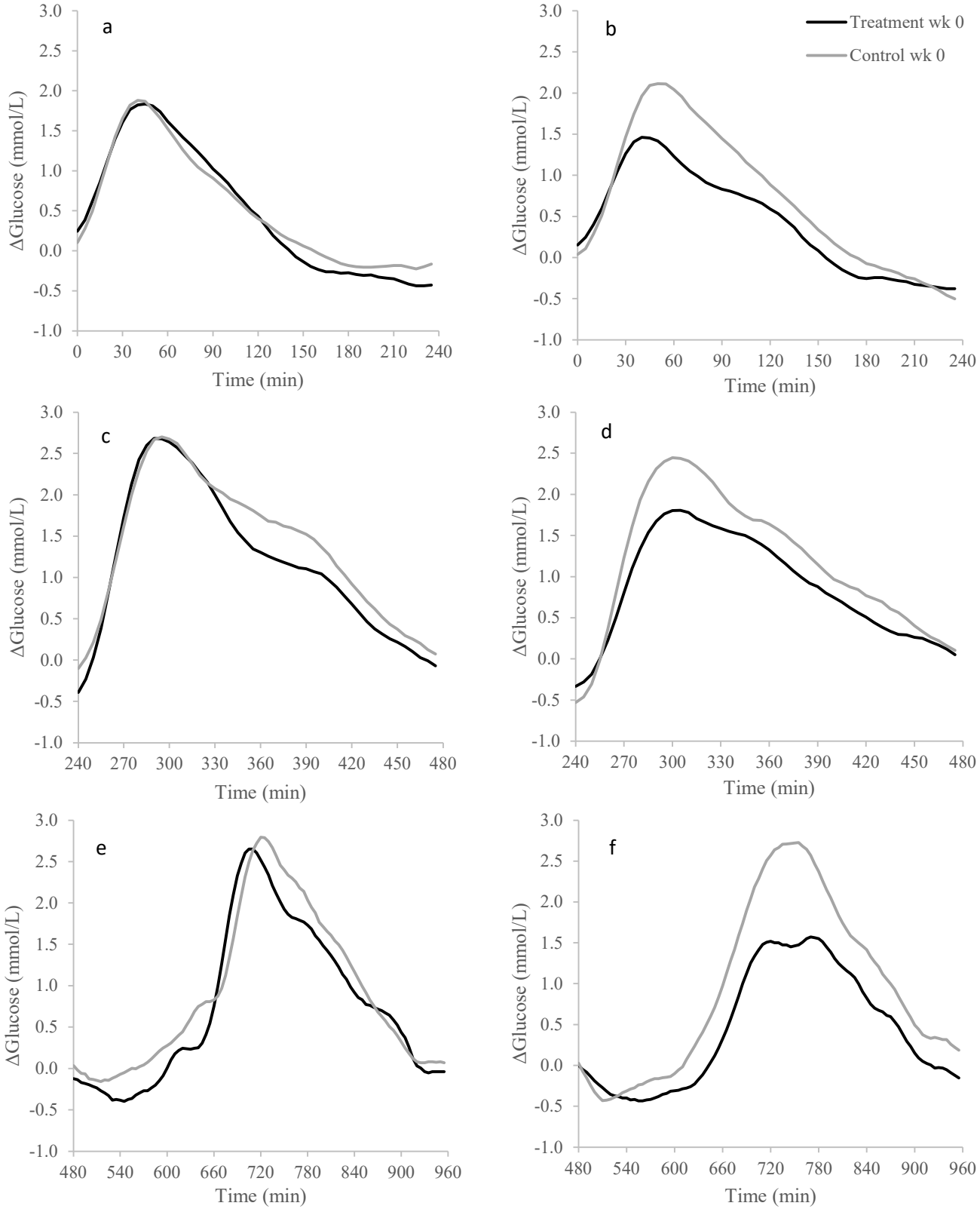
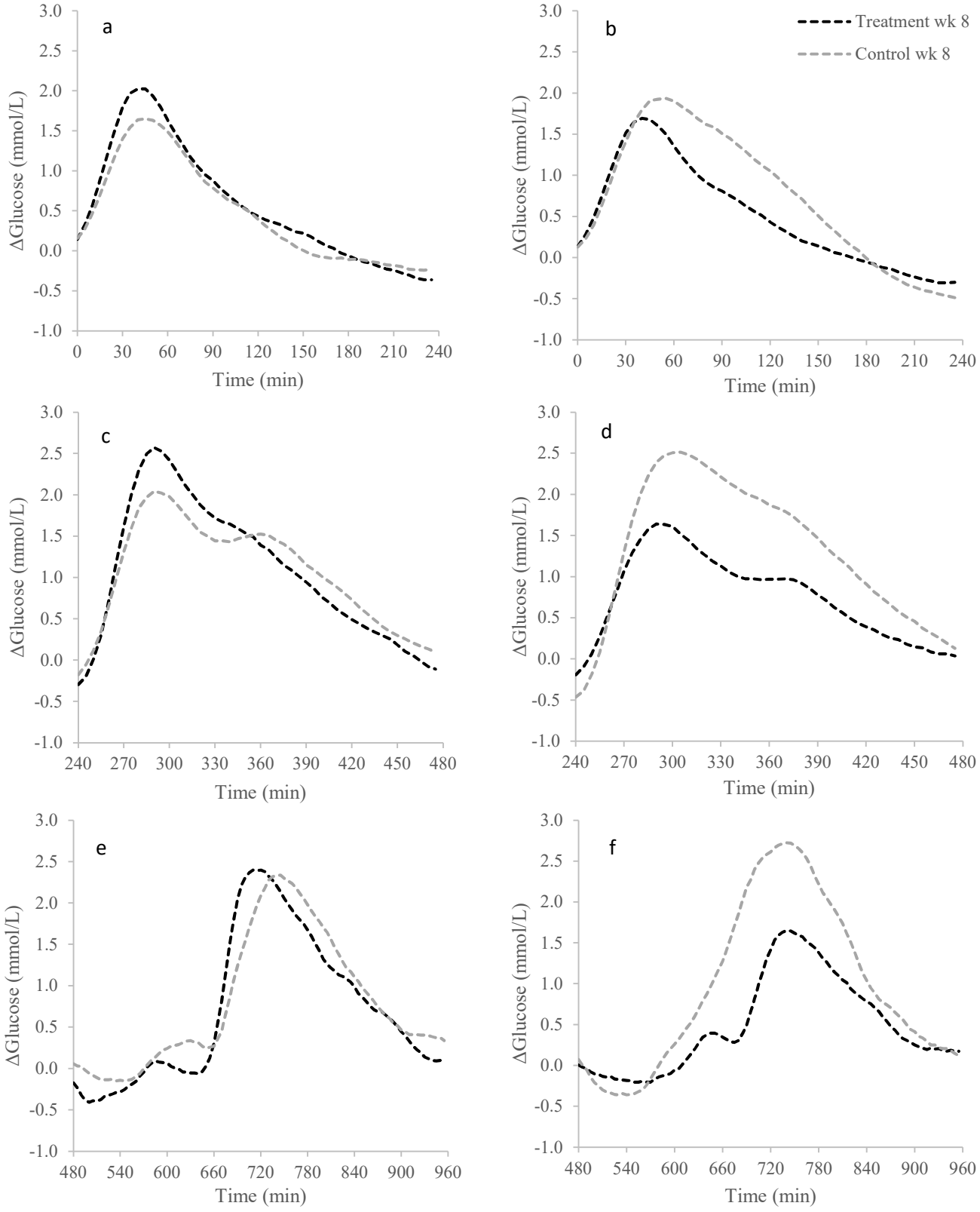


