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Comparing the effects of time-restricted eating on glycaemic control in people with type 2 diabetes with standard dietetic practice: A randomised controlled trial

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ABSTRACT

Aims: To test the efficacy of time-restricted eating (TRE) in comparison to dietitian-led individualised dietary guidance to improve HbA1c in people with Type 2 diabetes mellitus.
Methods: In a parallel groups design, 51 adults (35–65 y) with Type 2 diabetes mellitus and overweight/obesity (HbA1c ≥6.5% (48 mmol/mol), BMI ≥25-≤40 kg/m^2) commenced a six-month intervention. Following baseline, participants were randomised to TRE (1000–1900 h) or DIET (individualised dietetic guidance) with four consultations over four months. Changes in HbA1c (primary), body composition, and self-reported adherence (secondary) were analysed using linear mixed models. A non-inferiority margin of 0.3% (4 mmol/mol) HbA1c was set *a priori*.
Results: Forty-three participants (56 ± 8 y, BMI: 33 ± 5 kg/m^2, HbA1c: 7.6 ± 0.8%) completed the intervention. HbA1c was reduced (P=0.002; TRE: −0.4% (−5 mmol/mol), DIET: −0.3% (−4 mmol/mol)) with no group or interaction effects; TRE was non-inferior to DIET (−0.11%, 95%CI: −0.50% to 0.28%). Body mass reduced in both groups (TRE: −1.7 kg; DIET: −1.2 kg) via ~900 kJ/d spontaneous energy reduction (P<0.001). Self-reported adherence was higher in TRE versus DIET (P<0.001).
Conclusions: When individualised dietary guidance is not available, effective, and/or suitable, TRE may be an alternative dietary strategy to improve glycaemic control in people with Type 2 diabetes mellitus.

Abbreviations: APD, Accredited Practising Dietitian; CGM, Continuous glucose monitor; CHO, Carbohydrate; DIET, Individualised dietary guidance intervention group; DBP, Diastolic blood pressure; DXA, Dual-energy x-ray absorptiometry; EAT-26, Eating Attitudes Test-26; EO, Eating occasions; GMI, Glucose management indicator; HR, Heart rate; ITT, Intent to treat; MEQ-SA, Morning-Eveningness Questionnaire Self-Assessment; LMM, Linear effects mixed model; PP, Per protocol; REDCap, Research Electronic Data Capture; RFD, Research Food Diary app; SBP, Systolic blood pressure; TAR, Time above range; TIR, Time in range; TITR, Time in tight range; TRE, Time-restricted eating; VAT, Visceral adipose tissue.

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1. Introduction

Dietary modification is a first-line strategy for the management of Type 2 diabetes mellitus in isolation or, most commonly, in conjunction with pharmacotherapy. Intensive dietetic support (i.e., once weekly for 12 weeks) is effective in reducing HbA1c by 1% [1] and improving clinical outcomes [2] in people with Type 2 diabetes mellitus. However, healthcare systems do not offer this level of support for people living with Type 2 diabetes mellitus; whereby individuals in Australia can obtain a General Practitioner Management Plan allowing access to five subsidised Allied Health sessions per year [3]. Individualised dietary counselling from an Accredited Practising Dietitian (APD) in Australia is considered standard practice for those with Type 2 diabetes mellitus [4]. Typically, a patient will meet with an APD following initial diagnosis, and then for a second follow-up session [5]. Dietetic sessions are a cost-effective [6] way to improve health outcomes for people living with Type 2 diabetes mellitus. However, there is a large proportion of people with Type 2 diabetes mellitus who face barriers to engaging with dietetic services [7] or adhering to prescribed dietary guidance [8,9].

The focus of dietary modification is reducing energy intake and improving diet quality, which contribute to better glycaemic management [10,11]. However, dietary choices and patterns are influenced by a combination of biological, psychological, social, cultural, economic, and environmental factors [9,12]. Collectively, these factors can create barriers to adhering to dietary recommendations regarding *what* and *how* much to eat. Therefore, changing dietary patterns, by centring on *when* to eat, has been the focus of targeted weight loss in people with overweight or obesity [13]. Specifically, time-restricted eating (TRE), where energy intake is reduced to 6–10 daylight hours, is effective in reducing glucose AUC [14,15], and improving insulin sensitivity and beta-cell function [16] independent of changes to body mass in those with prediabetes or overweight/obesity. Although TRE is more effective than no intervention for weight loss, the weight change obtained through TRE is less than what would be considered clinically significant [17].

Prior studies exploring the effects of TRE in people with Type 2 diabetes mellitus have reported improvements to HbA1c [18,19], reduced fasting glucose concentrations [20,21] or increase to glucose time in range [19,21,22], with minimal (<5%) changes to body mass [18,19]. However, these studies have either been of short duration (i.e. 2–4 weeks) [20–22] or have involved frequent (i.e., weekly-fortnightly) researcher and participant interaction [18,19]. Whether the same improvements in HbA1c are possible for people with Type 2 diabetes mellitus within the current healthcare provisions in Australia, or how they compare to individualised dietary guidance from a dietitian, is unknown. Accordingly, the aim of this study was to test the efficacy of TRE in comparison to "gold-standard" dietitian-led individualised dietary guidance to improve HbA1c in people with Type 2 diabetes mellitus. The hypothesis was that TRE would be as efficacious as dietetic counselling for improving glycaemic management over six months in people with Type 2 diabetes mellitus.

2. Subjects, materials and methods

2.1. Study design

A randomised, parallel group controlled clinical trial was conducted at Australian Catholic University's (ACU) Melbourne campus between April 2021 and September 2022. Ethical approval (ACU Human Research Ethics Committee; 2019-359H, March 2020) and prospective registration (Australia New Zealand Clinical Trial Registry, ACTRN12620000453987) were obtained. The study was conducted in accordance with the Declaration of Helsinki and all participants provided written informed consent prior to participation.

2.2. Participants

People with Type 2 diabetes mellitus (aged 35–65 y, BMI ≥25-≤45 kg/m^2) were recruited and initially screened via Research Electronic Data Capture [REDCap, Vanderbilt University, USA, 23,24]. Recruitment was conducted using targeted campaigns of flyers/posters, social media advertisements, databases from previous studies and via emails from the National Diabetes Service Scheme. Respondents were eligible if: diagnosed (by a GP/endocrinologist) with Type 2 diabetes mellitus, an HbA1c of ≥6.5–≤10%, taking a ≤2 oral hypoglycaemic agents (excluding sulphonylureas, insulin, and GLP-1 agonists), and habitually self-reported food/drink consumption for ≥12 h during a 24-h window (i.e., on five of seven d/week). The following exclusion criteria were used: not weight stable (>5 kg change over last 3 months); changed medications within 3 months; current smoker (tobacco, nicotine or marijuana) or within 3 months of quitting; women who were pregnant, breastfeeding (within 24 weeks); history of psychotic disorder, or current diagnosis of other major psychiatric illness (e.g. mood disorder, eating disorder, substance use disorder); diagnosed gastrointestinal conditions; unable to adequately complete dietary monitoring or habitual monitoring period.

