

Post-Meal Walking as A Strategy for Postprandial Glycemic Control After High-Carbohydrate Meals in Type 2 Diabetes Mellitus: A Literature Review of Timing, Duration, & Intensity

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ABSTRACT

Type 2 diabetes mellitus (T2DM) is characterized by impaired postprandial glycemic control, a condition of particular concern in Asian populations, including Indonesia, where rice serves as the dominant carbohydrate source and carries a high glycemic index. Postprandial exercise, particularly walking, has been proposed as a practical, low-cost, non-pharmacological strategy to attenuate glucose excursions after meals, yet no prior narrative review has specifically synthesized evidence on post-meal walking in relation to high-carbohydrate, rice-based diets. This narrative review aimed to synthesize existing evidence on the effectiveness, optimal timing, duration, and intensity of post-meal walking for glycemic control in individuals with T2DM, and to evaluate its relevance to high-carbohydrate meal contexts. Literature was searched through PubMed, Scopus, ScienceDirect, and Google Scholar for studies published between 2016 and 2026, yielding ten randomized controlled trials that met the inclusion criteria. Findings showed that post-meal walking consistently reduced postprandial glucose excursions, peak glucose, and area under the curve, with the most consistent benefits observed when walking was initiated within 15-30 minutes after meals, for 10-20 minutes, at light-to-moderate intensity. These effects are physiologically explained by insulin-independent GLUT4 translocation induced by muscle contraction. Compared with other modalities such as resistance training, cycling, high-intensity interval training, and spot marching, walking remained the most broadly applicable option due to its safety, accessibility, and minimal equipment requirements. However, no included study used rice as the test meal or was conducted in Indonesian or Southeast Asian populations, indicating that current recommendations remain extrapolated and warrant direct investigation in rice-consuming populations.

Keywords: post-meal walking; postprandial glycemia; type 2 diabetes mellitus; high-carbohydrate diet; rice; glycemic control; exercise training

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has become a serious global health threat, accounting for more than 90% of all diabetes cases worldwide and imposing a very heavy socioeconomic burden on patients and healthcare systems [1]. Pathophysiologically, the condition is characterized by peripheral insulin resistance accompanied by a progressive decline in pancreatic beta-cell secretory function [2]. A hallmark feature frequently seen in individuals with T2DM is the body's inability to control postprandial hyperglycemia, in which plasma glucose levels often rise above the safe target

of 10.0 mmol/L (180 mg/dL) within 1–2 hours after eating [1]. These glucose excursions, or glycemic spikes, not only contribute directly to elevated HbA1c levels but also trigger oxidative stress, vascular inflammation, and glucotoxicity that accelerate the onset of microvascular and macrovascular complications [3].

In Indonesia, the burden of T2DM has risen sharply over the past decade. Basic Health Research (Riskesdas) data recorded an increase in diabetes prevalence among adults from 1.5% in 2013 to 2.0% in 2018 [4].

This increase is further evident in reports from the International Diabetes Federation (IDF), which noted a surge in prevalence from 7% (equivalent to 19.5 million cases) in 2021 [5] to 11.3% (equivalent to 20.42 million cases) in 2024 [6], placing Indonesia among the ten countries with the highest number of T2DM cases in the world [7]. This burden is also reflected in mortality, with diabetes ranking as the third leading cause of death nationally in 2019 [5].

The problem of postprandial glucose spikes is especially critical for people in Asia, particularly Indonesia, because these high figures are closely related to a daily dietary pattern dominated by high carbohydrate intake. As the main staple food, refined white rice consumed daily has a high glycemic index (GI) (~80) and contributes more than 60% of total daily energy intake [1]. This diet, high in refined carbohydrates and relatively low in fiber and protein, causes very rapid digestion and absorption of glucose in the small intestine, triggering a sharp and persistent rise in blood glucose after meals [8]. Therefore, controlling postprandial glucose excursions through non-pharmacological interventions has become a very important pillar in the day-to-day management of T2DM.

One of the most physiologically rational lifestyle strategies to address this problem is physical activity after meals (postprandial exercise). At the molecular level, glucose uptake by skeletal muscle during activity can occur through a pathway that does not depend on insulin (insulin-independent pathway) [10][11]. Repeated contraction of the leg muscles during activity increases intracellular calcium and the AMP/ATP ratio, which in turn activates the protein kinases CaMKII and AMPK [11]. This pathway phosphorylates the AS160 protein and drives translocation of Glucose Transporter 4 (GLUT4) vesicles directly to the muscle cell membrane without passing through the insulin signaling that is impaired in people with T2DM [12]. In other words, postprandial physical activity can bypass the barrier of insulin resistance and draw glucose directly from the bloodstream to be used as energy [13].

Various postprandial exercise modalities have been studied for their effectiveness. However, walking has become the main focus because it is safe, inexpensive, and easy to perform. Resistance exercise, cycling, and interrupting sitting time with periodic light movement (breaks from sitting) have been shown to suppress postprandial glucose excursions and insulin responses [14][15][16]. Resistance exercise exploits the recruitment of large muscle mass to absorb glucose, while periodic sitting interruptions effectively prevent glucose accumulation caused by sedentary behavior [14]. That said, these non-walking modalities often have limitations in practicality and long-term adherence; resistance exercise requires equipment or technique supervision, while interrupting sitting every 30 minutes is difficult to implement in rigid work environments [17]. Therefore, post-meal walking is

the main focus of this review because it has a high safety profile, requires no special equipment, and is easily integrated after main mealtimes, making it highly applicable for T2DM patients of various fitness levels.

To date, most randomized controlled trials (RCTs) evaluating the timing of post-meal exercise still use standard Western test meals, such as bread, cereal, biscuits, or pure glucose solution. There has been no scientific review that comprehensively synthesizes the evidence on the effectiveness, ideal timing, and duration of post-meal walking tailored specifically to the context of consuming high-carbohydrate, high-GI foods such as white rice, especially in the Indonesian population, which faces such a substantial T2DM prevalence burden. This narrative review was prepared to bridge that knowledge gap, in order to formulate practical guidance on the timing and dose of post-meal walking that is relevant and applicable in the daily lives of T2DM patients.