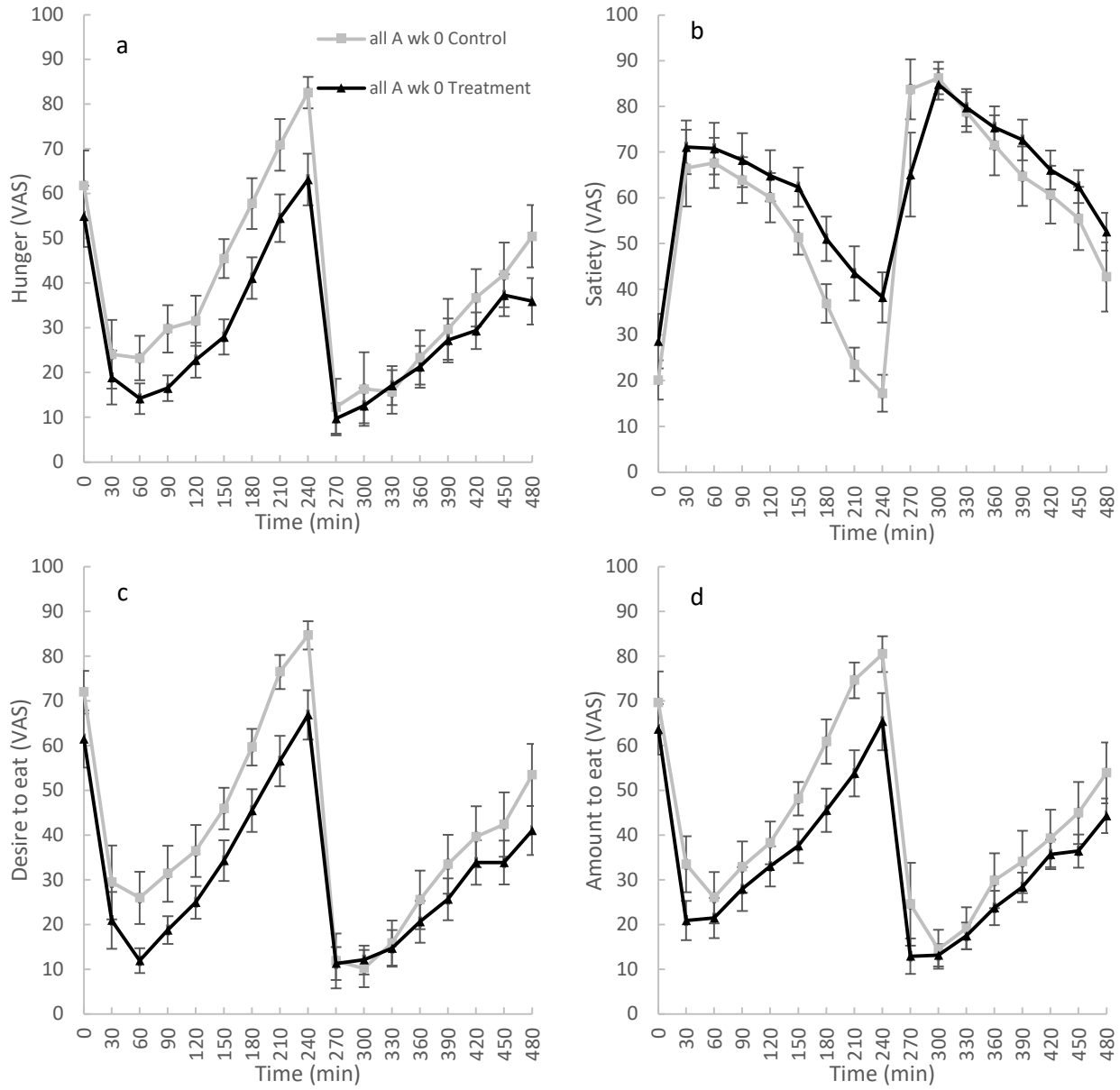
Figure 3. Mean change in glucose from baseline for (a) and (b) breakfast (0 – 240 min), (c) and (d) lunch (240 – 480 min) and (e) and (f) dinner (480 – 960 min) for participants in the control and treatment group before the intervention (at week 0) as measured with CGMS. (a),

(c), and (e) are participants in the GRG (n = 30), whereas (b), (d), and (f) are participants in the OG (n = 36).

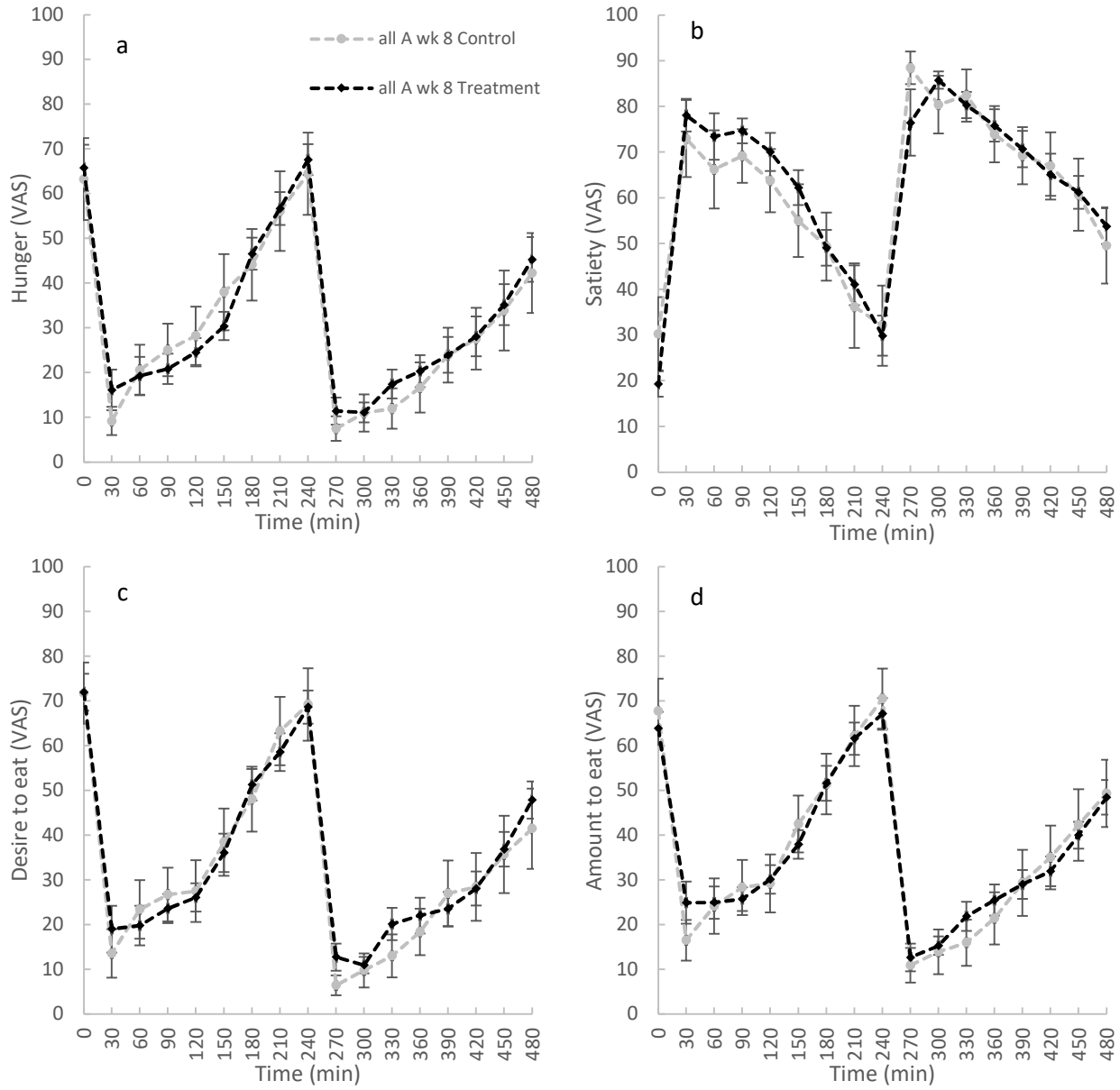


564 **Figure 4.** Mean change in glucose from baseline for (a) and (b) breakfast (0 – 240 min), (c)
565 and (d) lunch (240 – 480 min) and (e) and (f) dinner (480 – 960 min) for participants in the
566 control and treatment group after the intervention (at week 8) as measured with CGMS. (a),

(c), and (e) are participants in the GRG (n = 30), whereas (b), (d), and (f) are participants in the OG (n = 36).