Potentially-eligible participants were invited to provide informed consent (via REDCap) and complete Morning-Eveningness Questionnaire Self-Assessment (MEQ-SA) [25,26] and Eating Attitudes Test-26 [EAT-26; 27] questionnaires, to identify circadian preferences and screen for disordered eating tendencies (i.e. scores >20 excluded), respectively. Following consent, eligible participants attended the laboratory for a confirmatory HbA1c measurement (Cobas b 101, Roche Diagnostics Ltd., Basel, Switzerland).

2.3. Measurement visits and procedures

Fig. 1 illustrates the measurements and associated timepoints. Participants were instructed to record their habitual diet via the Research Food Diary app (RFD; Xyris Software, Brisbane, Australia) with photos of eating occasions [28] for a two-week baseline period. At this baseline visit, blinded devices were fitted including a continuous glucose monitor (CGM; FreeStyle Libre Pro, Abbott Diabetes Care, CA, USA) and inclinometer (ActivPAL^{TM}, PAL-technologies Ltd., Glasgow, Scotland). A subset of participants wore an ActiWatch (Spectrum, Phillips Respironics, Bend, USA) to capture sleep variables. A handbook was utilised to record sleep, report inclinometer non-wear, and for written food diaries, where required. Participants were instructed to take their prescribed medications as per usual at consistent times throughout the trial including on testing days.

Following the two-week baseline, participants attended the laboratory after an overnight fast (>10 h) and body composition was measured (DXA; GE Lunar iDXA Pro, enCORE software Version 18). Participants then had a fasting blood sample drawn (5 mL serum, 6 mL EDTA), monitors removed, and their baseline dietary intake and food photos discussed with a researcher for accuracy.

After the baseline metabolic visit, participants were randomly allocated to time-restricted eating (TRE) or individualised dietetic practice (DIET), by the study dietitian using REDCap, in a 1:1 ratio using block randomisation, stratified by sex (male/female) and baseline HbA1c (i.e. ≥6.5–≤8% (≥48–≤64 mmol/mol), >8–≤10% (>64–≤86 mmol/mol)). Participants then completed their first TRE or DIET consultation (1 h). Due to the nature of the study, the dietitian and participants were not blinded to condition, but all other study staff were.

Participants attended further laboratory visits in the two weeks prior to each consultation to be fitted with a new CGM sensor and ActivPAL monitor and asked to record all dietary intake using RFD and food photos throughout these periods. On days of consultations (one, two, and four months; 30 min each) as well as at the end of the six-month intervention, participants attended the laboratory in a fasted state for blood sampling and to retrieve the devices, RFD recording and food

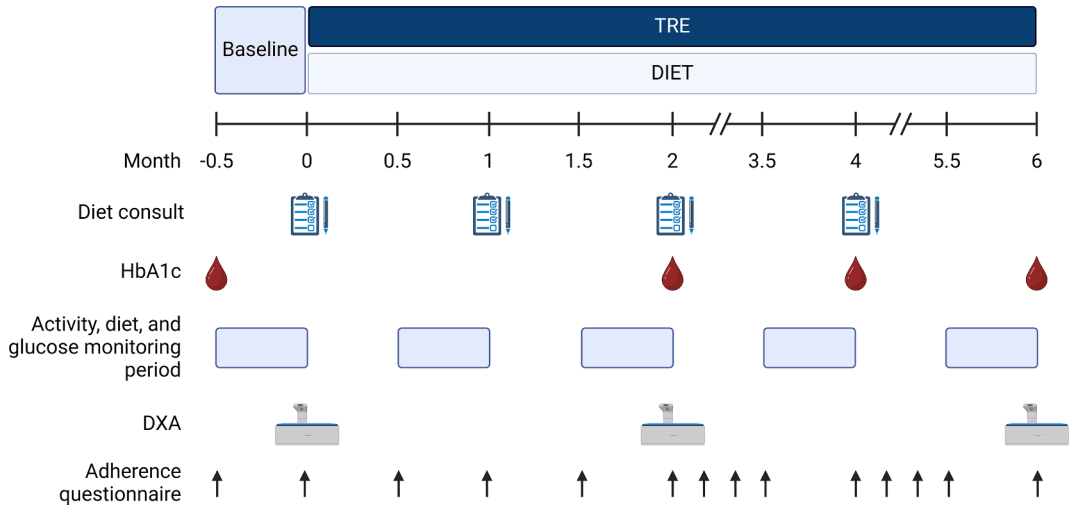

Fig. 1. Overview of the study design where individuals with type 2 diabetes completed a 2-week baseline monitoring period and were then subsequently randomised to one of two intervention conditions to follow for 6 months. Both groups received guidance via four diet consultations relative to the intervention group. HbA1c, as the primary outcome, was measured every 2 months, with five 2-week periods of monitoring (diet, activity, glucose), three body composition scans (0, 2 and 6 months) and self-reported adherence questionnaires every 2 weeks. Key: DIET, individualised dietary advice from an Accredited Practising Dietitian; DXA, dual x-ray absorptiometry scan; TRE, time-restricted eating (1000–1900 h). Figure created with BioRender.com.

photos. At the two- and six-month visits, participants had further DXA scans.

2.4. Intervention groups and consultations

Individuals randomised to TRE were asked to modify their eating times to between 1000 h and 1900 h (as per [28]) for as many days as possible during the six-month intervention. Individuals randomised to DIET were provided with publicly available nutrition guidance for individuals with Type 2 diabetes mellitus (i.e., Baker Heart and Diabetes Institute resources) along with a discussion about changes that they could make to their dietary intake. The four TRE consults included topics of defining TRE, what is permitted during the eating and fasting windows, strategies to help adhere to TRE, navigating social situations, and strategies to mitigate hunger while fasting. Explicitly, no dietary advice was provided to the TRE group. The four DIET consults included topics of diet quality and quantity, carbohydrate, label reading, and alcohol consumption, implemented in the order that was relevant to participant requirements as determined by the dietitian. Explicitly, no eating window advice was provided to the DIET group. Information in the consultations for both the TRE and DIET groups were delivered by the same APD, in a time-matched manner. Prior to each consult, where possible, participants dietary intake records were utilised (i.e., timing information in the TRE consults and food type information in the DIET consults).

Self-reported adherence to each intervention was captured using a REDCap question "*Thinking about the past two weeks, how frequently have you applied the regime above?*" every two weeks. Participants were asked to select "*never*", "*rarely (1–2 days/wk)*", "*sometimes (3–4 days/wk)*", "*most of the time (5–6 days)*" or "*everyday*". The fortnightly questionnaire also prompted participants to self-report any changes in medication and/or health status.

