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Modifying the timing of breakfast improves postprandial glycaemia in people with type 2 diabetes: A randomised controlled trial

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ABSTRACT

Aims: Investigate the effects of breakfast timing on postprandial glycaemia in adults with type 2 diabetes (T2D), and the impact of a 20-min walk after breakfast.

Methods: Eleven adults with T2D (57 ± 7 y; HbA1c 7.4 ± 1%) participated in a six-week randomised crossover controlled trial comprising three 4-day conditions: Early (0700 h), Mid (0930 h) and Delayed (1200 h). After each condition, a second 4-day intervention of 20-min walk after each condition was undertaken. Standardised breakfast was provided. Interstitial glucose and physical activity were measured. Incremental area under the curve (iAUC) 2-h post-breakfast, 24-h iAUC, and fasting glucose were analysed with linear mixed-effects models. Cohen's d of the 2-h iAUC post-breakfast 20-min walk was calculated.

Results: Mid and Delayed had lower 2-h post-breakfast iAUC ($p < 0.002$, −57 mmol/L×2h; $p < 0.02$, −41 mmol/L×2h) compared to Early. There were no differences in fasting (0600 h) glucose or 24-h iAUC. There was a small effect of the 20-min walk on lowering 2-h post-breakfast iAUC for Early ($d = 0.35$) and Delayed ($d = 0.37$), with no effect in Mid.

Conclusion: In people with T2D, delaying breakfast from 0700 h to mid-morning or midday reduced postprandial glycaemia. Additional post-meal walking for 20 min had a small effect in lowering postprandial glycaemia when breakfast was at 0700 h or midday, but provided no additional benefit when breakfast was at mid-morning.

1. Introduction

The prevalence of type 2 diabetes (T2D) is increasing globally and represents a major public health problem [1]. By 2030 it is estimated that one in nine adults will have T2D (i.e., 643 million adults). Currently, management of T2D focuses on primary lifestyle interventions (i.e., diet and exercise) to improve insulin sensitivity and glycaemic management [2–5]. In this regard, there is growing evidence that the timing of both meals and exercise can exert profound effects on numerous metabolic processes and may be important for maximising health outcomes [6,7].

There are several definitions of breakfast which include 1) the first meal eaten within the first 2 h after waking, 2) the first meal that breaks fasting after sleep within two or 3 h from awakening, or 3) the consumption of food between 0500 and 0900 h [8]. In women with overweight or obesity, consuming breakfast between 0600 and 0900 h reduced triglyceride levels, fasting glucose concentration, satiety scores, and HOMA-IR (Homeostasis Model Assessment of Insulin Resistance) [9]. Consuming breakfast, with nutritional quality, and balanced quantities, is associated with a reduced risk of chronic metabolic diseases and improved overall health [10,11].

For people with T2D, the greatest blood glucose excursions occur after breakfast [12,13]. The occurrence of peak cortisol levels (~0800 h) and the 'Dawn Phenomenon' (i.e., elevated glucose levels upon waking) elicit heightened glucose levels in the early morning in people with T2D [14–17]. Accordingly, interventions that reduce morning hyperglycaemia are important for overall daily glucose management in persons with T2D. However, no studies to date have compared the effects of varying the times of breakfast on blood glucose response in people with T2D. Therefore, the primary aim of the present study was to investigate the effects of modifying the timing of breakfast on postprandial breakfast glucose responses in free-living adults with T2D. We hypothesised that delaying the first eating occasion would result in lower iAUC when breakfast was delayed from 0700 to either 0930 or 1200 h.

Many studies have investigated the association between the timing of exercise relative to a single meal or over the course of a day on glycaemic

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management [18,19] and reported additive effects on blood glucose management [20,21] compared with altering the timing of meals or dietary modifications alone. An exploratory aim of the current study was to determine the effects of an acute bout of exercise after breakfast on postprandial glycaemia.

2. Methods

2.1. Study design

This study was a randomised crossover controlled trial conducted at Australian Catholic University's (ACU) Melbourne campus between October 2022 and March 2023. Ethics approval was obtained from the ACU Human Research Ethics Committee (2022-2741HC). The study was prospectively registered on the Australia New Zealand Clinical Trial Registry (ACTRN12622001177741). The participants were informed of all study requirements prior to the electronic informed consent via Research Electronic Data Capture (REDCap) [22]. At study completion, participants were provided with a $100 gift card to compensate for costs incurred over the length of the study (travel to the lab and parking).