570 **Figure 5.** Changes in rated (a) hunger, (b) satiety, (c) desire to eat, and (d) prospective
571 amount to eat for participants in the GRG, in the control and treatment group before
572 interventions (at week 0) as measured with visual analogue scales (VAS) questionnaires. The
573 total measured time (480 min) starts before breakfast (0 min) and ends 240 min after lunch.
574 Breakfast was consumed at 0 min and lunch was consumed at 240 min, after completing the
575 VAS questionnaires for that time point.



576 **Figure 6.** Changes in rated (a) hunger, (b) satiety, (c) desire to eat, and (d) prospective
577 amount to eat for participants in the GRG, in the control and treatment group after
578 interventions (at week 8) as measured with visual analogue scales (VAS) questionnaires. The
579 total measured time (480 min) starts before breakfast (0 min) and ends 240 min after lunch.
580 Breakfast was consumed at 0 min and lunch was consumed at 240 min, after completing the
581 VAS questionnaires for that time point.

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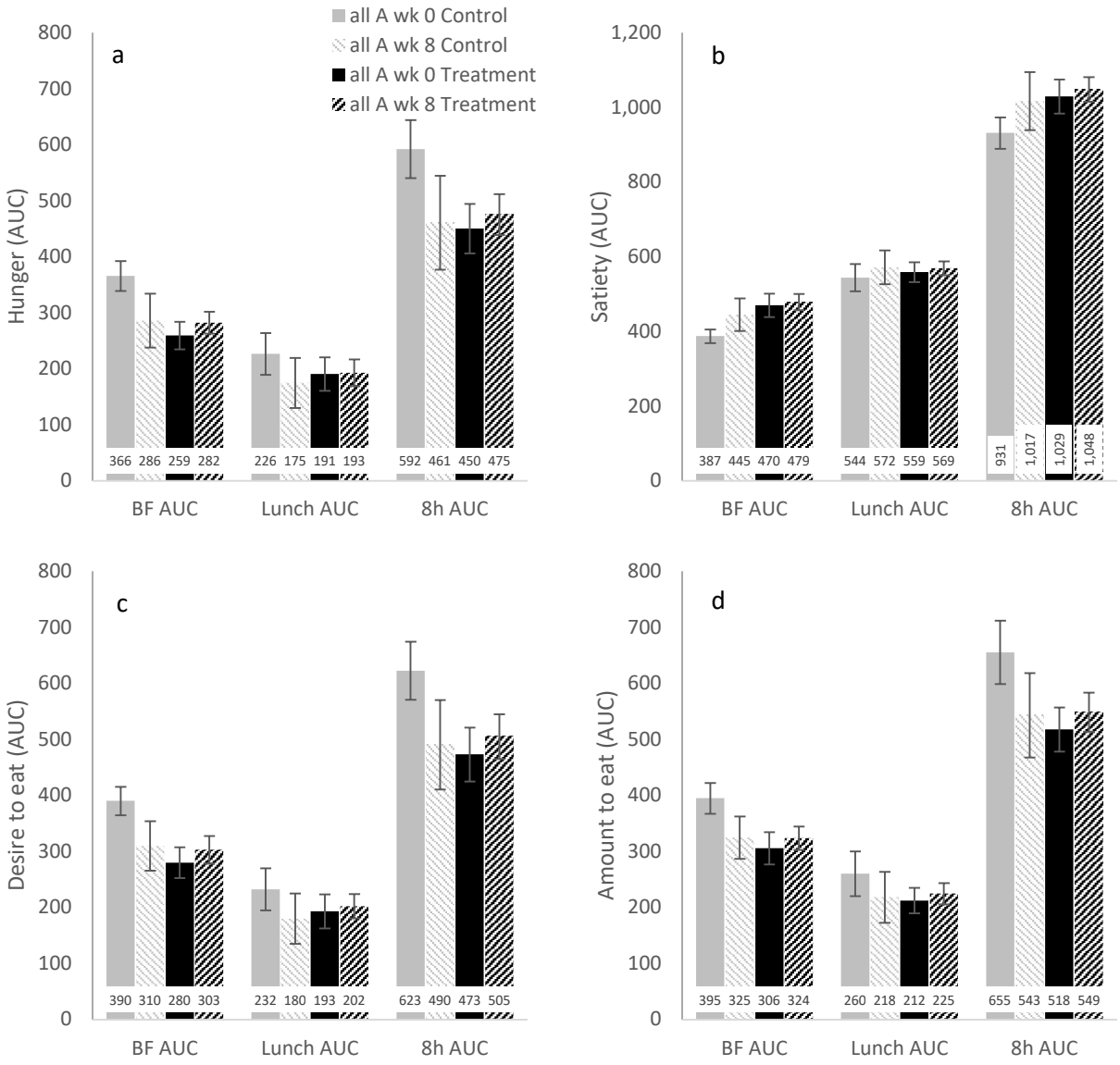

583 **Figure 7.** Area under the curve (AUC) for rated (a) hunger, (b) satiety, (c) desire to eat, and
584 (d) prospective amount to eat for all participants in the GRG, in the control and treatment
585 group before (at week 0) and after (at week 8) interventions as measured with visual analogue
586 scales (VAS) questionnaires. The total measured time (480 min) starts before breakfast (0
587 min) and ends 240 min after lunch. Breakfast was consumed at 0 min and lunch was
588 consumed at 240 min, after completing the VAS questionnaires for that time point.

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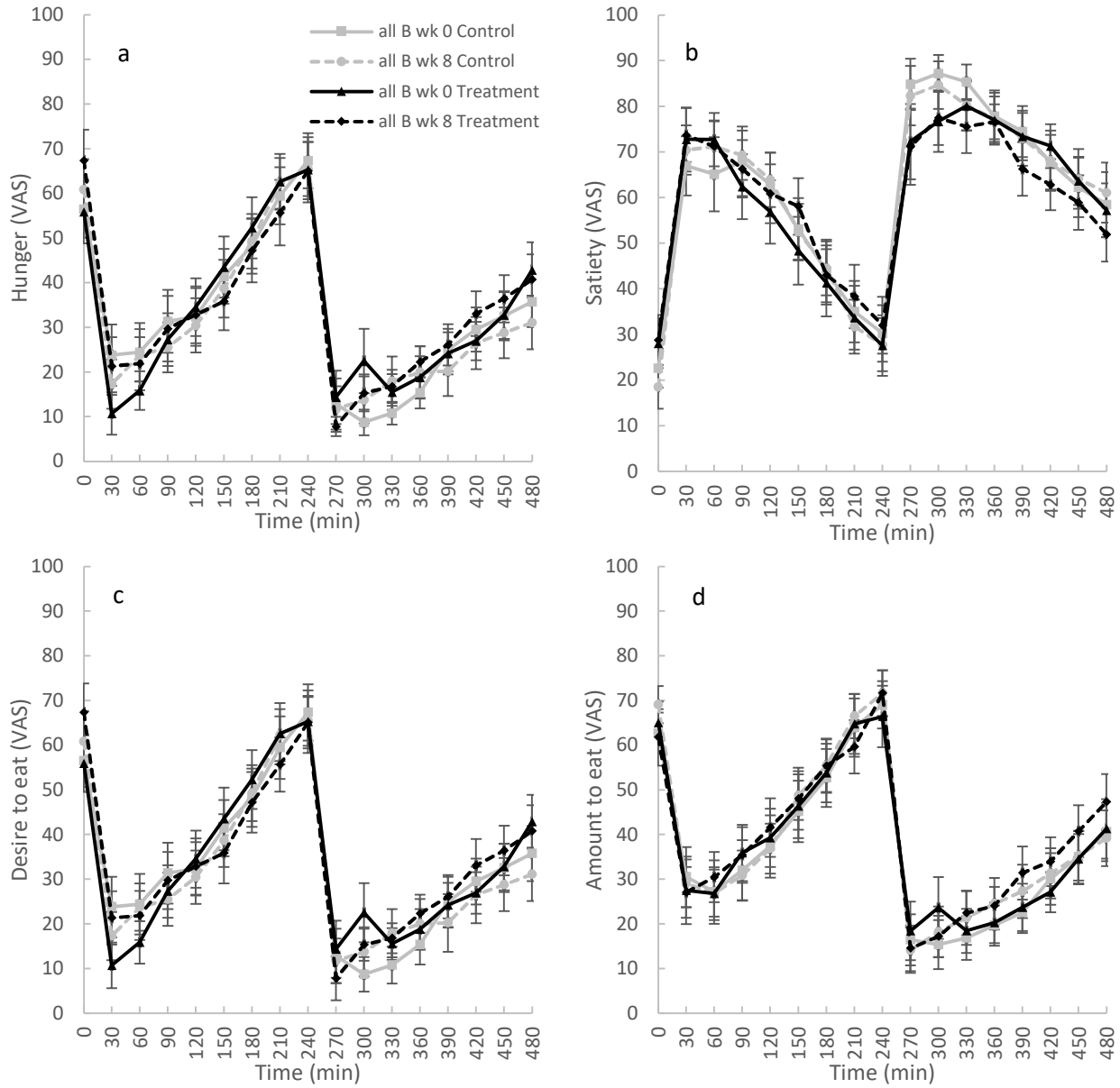

