



Time Restricted Eating and Glycaemic Control: Sorting Signal From Noise in Recent Trials

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Article Info

Volume 1, Issue 1, 2026

[doi: 10.62715/ijod.2026.v1.i1.pp8-14](https://doi.org/10.62715/ijod.2026.v1.i1.pp8-14)

Received: 08 October 2025

Accepted: 23 November 2025

Published: 27 January 2026

Abstract

Time restricted eating, in which all food is taken within a daily window of roughly four to ten hours, has moved from rodent chronobiology to clinical trials in little more than a decade. Its promoters claim benefits for glucose regulation that go beyond those of eating less. This narrative review examines how far recent human trials support that claim. It first sets out the circadian rationale, which predicts that an early window should matter more than a short one. It then considers the trials according to what each design can isolate: uncontrolled and single-arm studies, free-living comparisons with unrestricted eating, comparisons with matched energy restriction, and controlled feeding studies in which energy intake is fixed. Read in that order, the literature shows a consistent pattern. Glycaemic improvements are common when time restriction leads people to eat less and lose weight, and they shrink or disappear when energy intake is held equal, with early-window feeding studies as a partial and still fragile exception. Sources of noise are identified, among them short duration, small samples, self-reported adherence, differing outcome measures and uncontrolled medication changes. Time restricted eating is a reasonable way to reduce intake for people who prefer it, but a glucose-specific effect remains unproven.

Keywords: Time restricted eating, Intermittent fasting, Glycaemic control, Circadian rhythm, Type 2 diabetes, Randomised trials

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1. Introduction

Advice about diet has traditionally concerned what and how much to eat. Time restricted eating (TRE) concerns when. The person eats freely, or nearly so, within a fixed daily window and takes only water or non-caloric drinks for the remaining hours. The approach grew out of experiments in mice, in which confining access to a high-fat diet to the active phase prevented obesity, hyperinsulinaemia and hepatic steatosis although the animals ate as much as those fed at will (Hatori et al., 2012; Chaix et al., 2014). The striking feature of those experiments, benefit without a reduction in energy intake, is the claim that human research has since tried to reproduce.

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Interest spread quickly. A study using a smartphone application to log eating occasions suggested that many adults spread their intake across most of their waking hours and that shortening this span was feasible (Gill and Panda, 2015), and an influential review placed TRE within a wider account of the metabolic effects of fasting (de Cabo and Mattson, 2019). Trials followed in people with obesity, prediabetes and type 2 diabetes. Their results have been read both as confirmation and as refutation, often on the basis of the same papers.

This review asks a narrow question: what do recent trials show about TRE and glycaemic control, and how much of the apparent effect survives once the designs are taken into account. Weight, blood pressure and lipids are mentioned only where they bear on the interpretation of glucose outcomes.

2. Scope and Approach

This is a narrative review of published human trials together with the mechanistic work that motivates them. It is selective by intent. Trials were chosen because they are widely cited, because their design isolates a particular question, or because they enrolled people with impaired glucose regulation. No formal search strategy, screening record or pooled estimate is offered. Detailed accounts of the wider field are available elsewhere (Manoogian et al., 2022; Ezpeleta et al., 2024). The organising idea here is that each trial design removes some sources of confounding and leaves others, so that results should be compared within design classes before they are compared across them.

3. The Circadian Rationale

The physiological case for TRE rests on the daily rhythm of glucose metabolism. In healthy adults, glucose tolerance is better in the morning than in the evening, a pattern attributed to diurnal variation in insulin sensitivity and in beta cell responsiveness (Van Cauter et al., 1997). Laboratory protocols that separate the internal clock from behaviour confirm that the circadian system itself contributes to this variation and that eating and sleeping out of phase with the clock raises postprandial glucose (Scheer et al., 2009; Morris et al., 2015). A meal eaten late in the biological day is therefore handled less efficiently than the same meal eaten early.

Two predictions follow, and they are often blurred. One is that moving intake earlier should improve glycaemia regardless of the length of the window. The other is that lengthening the daily fast should help regardless of its position, through lower insulin exposure or a shift toward fat oxidation. Most free-living trials have used a window from about noon to early evening, which lengthens the fast but places intake in the later part of the day. Such a schedule is convenient and socially acceptable, but it tests the second prediction and works against the first. Figure 1 arranges the possible designs by these two properties and by whether energy intake is matched.

4. Reading the Trials by Design

Table 1 lists the principal trials discussed in this section, grouped by the comparison each makes. The sections that follow take the groups in order of increasing control over energy intake.

4.1. Uncontrolled and single-arm studies

The earliest human reports had no concurrent control. Gabel et al. (2018) compared adults with obesity who followed an eight-hour window for twelve weeks with a matched historical group and found mild weight loss and lower systolic blood pressure, with no clear change in fasting glucose or insulin. Wilkinson et al. (2020) followed people with metabolic syndrome through twelve weeks of a ten-hour window in a single-arm design and reported reductions in weight, blood pressure and atherogenic lipids. Studies of this kind establish that people can follow the regimen. They cannot separate its effect from regression to the mean, which is to be expected whenever participants are selected for elevated values (Bland and Altman, 1994), or from the general effect of enrolment and monitoring.