2.5. Dietary recordings and analysis

Participants recorded dietary intake via two-weekly dietary recordings (Fig. 1) using the RFD app or, if required, via a paper-based handbook and were asked to photograph each eating/drinking occasion using their mobile device during the two-weekly recording periods [28]. All available days of dietary recording were assessed and categorised systematically as "Complete", "Partially complete" or "Incomplete" (Figure S1) for energy intake to identify implausible reporting, and as "Valid" or "Not valid" with regards to the eating window (see Supplementary Material). Only "Complete" and/or "Valid" days were included in the analysis.

2.6. Data analysis

ActivPAL files were exported in 1-minute epochs using PAL Batch software (PAL Technologies Ltd, Glasgow, Scotland). The R programming language (Version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria) was used to extract mean data per participant per timepoint for further analysis. For the interstitial glucose analysis, the first and last day of monitor wear were removed due to being incomplete days and therefore a maximum of 12 days were available per timepoint. Measures of mean glucose, total area under the curve (trapezoid method), fasting proxy (i.e. lowest glucose concentration between 0000–0600 h), nocturnal glucose concentration (i.e. average between 0000–0600 h), time in range (TIR; 3.9–10.0 mmol/L), time in tight range (TITR; 3.9–7.8 mmol/L), time above range (TAR; >10 mmol/L), and glucose managment indicator (GMI) [29] were calculated.

To be included in the analysis, at least four valid days of dietary intake (based on [30]), eating window, interstitial glucose, physical activity and sleep data were required (Table S1). A valid day of CGM data was 24-h from midnight to midnight, and a valid day of ActivPAL data was 24-h wear time from midnight to midnight. Time in bed data was generated from the activity files and were manually checked against the self-reported diary for validity. When assessing adherence to TRE from the meal timing, adherent was considered with eating occasions within the 1) start time was >0945 h, 2) end time was <1915 h, 3) start and end time if eating occasions were from 0945 to 1915 h (inclusive), and 4) eating window duration if <9.5 h.

2.7. Blood processing and biochemical analysis

HbA1c was measured using venous whole blood samples (EDTA) with a Cobas b 101 instrument (Roche Diagnostics Ltd., Basel, Switzerland). Subsequently, a lipid panel (total, LDL and HDL cholesterol, triglycerides) was also run using whole blood. The remaining samples were spun at 1800 *g* for 10 min at 4 °C to obtain plasma samples which were frozen at −80 °C for later analysis. Serum samples were left at room temperature for 30 min before being spun as per the plasma samples. Glucose, insulin, and non-esterified fatty acid (NEFA)

concentrations were measured in duplicate from thawed plasma samples using YSI 2900 analyzer (YSI Life Sciences, Yellow Springs, OH, USA; mean coefficient of variation (CV) 1.9%), a commercially available ELISA kit (Alpco Ltd, Windham, New Hampshire, USA; CV 3.2%), and an enzymatic colorimetric method assay (Wako Pure Chemical Industries, Ltd., Osaka, Japan; CV 5.5%).

2.8. Sample size and statistical analysis

Without a similar study to compare to, we hypothesised that there would be no significant difference between groups and therefore the non-inferiority calculation (80% power, 0.05 alpha, effect size of 0.50 with a group standard deviation of 0.65) estimated 42 participants (n=21 per group) would be required. To account for ~20 % dropout, we intended to recruit n=52 participants.

Statistical analyses were performed on study completion, once all investigators were unblinded to condition, using the R programming language (Version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria). The change in HbA1c between 0 and 6 months was chosen as the primary outcome as it is the most relevant clinical outcome for individuals with type 2 diabetes and their clinicians. A 0.3% difference in HbA1c is considered a clinically relevant margin to reduce long-term complications of type 2 diabetes [8,9]. For the main outcome variable of HbA1c, sex and BMI at baseline were used as covariates in the model. The prespecified primary outcome was the change in HbA1c between baseline and six months, with intention to treat (ITT) analysis using the last observation carried forward. Non-inferiority was assessed using the mean change from baseline to six months between groups using 95% confidence intervals of the estimate change [31], with a 0.3% non-inferiority margin. For all continuous outcome variables, linear mixed effects models (LMM; *lmer* function from *lmerTest* package) were used, whereas for categorical outcome variable (Adherence), cumulative link mixed model with a logit link function was used with the *clmm* function from the *ordinal* package. Both intent to treat (ITT) and per protocol (PP) outcomes for HbA1c were assessed between groups using baseline and 6 month data. As a secondary outcome, HbA1c was assessed with factors of group and time, using all measurement timepoints. All models were built with a group by time interaction as fixed effects and participant ID included as a random intercept term. The residuals of each model were visually inspected to ensure they were somewhat normally distributed [10]. Where significant main effects were found from the linear mixed models, using the *anova* function, post-hoc tests were performed using *lsmeans* from the *emmeans* package with Tukey adjusted p-values for multiple comparisons. Model estimates and their 95% confidence intervals are reported. Differences in participant characteristics at baseline were assessed using independent t-tests for continuous, normally distributed variables, for those variables that violated these assumptions (ethnicity, type of medication used), Fisher's exact tests were used.

Secondary outcomes were all analysed per protocol (PP) using LMMs, including 0–6 month change in HbA1c and all timepoints of HbA1c, and measures of body composition, dietary intake and timing, CGM metrics, blood metabolites and hormones. All the aforementioned secondary outcomes have been reported here (or in the Supplementary Material) to ensure a holistic overview of this intervention. All data are reported as the mean ± SD unless stated otherwise, significance was set to $P<0.05$ *a priori*.

3. Results

With one participant withdrawing, 51 participants were randomised (26 to TRE and 25 to DIET [Figure S2]). Twenty-two participants completed the 6-month TRE intervention and 21 completed the DIET intervention. The DIET group were older, had a greater body mass and BMI at baseline than the TRE group (Table 1). There was a significant difference in ethnicity, with a greater proportion of those identifying as Caucasian (62%) in DIET compared to Asian (50%) in TRE. Considering ethnicity, there were no differences in ethnicity-specific BMI categories between groups.

Table 1
Baseline demographics and clinical characteristics of people with type 2 diabetes mellitus who began (all randomised), completed (all completers) and those who completed each of the individualised dietary counselling (DIET) and time-restricted eating (TRE) the 6-month intervention. Data are mean ± SD.