Fourteen men and women, aged 30 to 70 y, diagnosed with T2D (HbA1c ≥6.5 – <10%) and not currently taking sulphonylureas, insulin, or more than two oral hypoglycaemic agents were recruited through the Mary MacKillop Institute for Health Research database and the National Diabetes Service Scheme email advertisements. Participants were excluded if they reported meeting or exceeding the Australian physical activity guidelines (>150 min/week) [23], were following a ketogenic (i.e., <50 g carbohydrates/day), a time-restricted eating pattern (i.e., eating window <12 h), alternate day fasting, intermittent fasting or unwilling to consume breakfast at the prescribed times for the study period. Further exclusion criteria included shift workers (i.e., more than one shift per month between 2200 and 0500 h), smokers (including e-cigarettes, tobacco, marijuana, or within three months of quitting), people with a history of psychotic disorders, or current diagnosis of other major psychiatric illness (e.g. mood disorder, eating disorder, substance use disorder), or diagnosis of gastrointestinal conditions. All potential participants were screened for risk of disordered eating with the Eating Attitudes Test (EAT-26) questionnaire and were ineligible for the study if they had a score ≥20.

2.2. Study visits

Participants attended four separate laboratory visits over six weeks (Fig. 1). To ensure eligibility (diagnosis of T2D), a finger prick blood sample was initially obtained to measure HbA1c (Cobas b 101 System, Roche, Switzerland). A dual-energy X-ray absorptiometry (DXA) scan was conducted to assess body composition (GE Lunar iDXA Pro, encore software Version 16, USA), and a 12-h fasting mixed meal tolerance test (MMTT) was undertaken on the morning of the first visit to estimate baseline beta cell function.