591 **Figure 8.** Changes in rated (a) hunger, (b) satiety, (c) desire to eat, and (d) prospective
592 amount to eat for all subjects in the OG, in the control and treatment group before (at week 0)
593 and after (at week 8) interventions as measured with visual analogue scales (VAS)
594 questionnaires. The total measured time (480 min) starts before breakfast (0 min) and ends
595 240 min after lunch. Breakfast was consumed at 0 min and lunch was consumed at 240 min,
596 after completing the VAS questionnaires for that time point.

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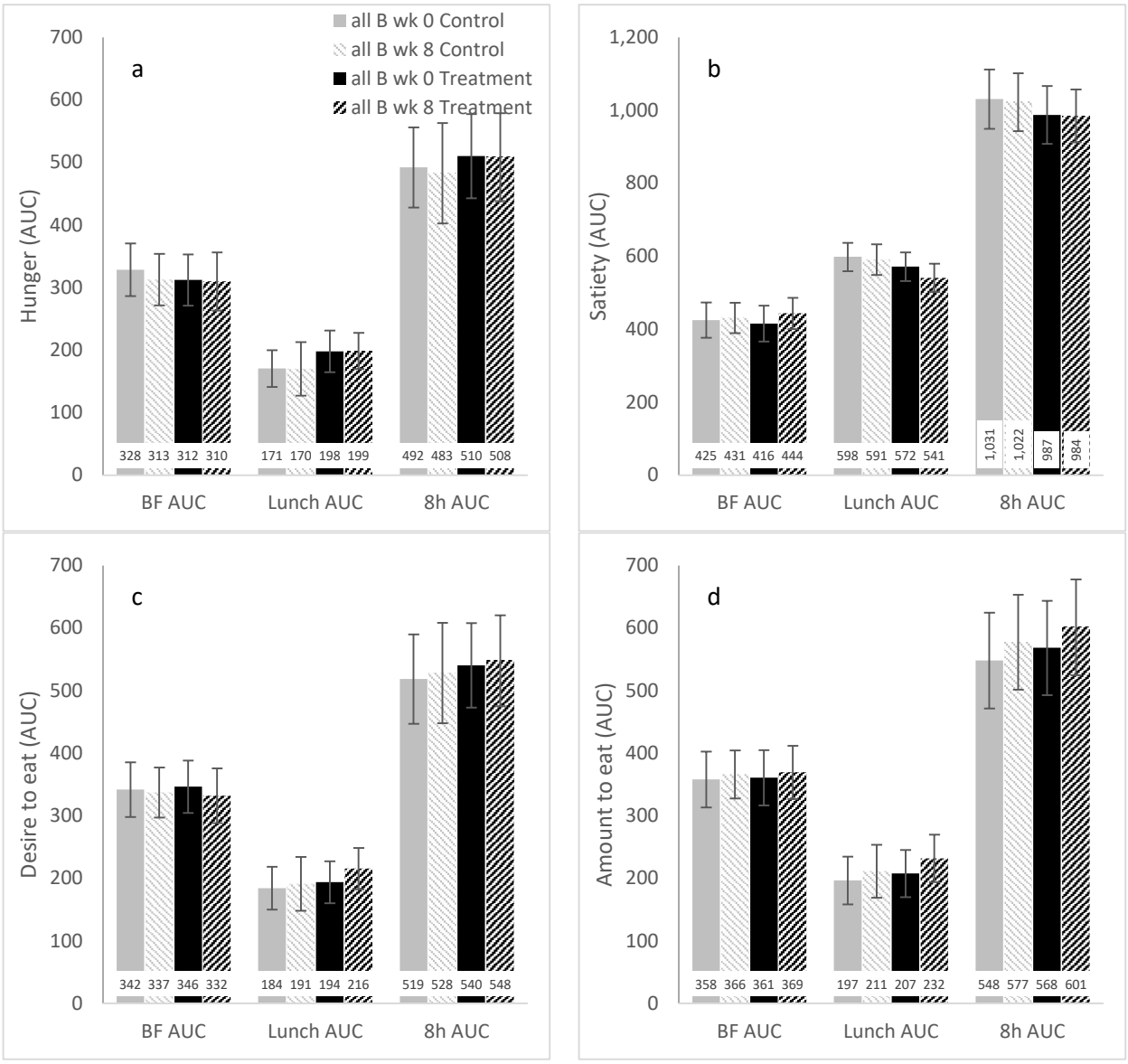

598 **Figure 9.** Area under the curve (AUC) for rated (a) hunger, (b) satiety, (c) desire to eat, and
599 (d) prospective amount to eat for all subjects in the OG, in the control and treatment group
600 before (at week 0) and after (at week 8) interventions as measured with visual analogue scales
601 (VAS) questionnaires. The total measured time (480 min) starts before breakfast (0 min) and
602 ends 240 min after lunch. Breakfast was consumed at 0 min and lunch was consumed at 240
603 min, after completing the VAS questionnaires for that time point.