	All randomised (n = 51)	All completers (n = 43)	DIET group (n = 21)	TRE group (n = 22)	*p*-value
Age, y	55.9 ± 8.1	55.6 ± 8.4	58.0 ± 6.9	53.4 ± 9.2	0.03
Sex, n (%)					
Female	21 (41%)	17 (40%)	7 (33%)	10 (45%)	1.00
Male	30 (59%)	26 (60%)	14 (66%)	12 (55%)	
Height, cm	170 ± 9	170 ± 9	172 ± 10	168 ± 8	0.39
BMI, kg/m^2	33.0 ± 4.7	32.5 ± 4.5	33.9 ± 4.4	31.1 ± 4.1	0.004
Ethnicity, n (%)					
Caucasian	23 (45%)	17 (39%)	13 (62%)	4 (18%)	0.001
European	12 (24%)	11 (26%)	5 (24%)	6 (27%)	
Asian	15 (29%)	14 (33%)	3 (14%)	11 (50%)	
Hispanic/ Latino	1 (2%)	1 (2%)	0	1 (5%)	
Ethnicity-specific BMI categories*					
Overweight	9 (18%)	8 (19%)	5 (24%)	3 (14%)	0.72
Obese	42 (82%)	35 (81%)	16 (76%)	19 (86%)	
Body mass (scales), kg	95.4 ± 16.2	93.9 ± 16.4	100.2 ± 17.4	87.9 ± 13.0	0.003
MEQSA	58 ± 9	58 ± 9	59 ± 9	58 ± 9	0.70
EAT-26	6 ± 5	6 ± 5	5 ± 5	8 ± 5	0.02
Duration of diabetes, y	6 ± 4	6 ± 4	6 ± 4	7 ± 4	0.39
Medication, n (%)					
None	5 (10%)	4 (9%)	2 (9%)	2 (9%)	0.64
1 OHA	29 (57%)	25 (58%)	14 (67%)	11 (50%)	
2 OHAs	17 (33%)	14 (33%)	5 (24%)	9 (41%)	
Medication type, n (%)					
Biguanides	45 (88%)	38 (88%)	18 (86%)	20 (91%)	1.00
SGLT2i	10 (20%)	8 (19%)	2 (10%)	6 (27%)	0.29
DPP4i	8 (16%)	7 (16%)	4 (19%)	3 (14%)	0.47
HbA1c, % (mmol/mol)	7.6 ± 0.8 (60 ± 8)	7.6 ± 0.8 (59 ± 8)	7.5 ± 0.8 (59 ± 8)	7.6 ± 0.8 (60 ± 8)	0.97

Key: DIET, dietary modification group; DPP4i, dipeptidyl peptidase IV inhibitors; EAT-26, eating attitude test; HbA1c, glycated haemoglobin; MEQSA, morningness-eveningness questionnaire self-assessment; OHA, oral hypoglycaemic agents; SGLT2, sodium-glucose cotransporter-2 inhibitors; TRE, time restricted eating group. Baseline differences between groups were conducted using independent samples *t*-test for parametric values and Fishers Exact test for non-parametric values. *for Caucasian, European and Hispanic/Latino, BMI 25–29.9 kg/m^2 = overweight, BMI >30 kg/m^2 = obese; for Asian, BMI >25 kg/m^2 = obese.

There was a significant (P=0.003) decrease in HbA1c between baseline and six months using ITT with a −0.3% (−4 mmol/mol) reduction (95%CI: −0.5 to −0.1% (−6 to −1 mmol/mol)) but no group or interaction effects (DIET: baseline: 7.5% ± 0.7% (59 ± 8 mmol/mol), six months: 7.2 ± 1.0% (56 ± 11 mmol/mol); TRE: baseline: 7.5 % ± 0.8% (58 ± 8 mmol/mol), six months: 7.2 ± 0.8% (54 ± 8 mmol/mol); Table S2). The difference in the change in HbA1c between groups was

−0.11%, (95%CI: −0.50 to 0.28%) in favour of TRE; as the upper bound did not cross the pre-defined non-inferiority margin of 0.3%, TRE is non-inferior to DIET. The secondary PP analysis followed a similar trend with a −0.4% (−5 mmol/mol) reduction in HbA1c (P=0.001; 95%CI: −0.6 to −0.2% (−7 to −2 mmol/mol). All subsequent analyses were conducted PP. Fig. 2A presents the PP HbA1c data measured over time which was not different between groups but reduced between baseline and two- (−0.5%; 95%CI: −0.7 to −0.3%; P<0.001), four- (−0.4%; 95%CI: −0.6 to −0.2%; P<0.001), and six-months (−0.4%; 95%CI: −0.6 to −0.1%; P<0.001). Fasting glucose and insulin concentrations, and HOMA-IR, were reduced over time (Fig. 2B-D), with no differences between groups in insulin concentrations and HOMA-IR. However, post-hoc tests showed no significant changes in [insulin] or HOMA-IR, and fasting [glucose] was lower at two months compared to baseline (P=0.002).

The CGM results over time for each group are presented in Fig. 2E-K. At least 85% of participants had valid data included for analysis (Table S1). There was nominal time spent in the hypoglycaemic range (<3.9 mmol/L), which was similar across time and group [*data not shown*]. Mean glucose, AUC, TAR, and GMI were all reduced over time, while TIR and TITR were increased over time, when measured over 24-h, day and night periods (Table S3), with interaction effects for mean glucose, AUC, TAR, GMI and TIR (Fig. 2E-K). Post-hoc tests revealed significant differences between baseline and months one, two and six for reduced total AUC (−0.7 mmol/L/h, P<0.02), mean glucose (−0.8 mmol/L, P<0.01), TAR (−7%, P<0.02), and GMI (−4 mmol/mol, P<0.002) variables and the increased TIR (+8%, P<0.05) and TITR (+10%, P<0.02). The fasting proxy, measured between midnight and 0600 h, was reduced over time, with no difference between groups or interaction. There was significantly greater variability in blood glucose in the TRE group as measured by SD of glucose with a group × time interaction (Table S3). There were no changes in CV, through 24-h, day, or night periods (Table S3).

Measurements of body composition and total body mass (BM) are presented in Fig. 3A-F. The spread of BM change over six months is displayed in Figure S3. Main effects of group were evident in total body mass (P=0.012), BMI (P=0.006) and VAT (P=0.012), with trends for differences between groups for lean mass, fat mass and body fat percentage (all P=0.06), where the DIET group were greater than the TRE group for all variables (Fig. 3A-F). A main effect of time was measured for BM (Fig. 3A), where the TRE group had a reduced BM after the intervention (−1.7 kg, P=0.01) with a smaller reduction in DIET (−1.2 kg, P=0.14). However, both groups had a reduction in BM between baseline and two months (TRE: −1.7 kg, P=0.01; DIET: −1.5 kg, P=0.04). BMI also decreased over time, with a main effect of group (Fig. 3D). Total fat mass reduced over time, with no between group difference (Fig. 3C). Specifically, there was a fat mass reduction in the TRE group between baseline, two (−1.2 kg, P=0.05) and six months (−1.4 kg, P=0.01). In DIET, fat mass was significantly reduced from baseline to two months (−1.4 kg, P=0.017) with no significant reduction over the six months (−1.2 kg, P=0.07). There were no changes in