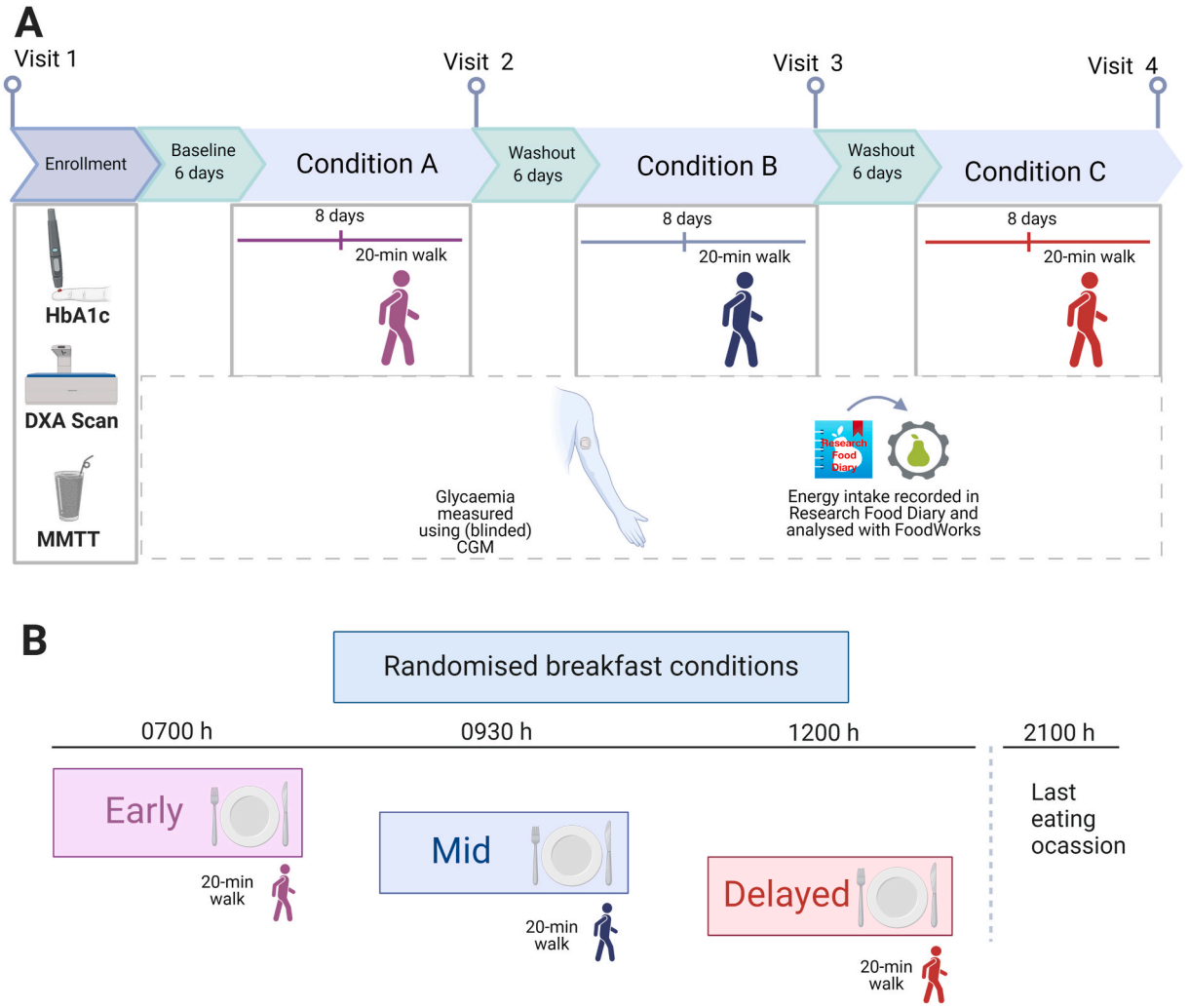

Fig. 1. Schematic overview of the study design (A) and breakfast timing conditions (B) completed by participants with type 2 diabetes. CGM, continuous glucose monitor, DXA, dual X-ray absorptiometry, HbA1c, glycated haemoglobin, MMTT, mixed meal tolerance test. Figure created with BioRender.com.

Participants completed a six-day harmonisation period in which they were asked to record daily food and beverage intake in a Research Food Diary App (Xyris, Brisbane, Australia) or using handwritten records. Participants took photos of all food consumed throughout the day, recorded the timing of their meals, and were provided with a handbook to self-record sleep quality and duration. During the harmonisation phase and for the duration of the study, a continuous glucose monitor (CGM) sensor (Freestyle Libre Pro iQ, Abbott, USA) and an activity monitor (ActivPAL4™, Pal Technologies, Scotland) were attached to the upper arm and mid-thigh, respectively, and worn throughout the six-week study period. Glucose and activity monitors were replaced every 14 days or when CGMs failed or dislodged.

2.3. Intervention

The order of completing the three different breakfast timing conditions (Early: 0700 h, Mid: 0930 h, or Delayed: 1200 h) was randomly allocated, and participants were instructed to consume breakfast at the prescribed time for eight consecutive days. Randomisation was implemented using REDCap, with blocked (n = 6) randomisation, where both participants and study staff were unblinded to the randomised condition. During the last four days of each condition, participants were asked to complete 20-min of brisk walking 30–60 min after consuming breakfast. Participants received individualised breakfast meals and were instructed not to eat for the 2-h following breakfast, to continue their usual eating habits for the rest of the day, and to finish consuming their last meal before 2100 h (i.e., no energy intake after 2100 h). No additional dietary advice was provided. Between each eight-day intervention period, a six-day washout occurred in which no instructions on the timing of breakfast or food intake were provided. However, participants continued wearing the glucose, activity, sleep monitors, recorded their food intake, and took photos of their meals.

2.4. Body composition and anthropometrics

For estimating body composition, participants underwent a total body DXA scan (12-h fasted and after voiding). Height (cm) was measured using a wall stadiometer, and weight (kg) was recorded using digital scales.

2.5. Mixed meal tolerance test (MMTT)

Glucose and insulin responses to the consumption of a liquid meal (milk and Sustagen Hospital Formula Nutritional Supplement Vanilla Flavour; ~300–400 mL; 20% of daily energy requirements using Schofield equation [24]; ~55% carbohydrates, 23% fat and 22% protein) were measured to provide an indirect estimate of beta cell function. Blood (5 mL) was sampled before consuming the drink and every 30 min thereafter for 2-h from an intravenous cannula.

2.6. Menstrual history questionnaire

Female participants completed the menstrual history questionnaire (via REDCap) to estimate the menstrual cycle phase in relation to the baseline dates or report menopause status.

2.7. Dietary assessment

Participants completed a food record using the Research Food Diary App (Xyris, Brisbane, Australia) or a written diary to record all dietary intake throughout the six-week intervention (including baseline and washout periods). Participants captured photos of their foods to validate meal timing. Each participant had an individually prescribed breakfast provided for each intervention day (i.e., 24 breakfast serves). The breakfast was planned with the participant and adjusted to a similar energy and macronutrient distribution to ensure adequate energy intake, providing ~25% of the total daily energy requirements according to the Schofield equation with 1.3 physical activity factor and the following macronutrient energy distribution: 50% carbohydrate, 30% fat, and 20% protein.