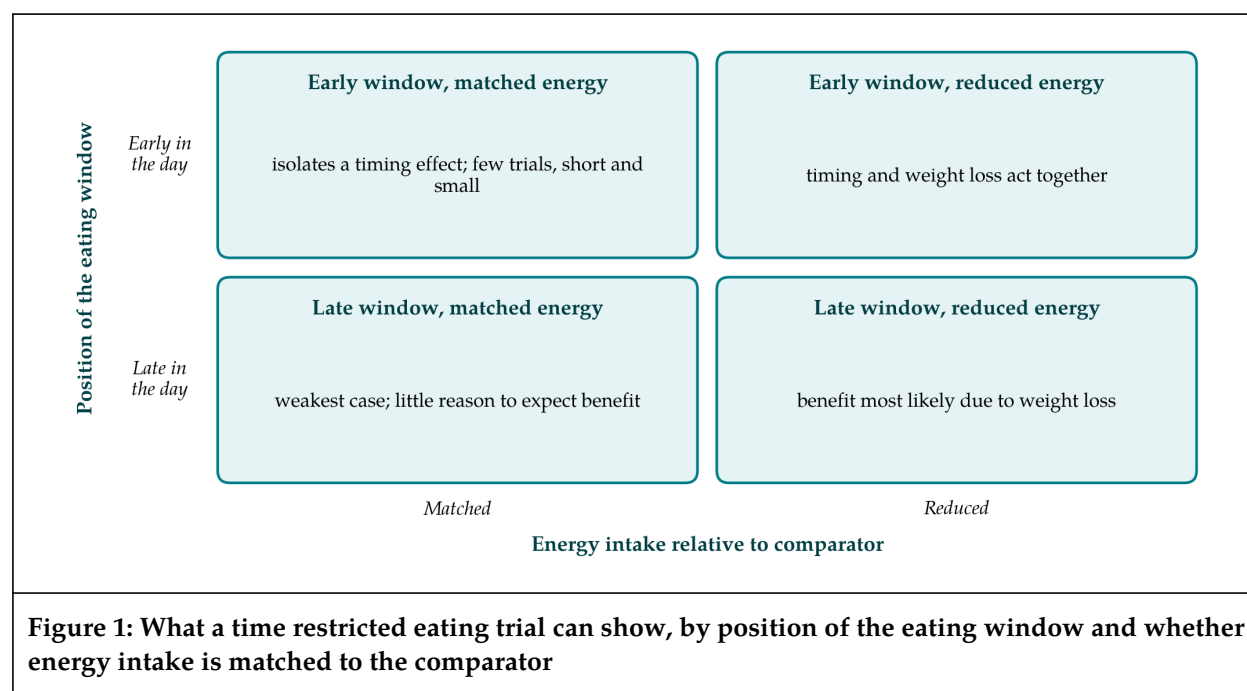


Table 1: Selected human trials of time restricted eating, grouped by what the comparison isolates			
Design class	Representative trials	What it can show	Glycaemic result in brief
Single arm or historical control	Gabel et al., 2018 ; Wilkinson et al., 2020	Feasibility; change over time	Modest or no change in glucose measures
TRE versus unrestricted eating	Cienfuegos et al., 2020 ; Lowe et al., 2020	Effect of TRE as a whole package	Mixed; follows the weight loss achieved
TRE plus energy restriction versus restriction alone	Liu et al., 2022 ; Jamshed et al., 2022	Added value of timing	No clear added glycaemic benefit
TRE versus energy restriction in type 2 diabetes	Pavlou et al., 2023	Alternative routes to lower intake	Similar fall in glycated haemoglobin
Controlled feeding, energy fixed	Sutton et al., 2018 ; Maruthur et al., 2024	Timing effect free of intake	Conflicting; one small positive, one null
Short crossover with glucose monitoring	Hutchison et al., 2019 ; Andriessen et al., 2022	Acute effects on daily glucose	Lower glucose over days to weeks

4.2. Free-living comparisons with unrestricted eating

Randomised comparisons with an unrestricted control give a mixed picture that tracks the amount of weight lost. [Cienfuegos et al. \(2020\)](#) tested four-hour and six-hour windows over eight weeks in adults with obesity; both groups spontaneously ate less, lost a few per cent of body weight and showed lower fasting insulin and insulin resistance than controls, without a change in fasting glucose or glycated haemoglobin. In the TREAT trial, [Lowe et al. \(2020\)](#) assigned adults with overweight or obesity to eat between noon and eight in the evening or to keep three structured meals. Weight loss was small and did not differ significantly between groups, glucose measures were unchanged, and a subgroup analysis raised the possibility of greater loss of lean mass with TRE. The contrast between these two trials is instructive: the narrower window produced a clear reduction in intake and a metabolic signal, the wider and later window produced neither.