MATERIALS AND METHODS

This work uses a narrative review approach to discuss the effectiveness of post-meal walking, including its duration, intensity, and timing, on glycemic responses in patients with Type 2 Diabetes Mellitus (T2DM). A narrative approach was used because there is considerable variation between studies in participant characteristics, walking protocols, timing after meals, test meal composition, and the glycemic parameters used as outcomes. Therefore, the results were synthesized narratively by comparing the intervention characteristics and main findings of each study.

The literature search was conducted through several electronic databases, namely PubMed, Scopus, ScienceDirect, and Google Scholar, to identify articles published in the last 10 years, from 2016 to 2026. The search strategy used a combination of keywords and Boolean operators covering post-meal walking, postprandial glucose, high-carbohydrate meal, glycemic response, timing, exercise duration, and mechanisms of glucose uptake by muscle.

Articles considered for the main synthesis were studies in adult humans with T2DM, primarily randomized controlled trials (RCTs), that evaluated post-meal walking and reported at least one glycemic parameter, such as AUC/iAUC, peak glucose, glucose excursion, or HbA1c. Animal or in vitro studies, populations without T2DM, studies whose main intervention was other than walking, and articles that did not provide full text were excluded. A total of 10 experimental studies meeting the inclusion criteria were selected for extraction and narrative synthesis. The extracted data included author and year of publication, study design, population characteristics and sample size, type and composition of carbohydrate (CHO) in the test meal, timing of exercise after the meal, duration and intensity of exercise, and outcomes and main findings. The extraction results of the ten studies are

presented in Table 1 to provide a comparison of the evidence on the effectiveness, timing, duration, and intensity of post-meal walking on the glycemic response in T2DM patients. For the graphical comparison of timing, duration, and intensity (Figures 2–4), we used the five studies that reported group means with a measure of dispersion for a glucose-exposure outcome and a control condition. Values were expressed as a percentage of each study's control mean so that different outcome measures could be compared. The remaining studies are described narratively.

RESULTS AND DISCUSSION

Postprandial Glycemic Response After High-Carbohydrate Meals

Postprandial hyperglycemia refers to the condition in which postprandial plasma glucose fails to fall below the standard safe threshold of the American Diabetes Association, namely 10.0 mmol/L (180 mg/dL), within 1–2 hours after a meal [1]. This condition arises from a disrupted balance between the insulin secretory capacity of pancreatic β -cells and the efficiency of glucose uptake by peripheral tissues [2]; in people with type 2 diabetes mellitus, it is aggravated by the combination of peripheral insulin resistance and a progressive decline in insulin secretion [3]. The magnitude of the postprandial glucose spike is determined by three main factors: the amount, rate, and extent of carbohydrate digestion, the insulin secretory response, and the rate of gastric emptying [8]. These factors are further affected by impaired early-phase insulin secretion [1] and a diminished response of incretin hormones such as GLP-1 and GIP, which normally enhance postprandial insulin secretion [2]. Besides the amount of carbohydrate consumed, the source and quality of the carbohydrate also determine the magnitude of the glycemic response, as reflected in the Glycemic Index (GI) and Glycemic Load (GL) of the food [18]. Refined staple foods such as refined white rice (GI ~80) and wheat-based noodles (GI ~74) are digested and absorbed rapidly, resulting in sharp glucose spikes [1]. Carbohydrate sources rich in resistant or slowly digestible starch, such as parboiled rice and brown rice, promote slower glucose release and lower glucose AUC [8]. Meal composition also plays a role: the presence of fiber, protein, and fat slows gastric emptying and intestinal glucose absorption, so replacing purely high-carbohydrate foods with legume-based meals or diabetes-specific formulas has been shown to suppress glucose spikes compared with refined staple foods [1]. A similar principle underlies meal sequencing, that is, consuming non-carbohydrate components several minutes before the carbohydrate, which has been shown to blunt the postprandial rise in glucose through stimulation of incretin hormones and slowing of gastric motility [19].

In this narrative review, the included studies were selected to specifically examine post-meal walking following meals containing a high proportion of carbohydrates. Based on the classification of the USDA and Health and Human Services Dietary Guidelines Advisory Committee, carbohydrate

intake of 45%–64% of total energy is categorized as a high-carbohydrate diet, whereas intake of $\geq 65\%$ is categorized as a very-high-carbohydrate diet [36]. Therefore, this review included studies investigating meals or dietary conditions providing $>45\%$ of total energy from carbohydrates, encompassing both high-carbohydrate (45%–64%) and very-high-carbohydrate ($\geq 65\%$) conditions. Among the ten studies included in this review, all met this carbohydrate-content criterion. This approach was applied to maintain consistency in the dietary context and to specifically evaluate the effect of post-meal walking on glycemic responses following carbohydrate-dense meals.

The clinical significance of these postprandial glucose spikes cannot be ignored, because recurrent postprandial hyperglycemia is an independent risk factor for diabetic complications and cardiometabolic risk [1], consistent with evidence that the development of diabetic complications is more closely related to postprandial glucose fluctuations than to chronic hyperglycemia alone [20]. Metabolically, repeated glucose fluctuations increase non-esterified free fatty acids and reduce the number of insulin receptors, which in turn worsens insulin resistance [21]. Postprandial glucose excursions make a dominant contribution to HbA1c, reaching 70% or more in patients with relatively good glycemic control (HbA1c $<7.3\%$) [3]. Suppression of postprandial glucose spikes contributes directly to improved long-term glycemic control [3][1].

This significance becomes even more important for Asian populations, including Indonesia, given that the projected increase in diabetes prevalence in Southeast Asia is among the highest globally [1]. White rice and noodles are common staple foods in Asian populations and have a high glycemic index (refined white rice ~80). Carbohydrates contribute almost 60% of total daily energy intake in Asian populations. Asia has the highest dietary glycemic load worldwide [1]. High white rice consumption is consistently positively associated with an increased risk of type 2 diabetes mellitus and metabolic syndrome, with the most significant risk impact found in Asian populations [9]. A typical Asian breakfast has even been recorded as having a higher GI value than a typical Western breakfast, owing to the predominance of refined rice flour and the low proportion of fat, protein, and fiber in the dishes [1]. These findings underscore that strategies for controlling postprandial glucose need to be examined specifically in the context of rice-based, high-carbohydrate diets, rather than simply extrapolated from studies in populations with different dietary patterns.