604

605 Table 1. Baseline characteristics of the study population (n = 78).

	Total (n = 78)			Females (n = 30)			Males (n = 48)		
Variables	T (n = 40)	C (n = 38)	p-value*	T (n = 16)	C (n = 14)	p-value*	T (n = 24)	C (n = 24)	p-value*
Age (y)	39.9 ± 10.6	39.0 ± 11.4	0.726	37.3 ± 7.5	35.4 ± 9.9	0.557	41.5 ± 12.2	41.0 ± 11.9	0.886
Height (cm)	168.0 ± 9.1	167.7 ± 7.7	0.866	160.3 ± 6.4	160.3 ± 5.9	0.984	173.1 ± 6.7	171.9 ± 5.0	0.492
Weight (kg)	70.5 ± 10.4	70.0 ± 8.2	0.812	64.7 ± 6.8	64.8 ± 6.7	0.959	74.4 ± 10.7	73.1 ± 7.5	0.612
BMI (kg/m^2)	25.0 ± 2.5	24.9 ± 2.7	0.988	25.2 ± 2.3	25.3 ± 3.3	0.907	24.8 ± 2.7	24.7 ± 2.4	0.931
WC (cm)	86.1 ± 8.7	85.1 ± 8.7	0.628	80.9 ± 7.2	81.2 ± 8.9	0.922	89.6 ± 7.9	87.4 ± 7.9	0.358
HC (cm)	101.2 ± 6.3	99.9 ± 5.7	0.333	101.5 ± 6.5	100.6 ± 6.8	0.725	101.1 ± 6.3	99.5 ± 5.1	0.343
WHR	0.85 ± 0.08	0.85 ± 0.07	0.990	0.80 ± 0.05	0.81 ± 0.06	0.676	0.89 ± 0.07	0.88 ± 0.06	0.622
PBF (%)**	32.5 ± 6.5	31.6 ± 7.0	0.550	37.9 ± 4.3	38.2 ± 5.6	0.888	28.9 ± 5.1	27.7 ± 4.4	0.406
FM (kg)**	22.4 ± 5.1	21.7 ± 5.4	0.549	24.1 ± 4.0	24.5 ± 5.6	0.831	21.2 ± 5.5	20.0 ± 4.6	0.414
FFM (kg)**	44.4 ± 8.2	44.6 ± 7.1	0.915	37.1 ± 4.3	36.8 ± 2.9	0.856	49.3 ± 6.2	49.2 ± 4.1	0.911
SBP (mmHg)	117 ± 11	119 ± 11	0.570	110 ± 9	112 ± 8	0.462	122 ± 10	122 ± 11	0.928
DBP (mmHg)	76 ± 9	77 ± 8	0.492	74 ± 7	75 ± 7	0.530	78 ± 9	78 ± 9	0.724
FBG (mmol/L)	4.9 ± 0.5	4.8 ± 0.5	0.662	4.8 ± 0.3	4.8 ± 0.3	0.602	4.9 ± 0.6	4.9 ± 0.5	0.770
Insulin (mU/L)***	7.6 ± 5.8	6.9 ± 4.1	0.525	8.5 ± 8.2	7.8 ± 5.1	0.785	7.0 ± 3.5	6.4 ± 3.4	0.527
HOMA-IR	1.7 ± 1.3	1.5 ± 1.0	0.526	1.8 ± 1.7	1.7 ± 1.2	0.834	1.6 ± 0.9	1.4 ± 0.8	0.494
TG (mmol/L)	1.3 ± 0.8	1.3 ± 0.6	0.679	1.1 ± 0.4	1.2 ± 0.5	0.615	1.5 ± 0.9	1.3 ± 0.6	0.420
TC (mmol/L)	5.0 ± 0.9	5.1 ± 1.0	0.856	4.8 ± 0.6	5.1 ± 1.4	0.450	5.2 ± 1.1	5.1 ± 0.8	0.623
HDL-C (mmol/L)	1.4 ± 0.4	1.3 ± 0.3	0.166	1.6 ± 0.4	1.5 ± 0.3	0.308	1.3 ± 0.3	1.2 ± 0.3	0.408
LDL-C (mmol/L)	3.4 ± 0.7	3.5 ± 0.8	0.485	3.1 ± 0.5	3.4 ± 1.1	0.299	3.7 ± 0.8	3.6 ± 0.6	0.885

606 *One-Way ANOVA analysis between treatment (T) and control (C) group. **Measured by DEXA. ***To convert insulin concentrations from
607 mU/L to pmol/L, multiply value by 6.945. BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio;
608 PBF; percent body fat; FM, fat mass; FFM, fat free mass; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood
609 glucose; HOMA-IR, homeostatic model assessment for insulin resistance; TG, triacylglycerol; TC, total cholesterol; HDL-C, high- density
610 lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

611 Table 2. Glycaemic parameters assessed by real-time CGMS for the study population (n = 66) in the control (C) and treatment (T) group at week

612 0. During the 24 h CGMS session, participants consumed their assigned control or treatment meal.

	Total (n = 66)			Females (n = 24)			Males (n = 42)		
Variables	T (n = 33)	C (n = 33)	p-value*	T (n = 14)	C (n = 10)	p-value*	T (n = 19)	C (n = 23)	p-value*
24 h average	5.51 ± 0.68	5.67 ± 0.98	0.460	5.64 ± 0.81	5.51 ± 0.97	0.739	5.43 ± 0.58	5.74 ± 1.00	0.238
24 h iAUC	965 ± 639	1121 ± 751	0.367	985 ± 635	1355 ± 698	0.192	949 ± 659	1019 ± 764	0.756
24 h Max	8.38 ± 1.78	8.70 ± 2.17	0.514	8.82 ± 1.92	9.41 ± 2.47	0.518	8.06 ± 1.65	8.40 ± 2.01	0.561
24 h ΔMax-Min	4.62 ± 1.97	4.83 ± 2.17	0.679	4.95 ± 1.96	5.85 ± 2.61	0.344	4.37 ± 1.99	4.38 ± 1.84	0.981
24 h SD	1.00 ± 0.48	1.11 ± 0.65	0.444	1.16 ± 0.54	1.42 ± 0.67	0.302	0.88 ± 0.41	0.95 ± 0.45	0.580
24 h MAGE	2.81 ± 1.35	3.11 ± 1.64	0.415	3.05 ± 1.43	3.83 ± 1.87	0.260	2.63 ± 1.29	2.80 ± 1.46	0.696
TIR < 4.5 (%)	13.4 ± 16.4	13.3 ± 17.3	0.993	12.1 ± 13.0	20.1 ± 19.8	0.249	14.3 ± 19.0	10.4 ± 15.7	0.473
TIR 4.5 - 7.0 (%)	71.7 ± 18.3	66.3 ± 21.9	0.222	68.1 ± 15.9	57.3 ± 24.0	0.200	75.7 ± 20.7	70.2 ± 20.2	0.384
TIR > 7.0 (%)	14.8 ± 15.2	20.4 ± 21.1	0.173	19.8 ± 18.0	22.6 ± 18.9	0.715	10.0 ± 11.7	19.4 ± 22.3	0.105
Breakfast iAUC	156 ± 113	189 ± 173	0.356	127 ± 106	150 ± 95	0.594	177 ± 139	206 ± 197	0.571
Breakfast Max	6.99 ± 1.26	7.30 ± 1.75	0.413	7.08 ± 1.49	6.92 ± 1.57	0.804	6.92 ± 1.11	7.46 ± 1.83	0.266
Breakfast ΔMax-Min	2.49 ± 1.27	2.78 ± 1.28	0.368	2.55 ± 1.33	2.71 ± 1.45	0.782	2.45 ± 1.26	2.80 ± 1.24	0.362
Breakfast SD	0.80 ± 0.47	0.86 ± 0.41	0.582	0.85 ± 0.55	0.89 ± 0.51	0.851	0.78 ± 0.45	0.86 ± 0.38	0.536
Breakfast Tmax (min)	44.4 ± 18.5	43.3 ± 13.1	0.789	38.2 ± 2.80	37 ± 3.09	0.776	48.9 ± 21.9	46.1 ± 13.6	0.607
Lunch iAUC	275 ± 200	323 ± 249	0.391	282 ± 208	400 ± 242	0.215	269 ± 198	289 ± 250	0.779
Lunch Max	7.63 ± 1.55	7.93 ± 1.96	0.502	7.79 ± 1.73	8.07 ± 2.27	0.731	7.52 ± 1.44	7.87 ± 1.87	0.515
Lunch ΔMax-Min	3.05 ± 1.31	3.29 ± 1.62	0.507	3.05 ± 1.46	3.63 ± 2.27	0.453	3.05 ± 1.23	3.15 ± 1.29	0.809
Lunch SD	0.92 ± 0.44	0.99 ± 0.53	0.592	0.91 ± 0.46	1.19 ± 0.78	0.286	0.91 ± 0.42	0.90 ± 0.39	0.933
Lunch Tmax (min)	63.5 ± 24.3	58.5 ± 18.6	0.351	70.7 ± 6.52	70.5± 6.21	0.982	58.2 ± 23.5	53.3 ± 15.8	0.425
Dinner iAUC	384 ± 240	509 ± 342	0.090	430 ± 240	687 ± 353	**0.045**	350 ± 240	431 ± 313	0.357
Dinner Max	8.13 ± 1.84	8.49 ± 2.19	0.475	8.66 ± 1.92	9.32 ± 2.39	0.464	7.74 ± 1.72	8.13 ± 2.05	0.515
Dinner ΔMax-Min	3.73 ± 1.79	4.04 ± 2.13	0.521	4.20 ± 1.68	5.30 ± 2.48	0.208	3.38 ± 1.83	3.49 ± 1.74	0.840
Dinner SD	1.12 ± 0.58	1.25 ± 0.77	0.433	1.32 ± 0.63	1.75 ± 0.67	0.162	0.97 ± 0.50	1.03 ± 0.65	0.727
Dinner Tmax (min)	107.4 ± 58.9	93.8 ± 54.4	0.332	128.2± 14.02	111.5 ± 10.0	0.381	92.1 ± 59.9	86.1 ± 60.7	0.749

613 *One-Way ANOVA analysis between treatment (T) and control (C) group. iAUC, incremental area under the curve; SD, standard deviation;
614 MAGE, mean amplitude of glycaemic excursion; Tmax, time at which the respective maximum concentration is reached. Breakfast and lunch
615 parameters are measured over 4 h and dinner parameters over 8 h. All parameters are expressed in mmol/L unless otherwise indicated.
616 TIR, time in range, is expressed as a percentage of a 16-h glucose reading.