4.3. Comparisons with matched energy restriction

If TRE works only by reducing intake, adding it to a prescribed energy deficit should add nothing. [Liu et al. \(2022\)](#) tested this over twelve months in adults with obesity in China, all of whom followed the same calorie-restricted diet, half with intake confined to an early eight-hour window. Weight loss, fasting glucose, two-hour postprandial glucose and insulin resistance improved in both groups to a similar degree. [Jamshed et al. \(2022\)](#) compared an early window with eating over twelve hours or more during a weight loss programme and found somewhat greater weight loss and better diastolic blood pressure with the early window, but no clear advantage in fasting glucose, insulin or glycated haemoglobin.

In adults with type 2 diabetes, [Pavlou et al. \(2023\)](#) compared an eight-hour window without calorie counting against daily calorie restriction and a control group over six months. Time restriction produced more weight loss than control, and glycated haemoglobin fell to a similar extent in the time-restricted and calorie-restricted groups. This is an important practical result, since it shows that the regimen is safe and workable in people taking glucose-lowering drugs when medication is adjusted, yet it describes equivalence of two routes to a lower intake and not a timing effect.

4.4. Controlled feeding and short crossover studies

The strongest test fixes energy intake by providing all food. [Sutton et al. \(2018\)](#) did so in a crossover study of men with prediabetes, comparing a six-hour window ending in mid-afternoon with a twelve-hour schedule for five weeks each. Weight was held constant, and the early window improved insulin sensitivity, beta cell responsiveness and blood pressure. The study is the main support for a weight-independent effect, and it involved only a handful of participants, all male. A related study by the same group found lower 24-hour glucose and smaller glycaemic excursions after four days of early time restriction ([Jamshed et al., 2019](#)).

A larger controlled feeding trial did not confirm the finding. [Maruthur et al. \(2024\)](#) provided isocaloric diets for twelve weeks to adults with obesity and prediabetes or diet-controlled diabetes, with most energy eaten early in a ten-hour window or spread across a usual pattern, and observed no difference in weight or in measures of glucose homeostasis. Short crossover studies using continuous glucose monitoring sit between these results. [Hutchison et al. \(2019\)](#) found improved glucose tolerance after one week of a nine-hour window in men at risk of diabetes whether the window was early or delayed, and [Andriessen et al. \(2022\)](#) found lower 24-hour glucose after three weeks of a ten-hour window in adults with type 2 diabetes without any improvement in insulin sensitivity. In neither was energy intake fully controlled.

5. Sources of Noise

Several features of this literature make individual results unreliable guides. Samples are small and durations short in exactly the designs that could isolate a timing effect, because controlled feeding is expensive. Adherence to the window is usually self-reported, and energy intake in free-living arms is estimated by food records whose errors are larger than the effects sought. The definition of the intervention varies in window length, clock position and whether energy is prescribed, so that trials bearing the same label test different hypotheses.

Outcome measures add further scatter. Fasting glucose, glycated haemoglobin, clamp-derived insulin sensitivity, oral glucose tolerance and sensor-derived mean glucose respond over different time scales. Glycated haemoglobin reflects average glucose over the preceding two to three months ([Nathan et al., 2008](#)) and cannot register a change in a study lasting a few weeks. Fasting samples taken after a longer overnight fast in the intervention arm than in the control arm are not strictly comparable. In trials enrolling people with diabetes, reductions in medication made for safety can mask an effect or, if unrecorded, create an apparent one. Finally, many trials report numerous metabolic outcomes without a prespecified glycaemic primary endpoint, which invites selective emphasis.

6. What Can Be Said, and Gaps That Remain

Taken by design class, the trials support three statements. Time restricted eating often leads people to eat less without counting calories, and when it does, weight and glycaemic markers improve to about the extent expected from the weight change. When energy intake is matched by prescription, the addition of a time window has not improved glycaemic control in the larger and longer trials. Evidence for a weight-independent benefit of an early window exists but rests on small, short feeding studies and is contradicted by a larger one. Current nutrition guidance for diabetes, which treats a range of eating patterns as acceptable and emphasises the one a person can sustain, is consistent with this reading (Evert et al., 2019).

The gaps are specific. An adequately powered controlled feeding trial comparing early and late windows of equal length, with a prespecified glycaemic endpoint, would settle whether clock position matters. Trials in type 2 diabetes need systematic recording of medication changes and hypoglycaemia. Objective measurement of adherence, for example by continuous glucose monitoring or timestamped photographs, should replace diaries. Follow-up beyond a year is almost absent, as are data on older adults, shift workers and people using insulin, for whom the balance of benefit and risk may differ.

7. Conclusion

The signal in recent trials of time restricted eating is that it offers one more way to lower energy intake, with glycaemic benefits in proportion. The noise comes from short, small and heterogeneous studies that have been read as evidence for something more. A specific effect of meal timing on glucose regulation is biologically plausible and has been seen under tightly controlled conditions, but it has not been reproduced at scale. Clinicians can offer the approach to people who find it easier than calorie counting, with attention to medication in those with diabetes, while describing its advantages over other methods of energy restriction as unproven.

Conflict of interest

To be declared by the author(s) before publication.

Funding

To be declared by the author(s) before publication.

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