Mechanisms Underlying Exercise-Induced Glucose Uptake

Skeletal muscle is the body's largest glucose consumer, and glucose uptake from the bloodstream into muscle cells is facilitated by the carrier protein Glucose Transporter 4 (GLUT4) via two signaling pathways that operate independently of each other (Figure 1).

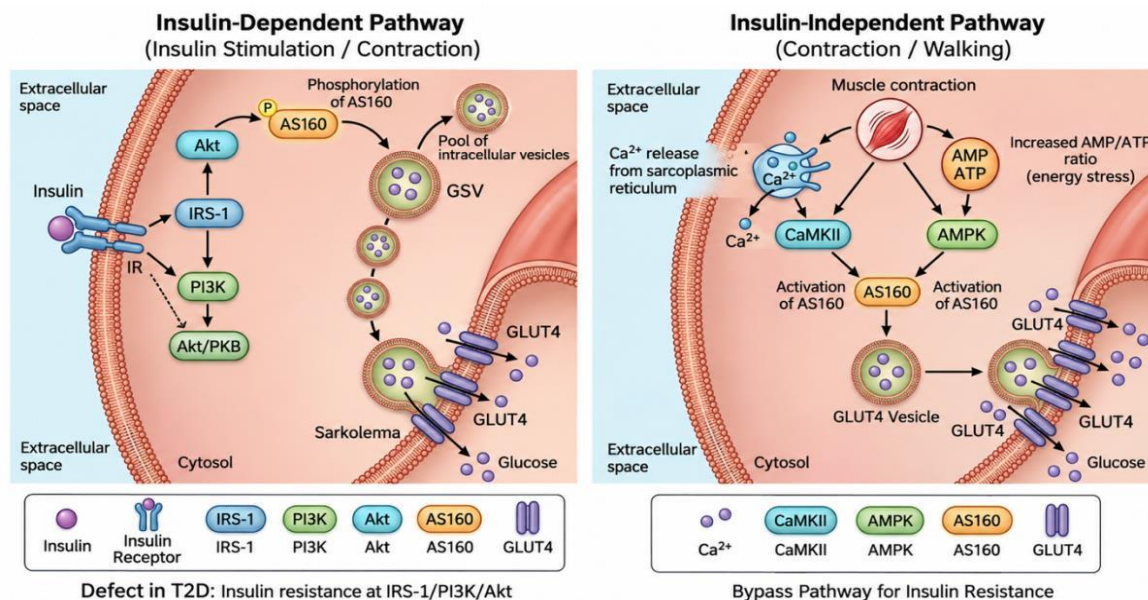


FIGURE 1: Insulin-dependent (left) versus insulin-independent, contraction-mediated (right) pathways of GLUT4-facilitated glucose uptake in skeletal muscle. AI-assisted illustration based on Petersen & Shulman [22], Sylow et al. [10], and Joseph et al. [11].

The first, the insulin-dependent pathway, is activated when insulin binds to its receptor on the muscle cell membrane, triggering a phosphorylation cascade involving Insulin Receptor Substrate-1 (IRS-1), phosphoinositide 3-kinase (PI3K), and Akt/Protein Kinase B, which ultimately phosphorylates the AS160 protein and drives GLUT4 storage vesicles to fuse with the cell membrane [22]. Second, the insulin-independent pathway is activated by muscle contraction itself without any need for insulin: contraction triggers the release of calcium from the sarcoplasmic reticulum, which activates CaMKII, and raises the intracellular AMP/ATP ratio, which activates AMPK [10][11]. Both kinases phosphorylate AS160 at sites different from those of the insulin pathway, yet converge on the same result, namely translocation of GLUT4 to the cell membrane [12].

When a person walks, repeated contraction of the lower-leg muscles (particularly the quadriceps and gastrocnemius) keeps AMPK and CaMKII activation going continuously throughout the activity. This condition drives GLUT4 vesicles to move from the intracellular compartment to the cell membrane, so that the capacity for glucose transport into the muscle increases significantly compared with resting conditions, and this increase persists for several hours after the activity ends [10]. This characteristic explains why the glucose-lowering effect of walking is not merely momentary but can continue to be felt even after the activity has stopped.

The relevance of this mechanism for people with type 2 diabetes mellitus lies in its ability to bypass impaired insulin signaling. Insulin resistance in people with T2D is generally caused by defects in the early segment of the insulin pathway, such as impaired function of IRS-1 or PI3K/Akt due to the accumulation of ectopic lipids in muscle tissue [22]. Because the muscle contraction pathway does not pass through the IRS-1/PI3K/Akt cascade at all and

directly activates AS160 via AMPK and CaMKII, contraction-induced GLUT4 translocation can proceed normally even when the insulin pathway is severely impaired [11]. This is the fundamental reason why blood glucose still needs to be actively lowered in T2D patients: persistent, untreated hyperglycemia triggers glucotoxicity in the remaining pancreatic beta cells and the formation of advanced glycation end-products that provoke vascular inflammation, and it accelerates microvascular and macrovascular complications such as nephropathy, retinopathy, and cardiovascular disease [1].

This mechanistic understanding explains why post-meal walking controls blood glucose, as supported by clinical evidence in the next section. Because postprandial glucose peaks within 30–60 minutes after a meal, walking during this window triggers AMPK and CaMKII activation when extracellular glucose is highest [13]. This muscle contraction pathway bypasses insulin resistance, facilitating direct glucose uptake into muscle cells for oxidation without requiring extra insulin secretion [10]. Consequently, glucose clearance accelerates, exerting an insulin-sparing effect that reduces postprandial hyperinsulinemia and pancreatic beta-cell workload [10]. This mechanism forms the biological foundation for the clinical findings on walking timing, duration, and intensity discussed below.