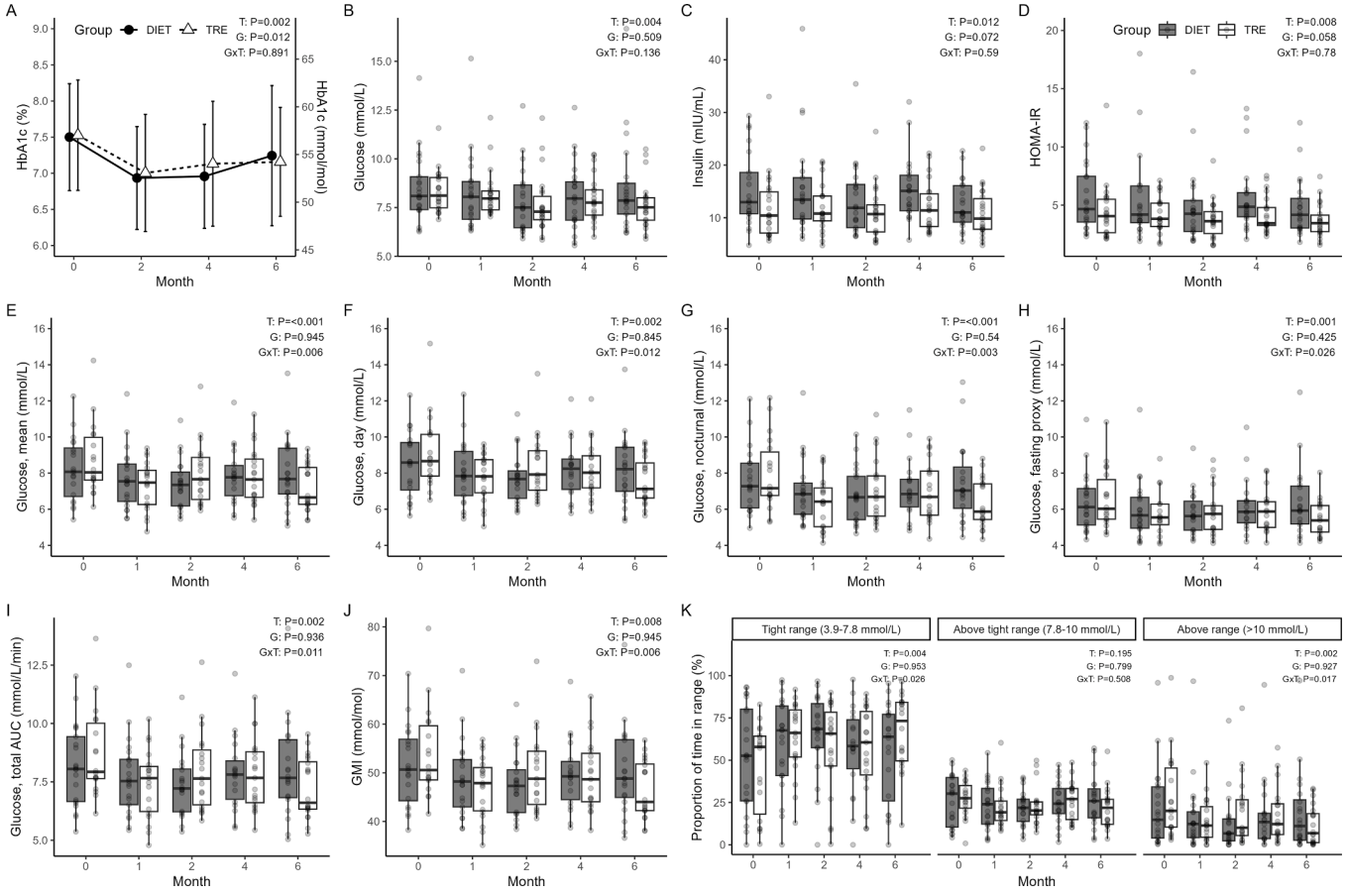

Fig. 2. HbA1c (% and mmol/mol, A), fasting glucose (mmol/L, B), fasting insulin (mU/mL, C), HOMA-IR (D) and CGM metrics (mean glucose over 24-h (mmol/L), E; mean glucose during day (0600 – 0000) hours (mmol/L), F; mean glucose during night (0000 – 0600) hours (mmol/L), G; fasting proxy (mmol/L), H; total AUC (mmol/L/min), I; glucose management index (GMI; mmol/mol), J; and proportion of time in tight range, above tight range and above range (%), K) across the 6-month intervention in people with type 2 diabetes mellitus who were randomised to individualised dietary guidance (DIET, black circles/boxes) or time-restricted eating (TRE, white triangles/boxes) groups. The boxplot centreline is the median value with upper and lower edges of the box being the first and third quartile, respectively, and the whiskers represent 1.5x the IQR. Statistical analysis was conducted with linear mixed effects models (using main effects of group (G), time (T) and interaction (GxT)).