617

618 Table 3. Glycaemic parameters assessed by real-time CGMS for the study population (n = 66) in the control (C) and treatment (T) group at week

619 8. During the 24 h CGMS session, participants consumed their assigned control or treatment meal.

	Total (n = 66)			Females (n = 24)			Males (n = 42)		
Variables	T (n = 33)	C (n = 33)	p-value*	T (n = 14)	C (n = 10)	p-value*	T (n = 19)	C (n = 23)	p-value*
24 h average	5.54 ± 0.63	5.66 ± 0.88	0.525	5.67 ± 0.85	5.60 ± 0.63	0.827	5.44 ± 0.41	5.68 ± 0.98	0.318
24 h iAUC	874 ± 523	1186 ± 809	0.067	1044 ± 647	1521 ± 868	0.136	748 ± 380	1041 ± 755	0.133
24 h Max	8.06 ± 1.56	8.43 ± 1.74	0.375	8.26 ± 2.01	8.66 ± 1.82	0.627	7.92 ± 1.16	8.33 ± 1.74	0.385
24 h ΔMax-Min	4.01 ± 1.67	4.51 ± 1.78	0.244	4.26 ± 2.08	5.27 ± 2.12	0.256	3.82 ± 1.33	4.17 ± 1.55	0.438
24 h SD	0.83 ± 0.41	1.01 ± 0.49	0.107	0.94 ± 0.55	1.16 ± 0.56	0.344	0.76 ± 0.26	0.95 ± 0.45	0.103
24 h MAGE	2.44 ± 1.23	2.95 ± 1.42	0.119	2.62 ± 1.59	3.27 ± 1.67	0.347	2.30 ± 0.92	2.82 ± 1.31	0.155
TIR < 4.5 (%)	11.0 ± 13.4	14.5 ± 21.4	0.427	10.8 ± 13.1	17.4 ± 20.9	0.349	11.1 ± 13.9	13.2 ± 21.9	0.719
TIR 4.5 - 7.0 (%)	77.4 ± 17.8	66.3 ± 21.0	**0.014**	74.6 ± 19.3	61.2 ± 18.3	0.101	80.8 ± 14.6	68.6 ± 22.1	**0.046**
TIR > 7.0 (%)	11.5 ± 14.0	19.2 ± 18.7	**0.042**	14.6 ± 18.3	21.4 ± 14.2	0.339	8.1 ± 7.8	18.2 ± 20.6	0.050
Breakfast iAUC	146 ± 81	192 ± 125	0.080	156 ± 102	179 ± 125	0.632	139 ± 63	198 ± 127	0.071
Breakfast Max	6.97 ± 1.22	7.11 ± 1.50	0.667	7.09 ± 1.49	6.90 ± 1.55	0.765	6.88 ± 1.07	7.20 ± 1.50	0.433
Breakfast ΔMax-Min	2.35 ± 1.05	2.65 ± 1.30	0.320	2.40 ± 1.26	2.62 ± 1.46	0.697	2.31 ± 1.05	2.66 ± 1.26	0.339
Breakfast SD	0.73 ± 0.37	0.86 ± 0.47	0.225	0.75 ± 0.48	0.88 ± 0.56	0.573	0.71 ± 0.30	0.85 ± 0.45	0.261
Breakfast Tmax (min)	42.6 ± 17.1	50.2 ± 26.0	0.166	41.07 ± 5.11	45.0 ± 5.22	0.605	43.7 ± 15.8	52.4 ± 29.2	0.251
Lunch iAUC	231 ± 163	315 ± 239	0.098	294 ± 209	424 ± 271	0.198	184 ± 101	268 ± 213	0.124
Lunch Max	7.35 ± 1.55	7.76 ± 1.81	0.328	7.44 ± 1.74	7.94 ± 1.93	0.517	7.28 ± 1.44	7.68 ± 1.80	0.439
Lunch ΔMax-Min	2.66 ± 1.45	3.15 ± 1.61	0.202	2.69 ± 56	3.48 ± 1.83	0.269	2.64 ± 1.40	3.00 ± 1.52	0.429
Lunch SD	0.79 ± 0.47	0.92 ± 0.51	0.301	0.83 ± 0.51	1.03 ± 0.63	0.394	0.77 ± 0.42	0.85 ± 0.45	0.552
Lunch Tmax (min)	54.2 ± 21.8	63.8 ± 26.9	0.118	62.14 ± 7.43	81.5 ± 7.49	0.088	48.4 ± 14.1	56.1 ± 24.8	0.239
Dinner iAUC	360 ± 241	509 ± 339	**0.043**	441 ± 292.00	680 ± 335	0.077	300 ± 180	435 ± 319	0.109
Dinner Max	7.68 ± 1.59	8.24 ± 1.78	0.189	8.01 ± 2.21	8.56 ± 1.88	0.528	7.45 ± 0.92	8.10 ± 1.75	0.154
Dinner ΔMax-Min	3.21 ± 1.72	3.73 ± 1.58	0.210	3.46 ± 2.25	4.22 ± 1.95	0.401	3.03 ± 1.23	3.51 ± 1.39	0.242
Dinner SD	0.93 ± 0.57	1.17 ± 0.57	0.082	1.07 ± 0.77	1.37 ± 0.67	0.325	0.82 ± 0.34	1.09 ± 0.51	0.060
Dinner Tmax (min)	98.8 ± 51.1	95.0 ± 51.3	0.765	117.5 ± 12.00	100.0 ± 10.70	0.312	85.0 ± 52.1	92.8 ± 57.8	0.651

*One-Way ANOVA analysis between treatment (T) and control (C) group. iAUC, incremental area under the curve; SD, standard deviation; MAGE, mean amplitude of glycaemic excursion; Tmax, time at which the respective maximum concentration is reached. Breakfast and lunch parameters are measured over 4 h and dinner parameters over 8 h. All parameters are expressed in mmol/L unless otherwise indicated.
TIR, time in range, is expressed as a percentage of a 16-h glucose reading.

Table 4. Glycaemic parameters assessed by CGMS for all participants in different groups, i.e. GRG and OG, in the control and treatment group before the intervention period (at week 0). During the 24 h CGMS session, participants consumed their assigned control or treatment meal.