Evidence for Post-Meal Walking in Type 2 Diabetes

Based on the physiological mechanisms described in the previous section, a number of studies have evaluated the effectiveness of post-meal walking in helping to control the glycemic response in individuals with type 2 diabetes (T2D); these are summarized in the table below.

TABLE 1: Evidence for Post-Meal Walking in Type 2 Diabetes.

Author & Year	Study Design	Population & Sample	Meal Type & CHO Composition	Timing After Meal	Exercise Duration & Intensity	Outcome & Key Findings
Pahra et al. (2017) [23]	RCT crossover	n=64, T2D	Main meals; ~50% CHO, 30% fat, 20% protein	15 min after each meal	Brisk walking 15 min ×3; total 45 min/day; moderate intensity (3.3 MET, ±4.8 km/h)	HbA1c -0.7% vs +0.3% control (p<0.001); 5-point glucose profile improved (p<0.001). Post-meal walking improved glycemic control compared to a single bout of exercise.
Reynolds et al. (2016) [13]	RCT crossover	n=41, T2D	Habitual diet; daily CHO ±212–219 g, highest at dinner	5 min after each meal	Walking 10 min ×3; total 30 min/day vs. 30 min in one session; light-to-moderate intensity	3-h glucose iAUC decreased by 12% (p=0.031); after dinner, decreased by 22% (p=0.001). Greater effect after dinner, when CHO intake was highest.
Blankenship et al. (2019) [17]	RCT crossover	n=30, T2D	3 isocaloric meals; 55.4% CHO, 29.3% fat, 15.8% protein	30–60 min after breakfast	Brisk walking 20, 40, or 60 min; low-to-moderate intensity	Post-breakfast PPG decreased significantly (p≤0.05); daily hyperglycemia duration not significant (p=0.0875). No dose-response relationship by duration.
Li et al. (2018) [24]	RCT crossover	n=29, T2D	3 equal meals; 55% CHO, 18% protein, 27% fat	30 min after dinner	Walking 20 min; moderate intensity (~40% HRR; HR 111.0±7.9 bpm)	2-h glucose spike: 1.9±1.3 vs. 2.7±1.4 mmol/L (p=0.04); peak glucose: 9.3±1.6 vs. 10.3±2.3 mmol/L (p=0.02). 12-h glucose variability decreased.
Dempsey et al. (2016) [14]	RCT crossover	n=24, T2D	Breakfast & lunch; 50% CHO, 30% fat, 20% protein	Immediately postprandial; every 30 min for 7 h	Light walking 3 min/bout; 12 bouts; light intensity (3.2 km/h)	7-h glucose iAUC decreased by 39% and insulin iAUC by 26% (both p<0.001). Periodic light walking suppressed glucose and insulin responses throughout the day.
Bravo-Garcia et al. (2024) [25]	RCT crossover	n=11, T2D	Standard breakfast; 50% CHO, 30% fat, 20% protein	30–60 min after meal	Brisk walking 20 min; low-to-moderate intensity	2-h glucose iAUC decreased under early (d=0.35) and delayed (d=0.37) conditions, but not mid. Effect varied by meal timing and baseline glycemia.
Iida et al. (2020) [26]	RCT crossover	n=11, T2D	Main meals; 50% CHO, 30% fat, 20% protein	30 min after each meal	Walking 15 min ×3; total 45 min/day; moderate intensity (40% HRR)	24-h glucose SD decreased from 27.8 to 22.9 mg/dL (p=0.014). After Bonferroni correction, the effect remained significant mainly after breakfast (p=0.010).
Bellini et al. (2021) [27]	RCT crossover	n=8, T2D	Breakfast; 66% CHO, 19% fat, 15% protein; 310 kcal	30 min after meal	Walking 30 min; compared with 15-min resistance exercise and combined aerobic-resistance exercise; moderate intensity (100 steps/min)	Peak glucose and iAUC 0–120/180 min decreased across all modalities vs. control (p<0.048). Greatest reduction in peak glucose with walking alone and walking-resistance combination.
Terada et al. (2016) [15]	RCT crossover	n=10, T2D	Breakfast; 600±57 kcal; 50% CHO, 20% protein, 30% fat	60 min after meal	Walking 60 min; moderate intensity (55% VO ₂ peak)	120-min glucose AUC: 240±183 vs. 355±66 mmol·min·L ⁻¹ (post-meal vs. control). Post-meal walking reduced AUC, but the timing effect was not fully consistent.
Nygaard et al. (2017) [28]	RCT crossover	n=12, T2D	Semi-skimmed milk and cornflakes breakfast; 380±48 kcal; 74.9 g CHO	15 min after meal	Walking 60 min; moderate intensity (8% incline; RPE 12)	210-min glucose AUC: 1477±105 vs. 1576±199 mmol·min·L ⁻¹ (post-meal vs. control). Post-meal walking reduced postprandial glucose exposure.

Of the ten studies summarized in the table, almost all agree on one direction of findings: walking after meals suppresses the blood glucose response in people with type 2 diabetes mellitus, whether seen as a reduction in peak glucose, glucose excursion, or area under the curve (AUC/iAUC). Nygaard et al. [28] and Terada et al. [15], for example, both recorded lower glucose AUC in the walking condition than in the control, even though both used a single, fairly long (60-minute) walking session. A similar pattern was also seen in studies with more varied designs. Blankenship et al. [17] reported reduced postprandial glucose after breakfast across various walking durations (20, 40, and 60 minutes), although no clear dose-response relationship was found, meaning that increasing the duration does not automatically provide a multiplied benefit. Dempsey et al. [14] even showed that walking does not need to be long to have a large effect; 3-minute sessions repeated every 30 minutes over 7 hours reduced glucose iAUC by up to 39% compared with continuous sitting, indicating that the frequency and consistency of sitting interruptions can be as important as a single long duration.