2.8. Data analysis

Data were included when participants were adherent with the prescribed timing of the meal (±35 min, i.e., 0625 – 0735 h for Early, 0855–1005 h for Mid, and 1125–1235 h for Delayed) and when the energy consumed at breakfast was ±5% of the prescribed amount (Table S1), for a minimum of two days for each of the 4-day conditions. CGM data were analysed for the 2-h postprandial period. Incremental glucose area under the curve (iAUC) in the 2-h post-breakfast period of each condition (Early, Mid, and Delayed) was prespecified as the primary outcome, as the most relevant outcome from a short-term study with CGM data. 2-h iAUC was calculated using the trapezoid method using Microsoft Excel. In addition, 24-h iAUC, fasting glucose (0600 h) concentrations, and time in range (percentage of time glucose levels are in a target range between 3.9 and 10.0 mmol/L [25]) were calculated from the CGM data. As the exercise was an exploratory outcome, we did not include criteria for achieving the prescribed exercise, and thus, we analysed the total number of steps taken in the 2-h after commencing breakfast. Dietary analysis was undertaken using FoodWorks 10 software (Xyris, Brisbane, Australia). The breakfast time was taken from time-stamped photos participants took of each meal or from the reported time on the participant's handbook when photos were missing. Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) was calculated to estimate insulin resistance based on fasting glucose and insulin levels using the following formula: ([fasting insulin] × [fasting glucose]/22.5) [26]. The Disposition Index was calculated to assess the beta cell function response to the MMTT using the following formula: Insulin Sensitivity Index × (MMTT $AUC_{Insulin}$/MMTT $AUC_{Glucose}$) [27].

2.9. Statistical analysis

Power and sample size calculations were based on 2-h AUC data from our previous intervention [28] where n = 9 participants provided 80% power at a 5% significance level for detecting a change of 96 ± 168 mmol/L×2h between the three breakfast timing conditions. To account for participant dropout and monitor failures, fourteen participants were recruited. To assess the effect of condition (breakfast timing and exercise) on 2-h postprandial iAUC, 24-h iAUC, fasting glucose, time in range, 2-h postprandial peak glucose, energy consumption, and step count, statistical analyses were performed in R using linear mixed-effects models using the lmer function within the lmerTest package and fitted with restricted estimate maximum likelihood. Within each model timing of breakfast and exercise conditions were included as fixed effects with an interaction, and participant ID was included as a random intercept term; separate models were built for each outcome variable. The main effects were extracted from each model using the anova function. Post hoc pairwise comparisons were performed using the lsmeans function from the emmeans package, with a Tukey adjustment for multiple comparisons. The magnitude of differences were assessed Cohen's d effect size statistic with 95% confidence intervals using the Cohen.d function, effect sizes were interpreted as small, medium, large or negligible. Significance was set at $p < 0.05$ a priori, all data are reported as mean ± standard deviation (SD) unless specified.

3. Results

Thirteen of the fourteen recruited participants who met the screening criteria completed the study. One participant withdrew following the completion of the first condition. Two participants were excluded from the final analysis; one participant completed night shifts during one condition and was non-adherent to the prescribed timing or breakfast in

another condition. The second participant was excluded as the postprandial meal effects could not be measured accurately as they had elevated glucose levels (i.e., >15 mmol/L for the duration of the study). Therefore, eleven participants were included in the primary outcome analysis and ten in the secondary outcome (exercise) analysis (Fig. S1). The habitual breakfast timing was 0900 ± 0045 h, the habitual energy intake for breakfast was ~1461 kJ ± 363 kJ (Table S1) and the characteristics of all participants are presented in Table 1. An overview of the total data available for analysis (i.e., breakfast time, energy intake and CGM data) is presented in Supplementary Table S2. There was no difference in adherence to the three breakfast timing conditions.

Energy intake and macronutrient composition at breakfast were not different between conditions (Table 2). The change in breakfast timing had little effect on total daily energy, macronutrient intake, or the number of eating occasions, defined as >210 kJ [29]. The remainder of daily energy intake was not different between any of the conditions.

In the breakfast only (non-exercise) conditions, there was no difference between the number of steps taken in the 2-h postprandial period (Fig. 2A). However, as intended, there was a significant increase in the 2-h postprandial breakfast step count between the periods that participants were instructed to complete 20-min of walking after each condition compared to the periods they did not intentionally exercise after breakfast (Early $p = 0.0005$; Mid $p = 0.002$; Delayed $p = 0.01$). Furthermore, there was a significant increase in the step count from waking to breakfast in Delayed ($p = 0.0004$; +2119 mean steps) compared to Early (+182 mean steps) and Mid ($p = 0.03$; +1039 mean steps) (Fig. 2B). There was no difference in the total daily step count between Early, Mid and Delayed conditions (Fig. 2C; Table S3). However, there was a significant increase in the total daily step count between Early and EarlyEx ($p = 0.03$) and Mid with MidEx ($p = 0.01$), but not between Delayed and DelayedEx ($p = 0.26$; Fig. 2C).