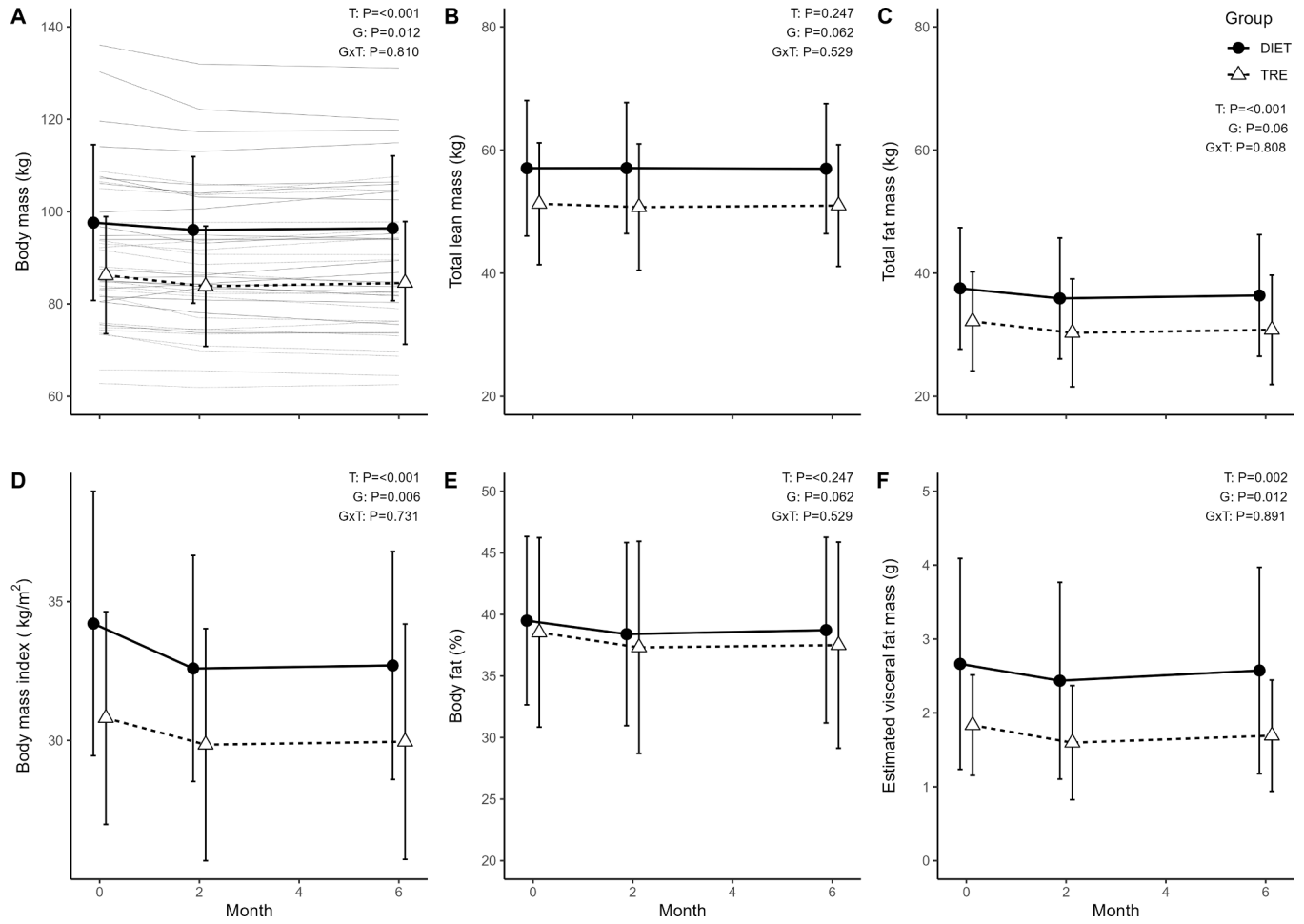

Fig. 3. Body mass (kg, A), lean mass (kg; B), fat mass (kg, C), body mass index (BMI, kg/m^2, D), body fat (%, E) and estimated changes in visceral fat mass (g, F) across the 6-month intervention in people with type 2 diabetes mellitus who were randomised to individualised dietary guidance (DIET, black circles) or time-restricted eating (TRE, grey triangles) groups. Statistical analysis was conducted with linear mixed effects models (using main effects of group (G), time (T) and interaction (GxT)).

total lean mass (Fig. 3B). Body fat percentage reduced over time, but this was only significant between baseline and six months in the TRE group (−1.0%, P=0.02; Fig. 3E). There was a main effect of group for estimated visceral adipose tissue (VAT) masses (Fig. 3F), as well as a main effect of time, but no interaction effect. Post-hoc tests revealed only the TRE group reduced VAT between baseline and two months (−0.25 kg, P=0.05), which did not persist to six-months (−0.14 kg, P=0.51).

Dietary intake (baseline and intervention) is presented in Table 2. Most DIET participants (>95%) had at least four days of valid dietary records, with the TRE group initially (month one and two) having 73% and 82% of participants with valid dietary records (Table S1). Daily energy intake was reduced between baseline and six months by ~900 kJ/d (~220 kcal/d; main effect time, P<0.001), with no effect of group or interaction. The variability of energy intake across participants is shown in Figure S4. There were main effects of time for reductions in total energy, carbohydrate (CHO), sugars, total fat, saturated fat, protein, and dietary fibre (all P<0.001), with no change to alcohol intake. The only difference between groups was for dietary fibre, which was lower in the TRE group. There was an interaction effect for saturated fat intake, where saturated fat intakes were lower than baseline at all intervention timepoints (one-to-six months) in the DIET group (~−5–6 g/d, P<0.003) but did not change in the TRE group. For CHO intake, there were reductions in TRE between baseline and all intervention timepoints (~−22–28 g/d; P<0.03) but only between baseline and one and two months in DIET (~−29 g/d, P<0.001). Sugar intake was significantly reduced in TRE between baseline and all intervention timepoints (~−12 g/d; P<0.04), with no changes in sugar intake in DIET.

Both interventions tended to reduce the number of eating occasions (EO) from baseline, and, overall, the TRE group had fewer EO (Table 2). Eating window data were included for 67% of DIET intervention days and 63% of TRE intervention days (Table S1). At baseline, there were no differences between groups in eating window duration (~11.0–11.7 h), time of first EO (~0830–0900 h) and time of last EO (~2000–2015 h; Table 2, Figure S5). During the intervention there were significant changes in eating window behaviours in the TRE group, with main effects of time, group and interaction on eating window duration, and time of first and last EO (all P<0.001). The TRE group reduced their eating window by ~2.8 h (95%CI: −3.5 to −2.1 h), by delaying their first EO by ~1.6 h (95%CI: +2.2 to +1.0 h) and having an earlier last EO by ~1.2 h (95%CI: 0.8 to 1.7 h), across the 6-months. For the TRE group, adherence to the 9-h duration of eating (Figure S6; 78–90%) higher than adhering to the 9-h advised (i.e., within 15 min of 1000 and 1900 h; 53–80%) eating window. Adherence to the start of the eating window was higher (81–97%) than adherence to the end of the eating window (67–83%; Figure S6). The DIET group did not change their eating window, or time of first or last EO (Table 2).

Across the intervention, self-reported adherence was ~20% higher in TRE than the DIET group (P<0.001), and adherence reduced over time in both groups (P<0.04). Thus, there was a significant interaction, where the DIET group reported lower adherence and greater reductions in adherence over time compared to the TRE group (Figure S7). There

Table 2
Dietary intake (≥4 days) from the two-week dietary recording periods across the 6-month intervention in people with type 2 diabetes mellitus who were randomised to individualised dietary counselling (DIET) or time-restricted eating (TRE) groups.