The benefits of walking after meals are also consistently seen when it is performed repeatedly throughout the day following the main meal schedule. Reynolds et al. [13] found that 10 minutes of walking after breakfast, lunch, and dinner reduced the 3-hour glucose iAUC by 12% compared with the control [13]. The reduction reached 22% after dinner, the meal associated with the highest carbohydrate intake in that study. This mealtime-dependent effect also appeared in Bravo-Garcia et al. [25], who found a meaningful reduction in glucose iAUC when walking was performed after the early-morning and midday breakfasts, but not after the 09:30 breakfast, because the baseline glucose spikes at that hour were already low. In other words, the benefit of walking appears more evident precisely at mealtimes with a higher glycemic load. This pattern is relevant to high-carbohydrate foods such as rice.

In addition to acute effects, several studies also evaluated the impact on broader and more sustained glycemic control. Pahra et al. [23] recorded a 0.7% reduction in HbA1c in the group that walked for 15 minutes after each meal, in contrast to a 0.3% increase in the control group, which walked only once for 45 minutes before breakfast. Iida et al. [26] reinforced this finding from the standpoint of daily glucose variability, in which 15 minutes of walking after each meal significantly reduced the 24-hour glucose standard deviation, although after strict statistical correction (Bonferroni) the effect remained significant only for the post-breakfast session. Li et al. [24] also showed consistent results from a different angle: 20 minutes of moderate-intensity walking after dinner suppressed both the 2-hour glucose spike and 12-hour glucose variability without triggering nocturnal hypoglycemic episodes, an important point indicating that this strategy is relatively safe. Interestingly, the benefit of walking is not limited to its use as a stand-alone modality. Bellini et al. [27],

who compared walking with resistance exercise and a combination of both, found that all exercise modes reduced glucose excursions compared with sitting, but the greatest reduction in peak glucose occurred when walking was performed alone or placed first, before resistance exercise. This finding indicates that walking is not merely one option among many but appears to be the most time- and outcome-efficient modality compared with other, more complex alternatives.

Although the direction of findings is generally consistent in supporting the benefits of walking, the available evidence still leaves heterogeneity that needs careful attention. Variation was found in the type and composition of the test meals (from standard foods to foods with 50–66% of energy from carbohydrate), the timing of walking initiation (5–60 minutes after a meal), the duration and intensity of exercise (10–60 minutes, light to moderate intensity), and the type of glycemic outcome measured, including peak glucose, excursions, and AUC/iAUC to glucose variability and HbA1c. In terms of design, all studies used randomized crossover trials with small to moderate sample sizes (ranging from 9 to 64 subjects), and most evaluated only acute effects over one to several days of observation rather than long-term effects.

Among the ten studies, Reynolds et al. [13] and Li et al. [24] are the most relevant to the context of high-carbohydrate meals. Reynolds et al. involved 41 T2D patients with a daily carbohydrate intake of about 212–219 g, and showed that brief 10-minute walks after each meal were more effective at reducing the 3-hour glucose iAUC than a single 30-minute session at a free time, with the greatest benefit at the mealtime with the highest carbohydrate intake. Li et al. reinforced this pattern in patients with a carbohydrate proportion of 55% of total energy, in whom 20 minutes of moderate-intensity walking after dinner significantly suppressed the 2-hour glucose spike. Both studies give an early signal that the benefit of walking tends to be greater precisely at meals with a high carbohydrate load, a finding that supports the relevance of this strategy for populations with high-carbohydrate dietary patterns, such as rice consumption in Indonesia.

However, one important gap must be underlined: of all the reviewed studies, not a single one specifically used rice as the main carbohydrate source in the test meal. Yet rice is the staple food for most of the Asian population, including Indonesia, and the glycemic response to this carbohydrate source has its own clinical relevance for that population [29]. This gap is one of the main research gaps that subsequent studies need to address, particularly in the context of rice consumption and Southeast Asian populations.

Overall, the available evidence is fairly convincing that post-meal walking is an effective strategy for suppressing the postprandial glucose response in people with type 2 diabetes mellitus. Nevertheless, the magnitude of the benefit appears to be influenced by the characteristics of the food consumed, the duration

and intensity of walking, and precisely when the activity begins after eating [30]. This pattern is discussed further in the next section, focusing on the most optimal time to start walking after a meal.

Timing of Post-Meal Walking

Timing refers to the interval between a meal and the start of walking. Across the published studies, this interval ranged from immediately after eating to 60 minutes or more afterward [31]. Because no universal cut-off separates immediate from delayed post-meal exercise, this review sorts initiation times into three groups: early (≤ 30 minutes), intermediate (>30 –60 minutes), and delayed (>60 minutes).

Seven of the ten studies began walking within 30 minutes of the meal, and each reported that this early start helped dampen the postprandial glucose rise. In Reynolds et al. [13] (41 participants with T2D), a 10-minute walk beginning 5 minutes after each meal lowered the 3-hour glucose iAUC by 12% compared with a single 30-minute walk at an unspecified time. Pahra et al. [23] found a similar advantage: three 15-minute brisk walks, each started 15 minutes after a meal, improved the 5-point glucose profile and HbA1c more than one 45-minute walk before breakfast. Dempsey et al. [14] took a different approach, using 3-minute light walks every 30 minutes from right after eating, which reduced glucose iAUC by up to 39% and lowered insulin concentrations. The benefit of an early start also extended to glucose stability over longer periods. Iida et al. [26] reported lower 24-hour glucose variability after 15-minute walks began 30 minutes after meals, and Li et al. [24] observed a smaller 2-hour glucose spike after a 20-minute walk starting 30 minutes after the first bite of dinner, with nocturnal hypoglycemia. Longer walks that still began within this window were effective as well: Bellini et al. [27] (30 minutes of walking, started 30 minutes after eating) and Nygaard et al. [28] (60 minutes of walking, started 15 minutes after eating) both reported smaller postprandial glucose excursions.

Walking that began 30–60 minutes after the meal was still effective, but the response depended more on which meal was eaten and on individual differences. Blankenship et al. [17] found that 20–60 minutes of brisk walking in this window significantly lowered mean glucose after breakfast and tended to shorten the daily duration of hyperglycemia, although the latter did not reach statistical significance ($p=0.0875$). Bravo-Garcia et al. [25] placed 20 minutes of walking 30–60 minutes after breakfast and saw a meaningful reduction in 2-hour glucose iAUC when breakfast was eaten early in the morning or at midday, but not at 09:30. This suggests that the benefit of timing also depends on the size of the baseline glucose spike, not only on the interval itself. Terada et al. [15] began walking exactly 60 minutes after breakfast and still recorded a lower glucose AUC than control, but the reduction was smaller than with exercise before the meal in the same study. This is consistent with the idea that a later start may miss part of the early glucose rise.