A main effect of breakfast time was observed for 2-h breakfast iAUC ($p = 0.002$; Fig. 3B), where Mid and Delayed had lower 2-h post-breakfast iAUC ($p < 0.002$, −57 mmol/L×2h; $p < 0.02$, −41 mmol/L×2h, respectively) compared with Early. There was no significant difference in 2-h iAUC between Delayed and Mid breakfast timings. There were no differences in 24-h iAUC (Fig. 3C) or fasting (0600 h) glucose concentrations (Fig. 3A), time in range (Fig. 3D) or 2-h postprandial peak glucose (Fig. 3E) between Early, Mid and Delayed breakfast timings. There was a small effect on the reduction of iAUC 2-h post-breakfast for Early (Cohen's d = 0.35; 95%CI = −0.62 to 1.33) and Delayed (Cohen's d = 0.37; 95%CI = −0.54 to 1.30) conditions, but no beneficial effect (Cohen's d = −0.50; 95%CI = −1.46 to 0.44) on Mid condition when 20-min of post-meal exercise was performed (Table S4).

Table 1
Baseline characteristics of the participants.

	All randomised (n = 14)	Completers (n = 13)	Adherent to meal conditions (n = 11)
Age, y	58.8 ± 7.6	58.0 ± 7.1	57.0 ± 7.1
Sex, n (%)			
Female	4 (29%)	3 (23%)	2 (18%)
Male	10 (71%)	10 (77%)	9 (82%)
Body mass, kg	81.1 ± 17.4	79.9 ± 17.5	77.6 ± 12.3
Lean mass, kg	49.7 ± 10.2	50.2 ± 10.4	50.3 ± 9.3
Fat mass, kg	29.3 ± 10.9	27.6 ± 9.3	25.2 ± 5.2
Body fat, %	35 ± 8	34 ± 6	32 ± 5
Height, cm	166.3 ± 8.7	167.0 ± 8.5	166.7 ± 7.8
BMI, kg/m^2	29.2 ± 5.3	28.5 ± 4.7	27.9 ± 4.1
EAT-26	5.2 ± 3.3	5.5 ± 3.3	5.9 ± 3.3
Medication, n (%)			
None	2 (14%)	1 (7%)	0
1 OHA	4 (29%)	4 (31%)	4 (36%)
2 OHAs	8 (57%)	8 (62%)	7 (64%)
HbA1c, %	7.4 ± 1.1	7.5 ± 1.1	7.4 ± 1.0
HbA1c, mmol/mol	54.9 ± 18.4	55.1 ± 19.2	52.9 ± 18.9
HOMA-IR	4.1 ± 1.7	3.7 ± 1.0	3.8 ± 1.1
Disposition Index	205.2 ± 102.3	198.6 ± 103.3	206.9 ± 106.7

Data presented as n (%) or mean ± SD. BMI, body mass index, EAT-26, Eating attitudes test to identify eating disorder risk (score ≥20 indicates risk), HOMA-IR, homeostatic model assessment and insulin resistance, OHA, oral hypoglycaemic agents.

Table 2
Total breakfast and non-breakfast energy intake (mean ± SD) for each period of the conditions consumed by people with type 2 diabetes.

Condition	Early	EarlyEx	Mid	MidEx	Delayed	DelayedEx
Breakfast						
Energy (kJ)	2237 ± 407	2219 ± 439	2209 ± 391	2239 ± 386	2195 ± 402	2257 ± 383
Protein (g)	25 ± 4	24 ± 4	25 ± 4	25 ± 4	25 ± 3	27 ± 7
Fat (g)	21 ± 7	19 ± 8	21 ± 7	21 ± 7	22 ± 7	23 ± 7
CHO (g)	56 ± 11	57 ± 15	55 ± 10	55 ± 10	53 ± 11	55 ± 16
Fibre (g)	10 ± 3	9 ± 3	10 ± 3	9 ± 3	9 ± 3	9 ± 3
Non-Breakfast						
Energy (kJ)	7627 ± 2052	9186 ± 2912	7260 ± 2427	7160 ± 2117	6730 ± 2646	6219 ± 2756
Protein (g)	75 ± 29	90 ± 33	72 ± 40	72 ± 28	67 ± 24	65 ± 31
Fat (g)	65 ± 70	70 ± 34	53 ± 30	60 ± 30	63 ± 29	54 ± 26
CHO (g)	134 ± 51	148 ± 68	144 ± 45	141 ± 57	123 ± 38	113 ± 56
Fibre (g)	20 ± 21	26 ± 21	21 ± 22	19 ± 19	18 ± 13	18 ± 11
Total						
Energy (kJ)	9864 ± 2192	11404 ± 3224	9305 ± 2652	9399 ± 2404	8925 ± 2993	8476 ± 3111
Protein (g)	100 ± 31	117 ± 32	98 ± 40	98 ± 31	92 ± 26	92 ± 35
Fat (g)	86 ± 35	91 ± 38	76 ± 34	81 ± 36	84 ± 34	76 ± 33
CHO (g)	190 ± 47	205 ± 70	197 ± 30	193 ± 60	179 ± 47	166 ± 72
Fibre (g)	30 ± 22	36 ± 23	30 ± 23	29 ± 21	27 ± 14	26 ± 13
Eating occasions (n)	3.9 ± 0.8	4.1 ± 0.8	3.8 ± 0.7	3.7 ± 0.7	3.5 ± 0.5	3.3 ± 0.8