	GRG (n = 30)			OG (n = 36)		
Variables	T (n = 15)	C (n = 15)	p-value*	T (n = 18)	C (n = 18)	p-value*
24 h average	5.47 ± 0.63	5.36 ± 0.79	0.663	5.55 ± 0.74	5.93 ± 1.08	0.226
24 h iAUC	1168 ± 718	1060 ± 603	0.658	795 ± 526	1171 ± 869	0.125
24 h Max	8.65 ± 1.80	8.26 ± 1.95	0.577	8.16 ± 1.79	9.07 ± 2.33	0.197
24 h ΔMax-Min	5.14 ± 1.96	4.59 ± 2.33	0.487	4.18 ± 1.92	5.03 ± 2.07	0.210
24 h SD	1.09 ± 0.43	1.06 ± 0.61	0.853	0.92 ± 0.52	1.15 ± 0.69	0.270
24 h MAGE	3.14 ± 1.40	2.98 ± 1.74	0.788	2.54 ± 1.28	3.22 ± 1.59	0.163
TIR < 4.5 (%)	18.0 ± 16.9	16.4 ± 20.1	0.823	9.6 ± 15.6	10.8 ± 14.7	0.811
TIR 4.5 - 7.0 (%)	66.3 ± 17.5	66.2 ± 25.4	0.993	77.7 ± 18.9	66.3 ± 19.2	0.084
TIR > 7.0 (%)	15.8 ± 13.9	17.4 ± 20.2	0.803	12.8 ± 16.7	22.9 ± 22.0	0.130
Breakfast iAUC	166 ± 116	161 ± 115	0.901	147 ± 112	213 ± 210	0.250
Breakfast Max	6.85 ± 1.16	6.82 ± 1.30	0.941	7.10 ± 1.37	7.69 ± 2.00	0.305
Breakfast ΔMax-Min	2.60 ± 1.32	2.43 ± 1.16	0.716	2.40 ± 1.26	3.06 ± 1.34	0.137
Breakfast SD	0.85 ± 0.47	0.77 ± 0.41	0.625	0.76 ± 0.49	0.94 ± 0.41	0.247
Breakfast Tmax (min)	41.7 ± 10.3	38.3 ± 11.9	0.419	46.7 ± 23.4	47.5 ± 12.9	0.895
Lunch iAUC	327 ± 241	337 ± 200	0.899	231 ± 151	310 ± 289	0.309
Lunch Max	7.76 ± 1.72	7.73 ± 1.89	0.960	7.53 ± 1.72	8.09 ± 2.06	0.345
Lunch ΔMax-Min	3.40 ± 1.33	3.18 ± 1.79	0.705	2.76 ± 1.26	3.39 ± 1.52	0.185
Lunch SD	1.05 ± 0.46	0.98 ± 0.64	0.762	0.82 ± 0.40	0.99 ± 0.47	0.241
Lunch Tmax (min)	61.7 ± 25.9	52.7 ± 16.1	0.263	65.07 ± 23.6	63.3 ± 19.5	0.819
Dinner iAUC	472 ± 265	485 ± 298	0.899	310 ± 194	528 ± 382	**0.038**
Dinner Max	8.45 ± 1.89	8.07 ± 1.90	0.593	7.87 ± 1.81	8.84 ± 2.40	0.182
Dinner ΔMax-Min	4.25 ± 1.76	3.82 ± 2.14	0.555	3.29 ± 1.74	4.22 ± 2.17	0.166
Dinner SD	1.29 ± 0.53	1.17 ± 0.75	0.624	0.98 ± 0.59	1.32 ± 0.81	0.159
Dinner Tmax (min)	96.3 ± 59.1	93.3 ± 64.0	0.895	116.7 ± 58.7	94.2 ± 46.9	0.213

626 * One-Way ANOVA analysis between treatment (T) and control (C) group. iAUC, incremental area under the curve; SD, standard deviation;
627 MAGE, mean amplitude of glycaemic excursion; TIR, time in range; Tmax, time at which the respective maximum concentration is reached.
628 Breakfast and lunch parameters are measured over 4 h and dinner parameters over 8 h. All parameters are expressed in mmol/L unless otherwise
629 indicated.
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Table 5. Glycaemic parameters assessed by CGMS for all participants in different groups, i.e. GRG and OG, in the control and treatment group after the intervention period (at week 8). During the 24 h CGMS session, participants consumed their assigned control or treatment meal.

	GRG (n = 30)			OG (n = 36)		
Variables	T (n = 15)	C (n = 15)	p-value*	T (n = 18)	C (n = 18)	p-value*
24 h average	5.58 ± 0.46	5.28 ± 0.97	0.296	5.50 ± 0.76	5.97 ± 0.67	0.060
24 h iAUC	968 ± 487	1053 ± 834	0.736	795 ± 552	1298 ± 794	**0.034**
24 h Max	8.41 ± 1.23	7.79 ± 1.83	0.286	7.78 ± 1.77	8.96 ± 1.52	**0.039**
24 h ΔMax-Min	4.38 ± 1.53	4.13 ± 2.01	0.701	3.69 ± 1.77	4.82 ± 1.55	0.050
24 h SD	0.93 ± 0.38	0.87 ± 0.51	0.730	0.75 ± 0.43	1.13 ± 0.44	**0.013**
24 h MAGE	2.82 ± 1.11	2.58 ± 1.54	0.628	2.11 ± 1.27	3.26 ± 1.27	**0.010**
TIR < 4.5 (%)	10.9 ± 15.8	23.4 ± 27.6	0.139	11.1 ± 11.4	7.1 ± 10.0	0.273
TIR 4.5 - 7.0 (%)	76.4 ± 17.6	63.7 ± 25.2	0.120	79.6 ± 16.4	68.5 ± 17.3	0.057
TIR > 7.0 (%)	12.7 ± 9.7	12.9 ± 18.4	0.964	9.3 ± 16.0	24.4 ± 17.8	**0.012**
Breakfast iAUC	159 ± 73	154 ± 104	0.877	135 ± 87	224 ± 135	**0.025**
Breakfast Max	7.28 ± 1.05	6.59 ± 1.42	0.143	6.71 ± 1.32	7.54 ± 1.46	0.080
Breakfast ΔMax-Min	2.67 ± 1.03	2.35 ± 1.28	0.456	2.08 ± 1.16	2.90 ± 1.30	0.055
Breakfast SD	0.81 ± 0.31	0.75 ± 0.47	0.690	0.66 ± 0.42	0.94 ± 0.47	0.062
Breakfast Tmax (min)	43.0 ± 16.9	48.0 ± 31.1	0.589	42.2 ± 17.7	51.9 ± 21.6	0.148
Lunch iAUC	271 ± 138	276 ± 240	0.945	197 ± 178	348 ± 241	**0.040**
Lunch Max	7.84 ± 1.21	7.10 ± 1.74	0.186	6.94 ± 1.71	8.31 ± 1.73	**0.023**
Lunch ΔMax-Min	3.17 ± 1.16	2.66 ± 1.57	0.324	2.24 ± 1.55	3.55 ± 1.56	**0.016**
Lunch SD	0.93 ± 0.40	0.79 ± 0.54	0.410	0.67 ± 0.50	1.02 ± 47	0.037
Lunch Tmax (min)	50.0 ± 12.8	60.0 ± 27.7	0.215	57.8 ± 27.0	66.9 ± 26.5	0.311
Dinner iAUC	406 ± 233	447 ± 355	0.711	321 ± 247	561 ± 325	**0.018**
Dinner Max	7.90 ± 1.22	7.60 ± 2.01	0.625	7.51 ± 1.87	8.77 ± 1.40	**0.028**
Dinner ΔMax-Min	3.44 ± 1.54	3.27 ± 1.83	0.781	3.02 ± 1.88	4.11 ± 2.17	0.050
Dinner SD	1.01 ± 0.55	1.01 ± 0.66	0.987	0.85 ± 0.59	1.31 ± 0.46	0.014
Dinner Tmax (min)	86.0 ± 36.2	103.3 ± 51.9	0.298	109.4 ± 59.8	88.1 ± 51.3	0.257

* One-Way ANOVA analysis between treatment (T) and control (C) group. iAUC, incremental area under the curve; SD, standard deviation; MAGE, mean amplitude of glycaemic excursion; TIR, time in range; Tmax, time at which the respective maximum concentration is reached. Breakfast and lunch parameters are measured over 4 h and dinner parameters over 8 h. All parameters are expressed in mmol/L unless otherwise indicated.

Table 6. Change in body composition and anthropometry characteristics of all participants in the GRG over 8 weeks.