To compare initiation times side by side, results from five studies with comparable glucose data were normalized as a percentage of control (100%) in Figure 2. Post-meal walking lowered glucose exposure across all tested initiation times, from immediately post-meal to 60 minutes later [13][14][15][24][28]. Four studies starting within 30 minutes showed reductions of 6%–39%, while the single study starting at 60 minutes reported a 32% reduction [15]. Because reduction size did not increase monotonically with earlier initiation, variations across studies likely reflect differences in meal composition, walking duration, and outcome measures rather than timing alone. Instead, the case for early initiation relies on broader evidence in Table 1: seven of ten trials began walking within 30 minutes, whereas partial or conditional findings were more common when walking began 30–60 minutes post-meal [15][17][25]. Thus, Figure 2 alone does not prove that earlier initiation maximizes glucose reduction. Furthermore, because no included trial started after 60 minutes, the impact of longer delays remains unknown.

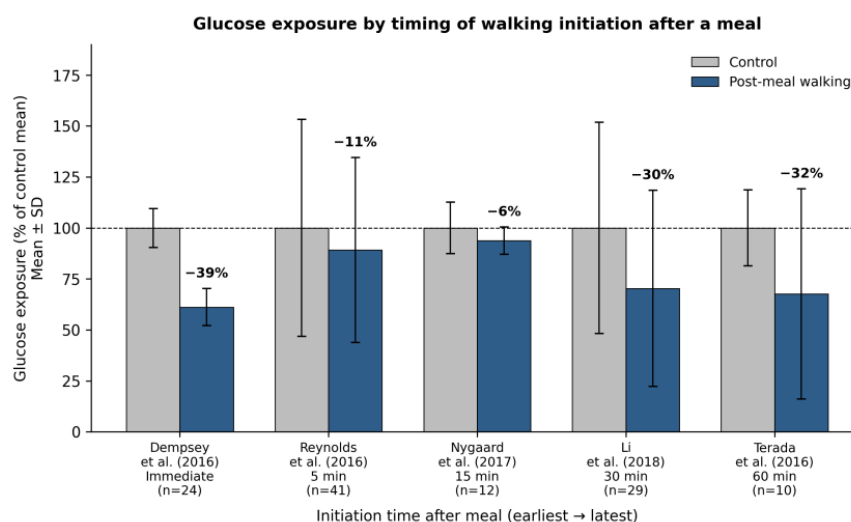


FIGURE 2: Glucose exposure by timing of walking initiation after a meal. Bars show group means $\pm$ SD, normalized to each study's control mean (control = 100%); the value above each walking bar is the relative reduction versus control. Studies are ordered from the earliest to the latest initiation time.

Taken together, the ten studies indicate that starting to walk within 30 minutes of a meal, especially 15–30 minutes after eating, is the most consistently supported option for glycemic control in T2DM. There is also a physiological reason for this timing: walking in this window coincides with the early rise in blood glucose, when muscle contraction triggers insulin-independent GLUT4 translocation and lets the muscle take up glucose as fuel before it reaches its peak [32]. An early start is also practical [13]. A short walk of 10–15 minutes right after each main meal is much easier to fit into a daily routine than one long session at an unspecified time, which may make it easier to sustain, while still helping to blunt glucose spikes after high-carbohydrate meals.

Duration and Intensity of Post-Meal Walking

Duration is how long a single walking session lasts (in minutes), and intensity is how hard the body works during the walk. Intensity is usually graded as light, moderate, or vigorous, using measures such as METs, the percentage of heart rate reserve (HRR), or perceived effort on the Borg scale (RPE). Following the American College of Sports Medicine (ACSM) classification [33], light activity is below 3 METs, below 40% HRR, or below 12 on the RPE scale, and a person can still talk normally. Moderate activity is 3–5.9 METs, 40–59% HRR, or an RPE of 12–13. Breathing quickens and sweating begins, but short sentences are still possible, and brisk walking at about 4.8–6.4 km/h usually falls in this range. Vigorous activity is 6 METs or more, 60% HRR or more, or an RPE of 14 or higher, where speech is limited to a word or two. This grading matters because the studies in this review used very different walking doses, and both duration and intensity may shape the glycemic benefit. We look first at duration and then at intensity.

Walking sessions ranged from 3 minutes repeated through the day to 60 minutes continuously, but longer sessions did not always give greater benefit. Dempsey et al. [14] showed that 3 minutes of light walking every 30 minutes lowered glucose iAUC

significantly compared with uninterrupted sitting (14.8 vs 24.2 mmol·h/L), so even very short but repeated bouts of muscle activity have a meaningful effect. The clearest evidence came from sessions of 10–20 minutes. Reynolds et al. [13] found that 10 minutes of walking after each meal lowered the 3-hour glucose iAUC by about 12%, with the largest effect (22%) after dinner. Pahra et al. [23] reported that 15 minutes of brisk walking after each meal improved the glucose profile and HbA1c more than one 45-minute session before breakfast, and Iida et al. [26] found that 15 minutes of moderate walking improved 24-hour glucose variability. Twenty minutes was also beneficial in Li et al. [24] and Bravo-Garcia et al. [25], although in the latter the effect depended on the time of breakfast. Sessions of 30–60 minutes were helpful too but did not show a consistent dose–response pattern. Bellini et al. [27] recorded a significant reduction in glycemic excursion after 30 minutes of walking, whereas Blankenship et al. [17] found no added advantage when sessions were extended from 20 to 40 or 60 minutes, and Nygaard et al. [28] noted that 60 minutes of walking reduced peak glucose but did not significantly lower total glucose AUC.