Mean ± SD energy intake (kJ) per day. CHO, carbohydrate. Conditions: Early 0700 h, Mid 0930 h, Delayed 1200 h; Conditions with 20 min of brisk walking after breakfast: EarlyEx, MidEx, and DelayedEx. The ANOVA statistical analysis showed no main effects of group.

4. Discussion

This is the first study to investigate the effects of modifying breakfast timing on 2-h postprandial glycaemia in people with T2D. For the purpose of this research, we have defined breakfast as the first meal of the day irrespective of time since waking. We report that delaying breakfast to mid-morning or midday was associated with improvements in the 2-h postprandial breakfast glycaemic response compared to eating at an earlier time (~0700 h). A 20-min brisk walk undertaken after breakfast had only a small effect on reducing postprandial glycaemia in early-morning and midday conditions. Modifying the timing of breakfast offers a simple, practical strategy for people with T2D to improve their postprandial breakfast response with the potential to reduce the risk of comorbidities when practised over the long term.

Our previous research found that delaying the first meal of the day until mid-morning (~1000 h) reduced peak glucose, insulin concentrations and iAUC after lunch in people at risk of T2D [30]. To extend on this work, the current study also tested a delayed breakfast at midday, distinct from skipping breakfast, and found that both mid-morning and

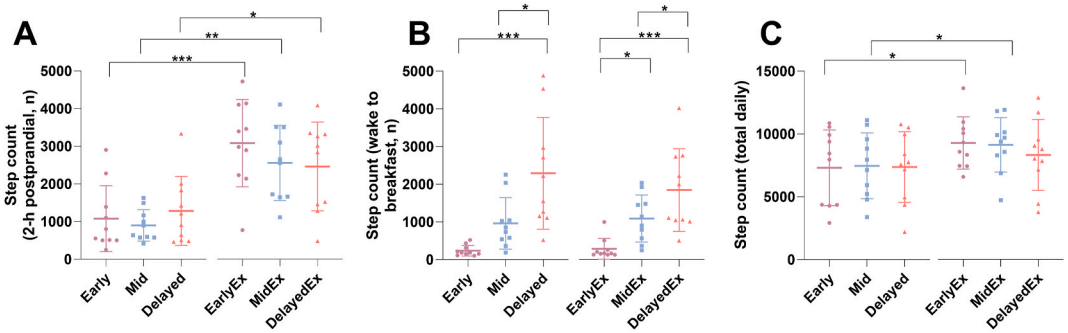

Fig. 2. Step count of 2-h postprandial breakfast (A), Step count from waking up to breakfast (B), Total daily step count (C). Conditions: Early 0700 h, Mid 0930 h, Delayed 1200 h; Conditions with 20 min of brisk walk after breakfast: EarlyEx, MidEx, and DelayedEx. The centre line indicates the mean; lower and upper boundaries are the 25th and 75th percentile, respectively. Key: pink circles, Early; blue squares, Mid; red triangles, Delayed. Data points illustrate individual responses. Statistical significance between non-exercise and exercise, or within breakfast or breakfast-exercise timing conditions; *p = 0.05, **p = 0.01, ***p = 0.001.