	DIET					TRE					*p*		
Month	**0**	**1**	**2**	**4**	**6**	**0**	**1**	**2**	**4**	**6**	**Time**	**Group**	**Interaction**
Energy (kJ/day (kcal/day))	9945 ± 3179 (2376 ± 759)	8975 ± 3063* (2144 ± 732)*	9021 ± 2812* (2155 ± 672)*	9335 ± 2980* (2230 ± 712)*	9017 ± 2924* (2154 ± 698)*	9430 ± 3323 (2253 ± 794)	8914 ± 2833* (2130 ± 677)*	9307 ± 3247 (2223 ± 776)	8315 ± 2796* (1986 ± 668)*	9005 ± 2828* (2151 ± 676)*	**<0.001**	0.58	0.13
CHO (g/day)	228 ± 78	202 ± 89*	203 ± 80*	214 ± 87	213 ± 87	236 ± 87	210 ± 81*	219 ± 88*	208 ± 81*	218 ± 88*	**<0.001**	0.52	0.37
Sugar (g/day)	84 ± 38	74 ± 35	77 ± 37	81 ± 41	79 ± 41	80 ± 47	68 ± 37*	72 ± 40*	67 ± 36*	68 ± 38*	**<0.001**	0.28	0.16
CHO (%TEI)	39 ± 9	38 ± 12	38 ± 9	39 ± 10	40 ± 10	42 ± 10	40 ± 10	40 ± 10	42 ± 10	40 ± 10	0.05	0.09	0.39
Fat (g/day)	107 ± 48	98 ± 44*	94 ± 39*	97 ± 41*	94 ± 39*	94 ± 49	91 ± 36	95 ± 42	83 ± 35*	93 ± 40	**<0.001**	0.34	0.15
Saturated fat (g/day)	39 ± 21	34 ± 18*	33 ± 17*	35 ± 17*	33 ± 17*	34 ± 18	35 ± 15	35 ± 17	31 ± 15	34 ± 15	**<0.001**	0.65	**0.03**
Fat (%TEI)	40 ± 9	40 ± 10	39 ± 9	38 ± 9	39 ± 9	37 ± 9	38 ± 9	38 ± 8	37 ± 9	39 ± 9	0.06	0.28	0.07
Protein (g/day)	106 ± 40	100 ± 36	105 ± 40	102 ± 37	96 ± 33*†	94 ± 36	96 ± 39	98 ± 43	85 ± 40*	95 ± 38	**<0.001**	0.13	**0.03**
Protein (g/kg/day)	1.1 ± 0.4	–	1.1 ± 0.4	–	1.0 ± 0.3*†	1.1 ± 0.4	–	1.1 ± 0.4	–	1.1 ± 0.4	**0.02**	0.98	**0.02**
Protein (%TEI)	18 ± 4	19 ± 5	20 ± 6*	19 ± 6	18 ± 5†	17 ± 5	18 ± 6	18 ± 6	17 ± 6	18 ± 6	**<0.001**	0.10	**<0.001**
Fibre (g/day)	33 ± 19	29 ± 12*	31 ± 15	30 ± 15	28 ± 12*	26 ± 11	25 ± 20	25 ± 11	23 ± 10	22 ± 10*	**<0.001**	**0.03**	0.26
Alcohol (g/day)	4 ± 14	2 ± 9	4 ± 12	5 ± 14	5 ± 12	6 ± 19	7 ± 19	9 ± 20	4 ± 14	7 ± 19	0.05	0.64	0.45
Number of EO#	4.5 ± 0.6	4.2 ± 0.5	4.2 ± 0.8	4.4 ± 0.7	4.1 ± 0.7	4.3 ± 0.7	3.8 ± 0.6	3.9 ± 0.9	3.8 ± 0.6	3.5 ± 0.9	0.06	**<0.01**	0.91
Eating window (h:min)	11:40 ± 02:34	11:08 ± 02:26	11:00 ± 02:09	11:24 ± 02:27	11:18 ± 02:37	11:03 ± 02:12	08:03 ± 01:53 ‡§	08:25 ± 01:28 ‡§	08:22 ± 01:24 ‡§	08:29 ± 01:40 ‡§	**<0.001**	**<0.001**	**<0.001**
Time of first EO (h:min)	08:32 ± 02:12	08:47 ± 02:21	08:52 ± 01:59	08:38 ± 02:10	08:53 ± 02:13	08:57 ± 01:57	10:31 ± 00:41 ‡§	10:27 ± 00:56 ‡§	10:19 ± 01:07 ‡§	10:25 ± 01:28‡§	**<0.001**	**<0.01**	**<0.001**
Time of last EO (h:min)	20:12 ± 01:31	19:55 ± 01:25	19:52 ± 01:31	20:02 ± 01:29	20:12 ± 01:26	19:58 ± 01:35	18:31 ± 01:24 ‡§	18:53 ± 01:25 ‡§	18:41 ± 01:06 ‡§	18:55 ± 01:17 ‡§	**<0.001**	**<0.001**	**<0.001**

Key: CHO, carbohydrate; EO, eating occasions; TEI, total energy intake. Days, number of valid days classified as "Good", see Supplementary Material Figure S1; #Eating occasion defined (see Supplementary Material) as < 210 kJ energy at least 15 min apart, as per Leech et al (2015). Statistical analysis was conducted with linear mixed effects models (using main effects of group and time). From post-hoc tests (with Tukey adjusted p-values for multiple comparisons), significantly different to *Month 0 (baseline) within group ($P < 0.01$), ‡Month 0 (baseline) within group ($P < 0.001$), †Month 2 within group ($P < 0.01$), §between groups ($P < 0.05$).

was no change to physical activity across the intervention, measured by step count, or proportion of wake time spent standing, stepping, or sitting, or time in bed, across time or group (Table S4).

Fasting concentrations of metabolites and hormones measured are presented in Table S5. Resting seated blood pressures, systolic (SBP) and diastolic (DBP), were not different between groups but improved over time with no change to resting HR. SBP was reduced at two (−4 mmHg, P=0.04) and six months (−5 mmHg, P<0.01), but DBP was reduced only at six months (−3 mmHg, P<0.01), compared to baseline.

Adherence reduced over time in both groups with significant reductions at 2.5 months, and from 4.5 to 6 months, compared to the 0.5-month adherence (P<0.04; Figure S6). In the TRE group, 99% of participants reported ≥ 5 days/week adherence in the first two weeks which reduced to 81% at 6 months. In the DIET group, 79% of participants reported ≥ 5 days/week adherence in the first two weeks which reduced to 62% by 6 months.

4. Discussion

The improvements in glycaemic control measured after a six-month intervention were comparable between TRE and those measured after standard dietetic care provided to people with Type 2 diabetes mellitus. Similar enhancements in glycaemic control support TRE being implemented by primary care professionals prior to engaging with dietitians, especially in a constrained resource environment. Both TRE and dietary guidance led to reductions in energy intake, but the differential dietary adaptations, with reductions in fat intakes from dietary guidance compared to reductions in carbohydrate intake with TRE, could be attributed to the different type of advice provided.

The greatest improvement in HbA1c was observed after the first two months of both interventions with distinct dietary modifications. Although no further improvements in HbA1c were observed between two and six months, the change in HbA1c concentrations remained clinically significant (−0.4% (−5 mmol/mol) after six-months despite ceasing consultations after four-months. A systematic review of 10 TRE RCTs measuring HbA1c found a −0.3% reduction in HbA1c [32], albeit with a single study that included people with Type 2 diabetes mellitus [18]. Che et al., [18] found significantly greater improvements to HbA1c (TRE: −1.5% vs. control: −0.7%) after a 12 week intervention with an eating window of 10-h (0800–1800 h). Others [19] show that late TRE (1200–2000 h) reduced HbA1c by −0.7% after six-months in adults with Type 2 diabetes mellitus, but was not superior over intentional daily energy restriction. The more frequent support (i.e. weekly contact [18,19]), coupled with the higher HbA1c levels at baseline [>8%; 18, 19], likely underpin the greater improvements in glycaemia than our investigation. While we placed restrictions on both type and number of medications for participant inclusion, our findings are translatable to a real-world scenario indicating TRE is a comparable intervention to individualised dietary guidance for improving glycaemic management.