To compare session lengths side by side, the five studies with comparable glucose data are shown in order of duration in Figure 3. Post-meal walking lowered glucose exposure relative to control in every study, from 3-minute bouts to 60-minute sessions [13][14][15][24][28]. Reductions ranged from 6% to 39% and did not grow with longer sessions: the two 60-minute protocols differed widely (about 6% and 32%) [15][28], while repeated 3-minute bouts already reduced glucose exposure by about 39% [14]. This agrees with the direct comparison in Blankenship et al. [17], where 20, 40, and 60 minutes gave similar results. These cross-study differences should be read cautiously, because the studies also differed in meal type, timing, and outcome measure. Overall, the evidence favors short sessions of 10–20 minutes taken after meals over the idea that longer sessions are always better.

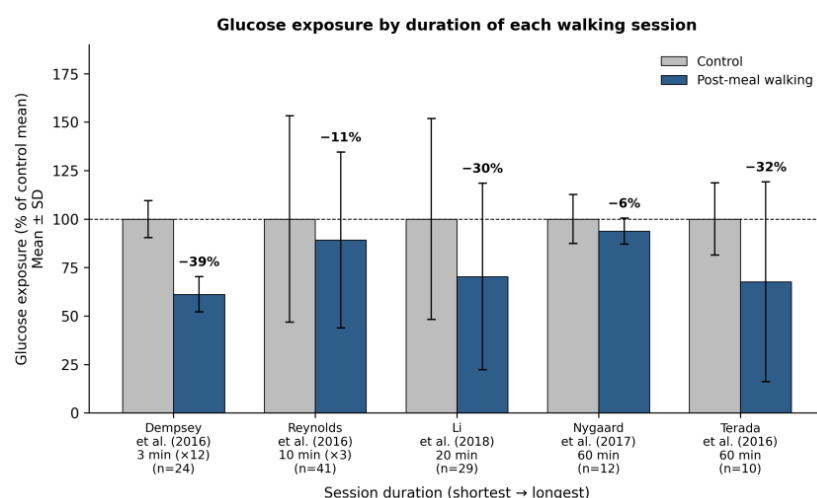


FIGURE 3: Glucose exposure by duration of each walking session. Bars show group means $\pm$ SD, normalized to each study's control mean (control = 100%); the value above each walking bar is the relative reduction versus control. Studies are ordered from the shortest to the longest session, and repeated-bout protocols show the number of bouts (e.g., 3 min $\times$ 12).

Intensity showed a similar pattern: benefit appeared from light intensity upward, so high intensity does not seem to be required. Dempsey et al. [14] used a speed of only 3.2 km/h (light intensity) and still lowered glucose iAUC by up to 39%. This matters clinically, because people with T2DM often have low fitness, older age, or other conditions that limit vigorous exercise. Moderate intensity was nonetheless the level most consistently supported. Pahra et al. [23] used brisk walking of about 3.3 METs, and Iida et al. [26] and Li et al. [24] both targeted 40% HRR; each reported lower glucose spikes or lower glucose variability. Bellini et al. [27] fit the same pattern, with 30 minutes of moderate walking producing one of the largest reductions in glycemic peak among the exercise conditions tested. Going above moderate intensity did not add a consistent benefit. In Terada et al. [15], the 120-minute glucose AUC after post-meal moderate walking (55% $\text{VO}_{2\text{peak}}$) was 240 mmol·min/L, numerically lower than the 314 mmol·min/L after

post-meal high-intensity interval exercise, although variability was wide. Reynolds et al. [13] is an exception for this question, because it described intensity only as light-to-moderate without a quantitative target, so it cannot define the best intensity even though its overall evidence is strong.

Figure 4 groups the same five studies by intensity. Glucose exposure was lower than control with light walking (about 39%) [14], light-to-moderate walking (about 11%, or 12% as originally reported) [13], and moderate walking (6% to 32%) [15][24][28]. A higher intensity therefore did not clearly mean a larger reduction: the light-intensity protocol produced one of the biggest reductions, while results at moderate intensity varied widely. No study in Figure 4 used vigorous walking, so the figure cannot show whether vigorous walking would help. As with duration, the studies differ in meal type, timing, and outcome measure, so this comparison is descriptive.

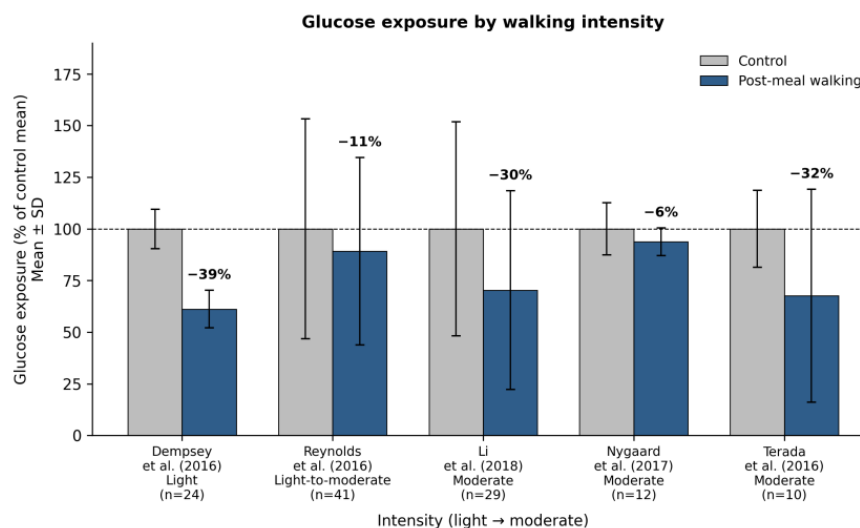


FIGURE 4: Glucose exposure by walking intensity. Bars show group means $\pm$ SD, normalized to each study's control mean (control = 100%); the value above each walking bar is the relative reduction versus control. Intensity categories follow the original authors' descriptions (light: 3.2 km/h; light-to-moderate; moderate: about 40% HRR, 55% $\text{VO}_{2\text{peak}}$, or RPE 12).