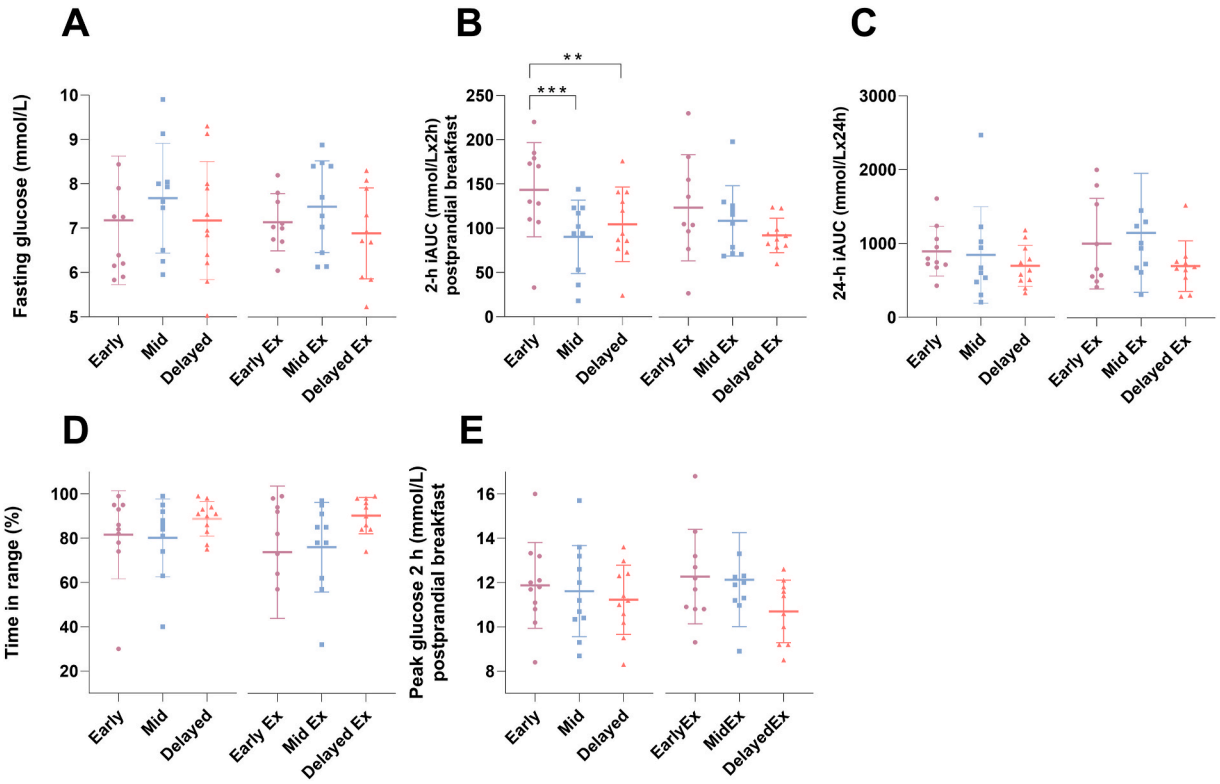

Fig. 3. Fasting glucose at 0600 h (A), iAUC for 2-h postprandial breakfast (B), 24-h iAUC (C), Time in range (3.9–10 mmol/L; (D)), Peak glucose 2-h postprandial breakfast (E) in free-living individuals with type 2 diabetes. The centre line indicates the mean; lower and upper boundaries are the 25th and 75th percentile, respectively. iAUC expressed in mmol/Lx2h. Key: Early, pink circles; Mid, blue squares; Delayed, red triangles. Data points illustrate individual responses. *p < 0.05, **p < 0.01, ***p < 0.001.

midday breakfast consumption reduced postprandial glycaemia. In a controlled laboratory setting, Jakubowicz et al. [31], showed that skipping breakfast (first eating occasion at midday) impaired glucose and insulin responses to lunch and dinner meals compared to an early (0800 h) first meal. Based on these findings, we hypothesised that breakfast at midday would not improve post-breakfast glycaemia. However, in the current study breakfast was delayed rather than omitted, as participants were requested to resume their usual dietary intake and physical activity was not constrained in our free-living intervention. Hence, the number of steps prior to breakfast in the delayed and mid-morning breakfast conditions may have had a confounding effect, via increased utilisation of circulating glucose and/or blunted circulating insulin concentrations [32,33], on reducing postprandial glycaemia compared to consuming breakfast early.