	Total (n = 37)			Females (n = 13)			Males (n = 24)		
Variables	T (n = 19)	C (n = 18)	p-value*	T (n = 7)	C (n = 6)	p-value*	T (n = 12)	C (n = 12)	p-value*
Weight (kg)	0.36 ± 1.06	0.35 ± 1.32	0.973	0.83 ± 1.25	0.43 ± 1.19	0.573	0.09 ± 0.88	0.31 ± 1.43	0.659
BMI (kg/m^2)	0.11 ± 0.37	0.10 ± 0.46	0.939	0.26 ± 0.45	0.13 ± 0.37	0.602	0.03 ± 0.30	0.08 ± 0.51	0.736
WC (cm)	-1.37 ± 4.03	1.43 ± 3.22	**0.026**	-0.13 ± 3.91	2.73 ± 2.11	0.138	-2.10 ± 4.08	0.78 ± 3.56	0.078
HC (cm)	-1.17 ± 5.23	3.53 ± 6.93	**0.025**	-0.74 ± 5.39	5.60 ± 10.90	0.200	-1.42 ± 5.36	2.50 ± 4.09	0.057
WHR	-0.00 ± 0.04	-0.01 ± 0.05	0.540	0.004 ± 0.04	-0.01 ± 0.08	0.631	-0.01 ± 0.04	-0.01 ± 0.03	0.752
PBF (%)**	0.30 ± 0.96	-0.18 ± 1.08	0.163	0.34 ± 1.12	-0.38 ± 1.20	0.282	0.28 ± 0.91	-0.08 ± 1.05	0.393
FM (kg)**	0.28 ± 0.76	-0.02 ± 0.89	0.284	0.47 ± 0.86	-0.08 ± 1.05	0.318	0.17 ± 0.71	0.02 ± 0.85	0.643
FFM (kg)**	0.03 ± 0.80	0.28 ± 0.93	0.392	0.33 ± 0.98	0.55 ± 0.39	0.616	-0.14 ± 0.65	0.14 ± 1.10	0.450
PBF (%)***	-0.61 ± 1.25	-0.08 ± 2.02	0.339	-0.17 ± 0.56	0.77 ± 1.57	0.167	-0.87 ± 1.49	-0.50 ± 2.14	0.631
FM (kg)***	-0.29 ± 1.00	-0.01 ± 1.35	0.472	0.14 ± 0.72	0.62 ± 1.08	0.355	-0.54 ± 1.08	-0.33 ± 1.41	0.673
FFM (kg)***	0.53 ± 0.88	0.21 ± 1.58	0.459	0.48 ± 0.77	0.05 ± 1.01	0.398	0.55 ± 0.97	0.29 ± 1.84	0.674
FMtrunk(kg)***	-0.18 ± 0.56	0.07 ± 0.74	0.240	0.06 ± 0.46	0.38 ± 0.67	0.338	-0.33 ± 0.58	-0.08 ± 0.75	0.377
FFMtrunk(kg)***	0.31 ± 0.59	-0.02 ± 0.82	0.168	0.51 ± 0.42	-0.21 ± 0.47	**0.014**	0.19 ± 0.65	0.07 ± 0.95	0.738
PBFtrunk(%)***	-0.76 ± 1.47	0.19 ± 2.18	0.127	-0.49 ± 0.88	1.30 ± 1.67	**0.031**	-0.93 ± 1.74	-0.37 ± 2.26	0.504
PAFM(%)***	-0.47 ± 1.92	0.51 ± 2.47	0.185	-0.11 ± 1.76	1.25 ± 1.24	0.142	-0.68 ± 2.06	0.13 ± 2.87	0.432
PGFM(%)***	-0.58 ± 1.49	-0.04 ± 2.17	0.382	-0.34 ± 1.16	1.08 ± 2.09	0.149	-0.73 ± 1.69	-0.61 ± 2.06	0.881

*One-Way ANOVA analysis between treatment (T) and control (C) group. **Measured by TANITA; *** Measured by DEXA. BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; PBF; percent body fat; FM, fat mass; FFM, fat free mass; PAFM, percentage android fat mass; PGFM, percentage gynoid fat mass.

Table 7. Change in body composition and anthropometry characteristics of all participants in the OG over 8 weeks.

	Total (n = 41)			Females (n = 17)			Males (n = 24)		
Variables	T (n = 21)	C (n = 20)	p-value*	T (n = 9)	C (n = 8)	p-value*	T (n = 12)	C (n = 12)	p-value*
Weight (kg)	0.57 ± 2.76	0.06 ± 1.30	0.460	0.86 ± 0.94	0.41 ± 1.70	0.509	0.35 ± 3.61	-0.18 ± 0.97	0.632
BMI (kg/m^2)	0.25 ± 0.96	0.06 ± 0.53	0.436	0.33 ± 0.56	0.29 ± 0.70	0.883	0.18 ± 1.20	-0.10 ± 0.34	0.440
WC (cm)	-0.02 ± 5.01	0.80 ± 5.29	0.611	1.83 ± 5.05	1.09 ± 6.26	0.790	-1.42 ± 4.71	0.61 ± 4.82	0.309
HC (cm)	-0.91 ± 4.56	-0.05 ± 4.29	0.536	-3.59 ± 4.20	1.20 ± 4.36	**0.036**	1.09 ± 3.84	-0.88 ± 4.22	0.243
WHR	0.01 ± 0.07	0.01 ± 0.05	0.943	0.05 ± 0.05	0.001 ± 0.04	0.061	-0.02 ± 0.06	0.01 ± 0.06	0.159
PBF (%)**	0.37 ± 1.00	0.15 ± 0.98	0.479	0.73 ± 0.94	0.46 ± 0.93	0.559	0.10 ± 1.00	-0.06 ± 1.00	0.702
FM (kg)**	0.45 ± 0.96	0.15 ± 0.98	0.324	0.88 ± 1.01	0.53 ± 1.13	0.506	0.13 ± 0.83	-0.11 ± 0.82	0.494
FFM (kg)**	0.03 ± 0.76	-0.11 ± 0.89	0.593	0.07 ± 0.37	-0.09 ± 0.62	0.538	0.01 ± 0.97	-0.12 ± 1.05	0.765
PBF (%)***	-0.38 ± 1.49	-0.02 ± 0.73	0.335	-0.76 ± 1.82	-0.15 ± 0.77	0.396	-0.10 ± 1.20	0.07 ± 0.73	0.685
FM (kg)***	-0.03 ± 1.19	0.06 ± 0.61	0.769	-0.12 ± 1.40	0.08 ± 0.61	0.718	0.03 ± 1.07	0.04 ± 0.64	0.981
FFM (kg)***	0.53 ± 0.98	0.05 ± 1.17	0.168	0.93 ± 0.98	0.27 ± 1.47	0.287	0.23 ± 0.91	-0.09 ± 0.97	0.421
FMtrunk(kg)***	-0.00 ± 0.73	0.06 ± 0.35	0.729	-0.09 ± 0.78	0.15 ± 0.33	0.444	0.06 ± 0.71	0.00 ± 0.37	0.799
FFMtrunk(kg)***	0.35 ± 0.64	0.02 ± 0.84	0.159	0.49 ± 0.55	0.08 ± 1.10	0.346	0.25 ± 0.71	-0.03 ± 0.66	0.334
PBFtrunk(%)***	-0.47 ± 1.88	0.05 ± 1.04	0.287	-0.90 ± 2.18	0.04 ± 1.35	0.311	-0.15 ± 1.65	0.05 ± 0.83	0.712
PAFM(%)***	-0.54 ± 2.18	0.08 ± 1.29	0.280	-1.14 ± 2.36	0.03 ± 1.76	0.270	-0.08 ± 2.01	0.12 ± 0.96	0.759
PGFM(%)***	0.05 ± 1.17	-0.09 ± 1.28	0.722	-0.43 ± 1.29	-0.46 ± 1.41	0.965	0.42 ± 0.98	0.17 ± 1.19	0.579

*One-Way ANOVA analysis between treatment (T) and control (C) group. **Measured by TANITA; *** Measured by DEXA. BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; PBF; percent body fat; FM, fat mass; FFM, fat free mass; PAFM, percentage android fat mass; PGFM, percentage gynoid fat mass.