Changes in HbA1c were reflected in improvements to TIR, TITR and TAR over the first two months with little further enhancement of these CGM-derived metrics subsequently. Some studies of TRE in people with Type 2 diabetes mellitus have not included CGM data [18,20], while others have reported comparable results [19,21]. Specifically, the −0.7 mmol/L mean reduction in glucose concentration and +8% increase in TIR we observed is similar to the −0.6 mmol/L reduction and the +5%

TIR after six months [19] and −0.8 mmol/L change in mean glucose after three weeks [21]. Across these interventions [19,21], as well as the present study, participants wore blinded CGMs: this was integral to our dietary guidance or timing only intervention, such that the data was not available in real time. There may have been synergistic effects of utilising the CGM data whilst adapting their dietary intake or patterns, which requires future investigation.

Physical activity monitoring showed no changes in physical activity patterns during the intervention, with the changes measured likely due to those made to dietary intake and/or timing. Distinct from other TRE interventions [19,33–35], we chose not to target weight loss and did not prescribe energy restrictive diets or request that participants decrease energy intake. However, both groups spontaneously reduced energy intake, although the associated weight losses were clinically insignificant (i.e. <5%) and variable in both groups (i.e., Figure S4). Free-living TRE induces energy restriction without intent [18,33,34,36,37], therefore the reductions observed in the current study were not surprising. However, as discussed previously [38], very few TRE studies report dietary intake data beyond estimates of total energy intake. Our detailed dietary analysis revealed the energy reduction after TRE was from decreased carbohydrate and total sugar intake, whereas total and saturated fat intakes were reduced in the DIET group. Our TRE intervention limited intake of the last meal to 1900 h likely reducing after dinner snacks which are often high in sugar. While intentional dietary advice may have been expected to induce greater improvements in diet quality over TRE, there were no changes to diet quality beyond reductions in sugar. Our dietitians focussed on what *to* eat for good health, rather than stressing what foods were to be avoided.

Consistent with our pilot study and other investigations [28,39,40], adherence was consistently higher in TRE compared to the DIET group, and greater at the start of the eating window (i.e. first EO) compared to the end of the eating window (Fig. 3, Figure S6). The greater adherence to eating window duration over the start and end of eating window times (Figure S6), which became more evident over the intervention, suggests the 9-h duration was easier to adhere to than the prescribed start and end times. Whilst qualitative investigations of TRE experiences support the notion of TRE being easy to adopt [41], the measure of self-reported adherence in the DIET group is potentially construed by greater subjectivity. It is likely that our participant cohort (~6 years since diagnosis) have tried many previous diet strategies and may be used to "failing" when complying to dietary guidance.

In Australia, access to individual dietetic advice for people with Type 2 diabetes mellitus is restricted to a small number of subsidised consults with APDs. As TRE does not involve dietary prescription, it can be administered by other healthcare professionals (i.e., GPs, nutritionists, practice nurses and diabetes educators) equipped with knowledge of TRE strategies. We interviewed dietitians about their experiences and knowledge of TRE and they requested that the timing of eating to be embedded into Australian dietary guidelines [42]. Therefore, our findings support the incorporation of timing of meals into the Australian dietary guidelines.

This study highlights that TRE is implementable within the current Australian healthcare system and does not rely on overburdened dietetic services. We ensured rigorous collection of dietary and physical activity data and used an active control group (i.e., DIET) to understand non-inferiority. Limitations of our work include the increased heterogeneity between groups due to the parallel groups design. The stratified randomisation matched for HbA1c and sex but there were more Caucasians in DIET and more Asians in TRE, and, consequentially, differences in BM at baseline. Our inclusion criteria were limited to those who were not taking insulin or sulphonylureas due to risk of hypoglycaemia with fasting, and use of GLP-1 agonists was excluded. Therefore, our results require replication across those with Type 2 diabetes mellitus on combination of antihyperglycaemic medication.

The combined effects of starting with TRE advice and then building dietary guidance has yet to be investigated. Anecdotally, our TRE participants requested dietary guidance after the two months, where they wanted to specifically address their dietary quality. Several studies have demonstrated that more regular dietetic support improves glycaemic outcomes for individuals with Type 2 diabetes mellitus [1,43], evidencing the need for increased Allied Health resourcing.

In conclusion, both TRE and individual dietary counselling improved glycaemic control over a six-month intervention. As TRE is a practical intervention with higher self-reported adherence that can be effectively delivered by a non-dietitian, it may facilitate early improvements in glycaemic control and prompt behaviour change and motivation for more individualised advice. As the greatest change in glycaemia occurred in the first two months, future interventions may look to support dietary change on top of TRE to further improve glycaemic management.

5. Contribution Statement

E.B.P., was involved in the conception, design, and conduct of the study and the analysis and interpretation of the results. B.E.R., and R.E.C., were involved in the design, conduct of the study and the analysis of results. S.A.F. was involved in the conduct of the study and the interpretation of the results. N.S.L., S.L.H, and Z.S., were involved in the analysis and interpretation of results. R.D.J. conducted the statistical analysis and interpretation of the results. L.B., I.W.K.K., B.L.D., and J.A.H., were involved in the conception, design, and interpretation of the results. E.B.P. wrote the first draft of the manuscript, and all authors edited, reviewed, and approved the final version of the manuscript. E.B.P. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

6. Prior presentation

Parts of this study were presented in abstract form at the ADS and ADEA Australasian Diabetes Congress, Adelaide, Australia, 23–25 August 2023, and the 59th Annual meeting of the European Association for the Study of Diabetes, Hamburg, Germany, 2–6 October 2023.

CRediT authorship contribution statement

Evelyn B. Parr: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Bridget E. Radford:** Writing – review & editing, Methodology, Investigation, Formal analysis. **Rebecca C. Hall:** Methodology, Investigation, Formal analysis. **Nikolai Steventon-Lorenzen:** Writing – review & editing, Formal analysis, Data curation. **Steve A. Flint:** Writing – review & editing, Investigation, Formal analysis. **Zoe Siviour:** Writing – review & editing, Formal analysis. **Connie Plessas:** Writing – review & editing, Formal analysis. **Shona L. Halson:** Writing – review & editing, Formal analysis. **Leah Brennan:** Writing – review & editing, Methodology, Formal analysis. **Imre W.K. Kouw:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Rich D. Johnston:** Writing – review & editing, Visualization, Formal analysis. **Brooke L. Devlin:** Writing – review & editing, Visualization, Methodology, Funding acquisition, Conceptualization. **John A. Hawley:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Evelyn B. Parr reports financial support was provided by Diabetes Australia. Evelyn B. Parr reports financial support was provided by Australian Catholic University. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2024.111893.

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