The post-breakfast exercise bout did not confer an additional benefit beyond the observed effect of modifying the timing on lowering the 2-h post-breakfast glucose levels. However, as supported by the results of other studies, a small effect was observed when exercise was added to early-morning and midday breakfast conditions. Post-meal walks are associated with an improvement in insulin sensitivity [34], insulin concentrations [19] and postprandial glycaemia response in people with T2D [35,36]. Reynolds et al. [36] instructed participants to either undertake a 30-min daily walk or 10-min walks after each meal and reported a lower iAUC only after the dinner meal. However, the 10-min walk post-breakfast did not increase the total post-meal step count, so there was no reduction in postprandial glycaemia after breakfast consumption [36]. In the present study, physical activity was manipulated in the postprandial period after breakfast, as the largest postprandial

excursions are in the morning [13]. We asked participants to walk for 20 min to meet physical activity guidelines and ensure there was adequate post-meal exercise stimulus. However, by changing the timing of breakfast, the number of steps accumulated before breakfast was also modified. Hence, the uninstructed step count rise before the Delayed condition, compared to Early and Mid conditions, might potentially influence the reduction of the 2-h postprandial glucose peak. Despite accumulating more steps before breakfast in Mid (~930 steps) and Delayed (~1820 steps) compared to Early (~230 steps), participants adhered to the exercise advice by increasing their post-breakfast step count by ~1800 steps in all exercise conditions. As a result, participants in the Early-exercise and Mid-exercise conditions increased their total daily count by ~1000 steps, in contrast to the Delayed condition where total step count was not different with the additional structured exercise. Increasing physical activity lowers the requirement for insulin whilst reducing glycaemia through contraction-mediated pathways [34]. We intentionally designed and conducted our study based on a glucose-centric model of glycaemia and negated measures of insulin due to the free-living nature of the study. As such, we are unable to assess the effects of the modified breakfast with or without exercise on post-meal insulinemia.

Despite a reduced glycaemic response to mid-morning or midday breakfast timings, we did not find differences in 2-h postprandial peak glucose, 24-h iAUC or time in range. Modifying the timing of breakfast did not change total daily energy intake, or the remainder of energy intake (after breakfast was accounted for). Despite the reduction in time available to eat induced by consumption of the first meal at midday, participants still self-reported eating "lunch" and "dinner" in all conditions and the number of eating occasions was not different between conditions. Consumption of a delayed breakfast and the subsequent reduction in the eating window in free-living individuals without a prescribed breakfast schedule may induce reductions in the number of eating occasions, underpinning any observed decreases in total daily energy intake, such as in time-restricted eating interventions [37,38].

4.1. Strengths and limitations

The strengths of the current study were the free-living conditions under which the trial was conducted, in which participants were not asked to change their daily routine or dietary choices except for breakfast. Attention to the timing of eating, especially the first meal of the day upon waking, is recommended for improved management of glycaemia in persons with T2D. We did not assess the feasibility of eating breakfast at the pre-specified times, as one of the inclusion criteria was the ability to complete the experimental conditions. Furthermore, we limited the timing of the last eating occasion to 2100 h, which may not reflect what would occur with altered breakfast timing in free-living dietary practices (i.e., people may eat later). Despite a longer overnight fast in the Mid and Delayed conditions, we did not observe changes in fasting glucose concentrations. We are likely underpowered for any exercise intervention outcomes and therefore have limited the analysis to only report effect size: future studies with greater participant numbers need to be undertaken to confirm or refute our preliminary findings. These results apply only to individuals who are not taking insulin or sulphonylurea medications, as both can contribute to hypoglycemia when meals are delayed. Therefore, individuals on insulin or sulphonylureas should seek medical advice before attempting any changes to their meal timing.

5. Conclusion

In people with T2D, delaying breakfast from 0700 h to mid-morning or midday lowered the 2-h postprandial glucose levels. When this practice is adhered to over the long-term, it is likely to result in improved glucose management, reducing many of the associated effects of suboptimal glycaemic control (i.e., insulin resistance or increased risk for cardiometabolic conditions). Additional post-meal walking for 20 min had a small effect in lowering postprandial glycaemia when breakfast was at 0700 h or midday, but provided no additional benefit when breakfast was at mid-morning. Future studies with larger sample sizes should explore the effects of postprandial physical activity on glycaemia and the long-term feasibility of manipulating the timing of breakfast and physical activity.

Data availability

Data may be made available upon request to the corresponding author.

Contribution statement

A.P.B.G was involved in the conception, design, and conduct of the study and the analysis and interpretation of the results. B.E.R., and A.J. R., were involved in the design and conduct of the study. J.A.H. and E.B. P, were involved in the conception, design, and interpretation of the results. A.P.B.G. 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.

Clinical trial registration

The study is registered at the Australia New Zealand Clinical Trial Registry (ACTRN12622001177741) and received ethics approval from the Australian Catholic University Human Research Ethics Committee (2022-2741HC).

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Declaration of competing interest

The authors 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.dsx.2024.103157.

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