Taken together, walking dose cannot be judged by minutes alone; how the minutes are spread across the postprandial period also matters. Reynolds et al. [13] and Pahra et al. [23] both found that dividing the daily total into short sessions after each meal worked better than one longer session, even though the daily total was equal. Because the comparison sessions differed in timing as well as number (an unspecified time in Reynolds, before breakfast in Pahra), these studies show that post-meal placement helps but cannot separate the effect of splitting from the effect of timing. Based on all of the evidence, 10–20 minutes of light-to-moderate walking after meals is the most sensible balance of effectiveness and feasibility for people with T2DM. A 30-minute walk remains effective for those who can manage it, but there is no consistent evidence that extending sessions to 40–60 minutes adds a comparable benefit. On safety, Li et al. [24] found no nocturnal hypoglycemia after a 20-minute moderate

walk, although one small study cannot establish that the risk is zero. This range is therefore best described as the practical dose with the most support at present, not the optimal dose, because no trial has compared all combinations of duration and intensity in the same population.

Clinical Implications and Future Directions

Based on the synthesis of evidence in the previous sections, the practical recommendation that can be formulated for people with type 2 diabetes mellitus is to walk within 15–30 minutes after a meal, for 10–20 minutes per session at light to moderate intensity (equivalent to brisk walking or about 40% heart rate reserve). This approach is ideally performed right after each main meal, rather than as one long single session in a free time, because distributing exercise to follow the postprandial period has been shown to be more effective at suppressing glucose spikes than activity volume alone [13][23].

This principle is especially relevant for the consumption of high-carbohydrate foods such as rice, where the postprandial glucose spike tends to be sharper and faster than with lower-glycemic-index foods [1], so the first 15–30 minutes after a meal is a crucial window for intercepting the rise in glucose before it reaches its peak. Table 2 summarizes evidence from other exercise modalities beyond walking that have also been studied for postprandial glycemic control.

TABLE 2: Supporting Evidence of Other Post-Meal Exercise Modalities for Glycemic Control.

Author & Year	Study Design + Population & Sample	Exercise Modalities Compared	Meal Type & CHO Composition	Timing, Duration & Intensity	Outcome & Key Findings
Duvivier et al. (2017) [34]	RCT n=19 T2D	Sit Less vs. structured cycling vs. sitting	Free-living diet	Activity distributed throughout waking hours; Sit Less: standing ~2.5 h + light walking ~2.2 h/day (total ~4.7 h); cycling: 1.1 h/day, 5.9 METs	24-h glucose iAUC: Sit Less 1263 ± 189 vs. sitting 1974 ± 324 min·mmol/L (p=0.002). HOMA2-IR improved with Sit Less (p=0.015) compared with structured cycling (p=0.177).
Terada et al. (2016) [15]	RCT n=10 T2D	Post-meal HIIT vs. pre-meal HIIT vs. control	Breakfast; 600±57 kcal; 50% CHO, 20% protein, 30% fat	60 min after breakfast; 60-min HIIT: 15 intervals (1 min at 100% VO ₂ peak + 4 min at 40% VO ₂ peak)	120-min glucose AUC: 314±188 (post-meal), 267±184 (pre-meal), 355±66 mmol·min/L (control). Post-meal HIIT reduced AUC versus control but was not superior to pre-meal HIIT.
Verboven et al. (2020) [16]	RCT n=14 T2D	Postprandial cycling + resistance vs. pre-meal	Standard breakfast; 50% CHO, 20% protein, 30% fat	1–2 h after meal; combined cycling + resistance for 45 min, moderate intensity	AUC and peak glucose decreased significantly with postprandial exercise (p<0.05), indicating improved glycemic response.
Bellini et al. (2021) [27]	RCT n=8 T2D	Walking vs. resistance vs. combined aerobic-resistance	Breakfast; 310 kcal; 66% CHO, 19% fat, 15% protein	30 min after meal; walking 30 min, resistance 15 min, combined 30 min	All modalities reduced peak glucose and 0–120/180-min glucose iAUC versus control (p<0.048). The greatest reduction in peak glucose occurred with walking and the walking-resistance combination.
Yonglitthipagon et al. (2023) [35]	RCT n=24 T2D	Post-meal spot marching vs. standard care	Standard diabetes care diet	30 min after meal; 15 min/session × 3 sessions/day, 4 days/week for 8 weeks; moderate intensity	HbA1c: 7.99% (exercise) vs. 9.16% (control) (p<0.05). Heart rate recovery and leg muscle strength (FTSST) also improved compared with control.

Although walking is the focus of this review, other exercise modalities have also been shown to provide postprandial glycemic benefits, each with a different context of application. Duvivier et al. [34] showed that interspersing sitting time with standing and light walking throughout the day ("Sit Less") gave greater improvements in 24-hour glucose iAUC and insulin sensitivity than a single structured cycling session, making it a relevant option for patients whose jobs require prolonged sitting. For patients with better fitness and access to exercise facilities, postprandial HIIT has been shown to significantly reduce AUC and peak glucose. Combined cycling-resistance exercise has produced similar improvements in these glycemic outcomes [15][16], although both require technique supervision and are not always superior to pre-meal exercise. Bellini et al. [27] directly compared walking, resistance exercise, and a combination of both modalities. The greatest reduction in peak glucose occurred with walking alone and with the combined protocol that placed walking before resistance exercise. These findings support walking as an efficient foundational exercise modality.

Meanwhile, for patients who lack access to open space or face mobility limitations, post-meal spot marching (marching in place) has been shown to significantly improve HbA1c after eight weeks of intervention, while also increasing leg muscle strength and aerobic fitness [35], making it a practical home-based alternative that requires no special equipment or space. Overall, the pattern across these five modalities confirms that the principle of postprandial timing applies across modalities, but walking remains the most widely applicable choice because it requires no equipment, carries a low risk of injury, and can be performed by almost all patients regardless of age or fitness level. Although the evidence on post-meal walking in type 2 diabetes mellitus is fairly substantial, there is a fundamental research gap that is the central point of this review. Among the reviewed studies, no RCT specifically used rice or typical Asian foods, including Indonesian foods, as the test meal. Most studies used bread, cereal, biscuits, or glucose-based drinks as the glycemic stimulus [13][24][27]. Yet rice has glycemic index and digestive response characteristics that differ from those test foods [1], so the timing and duration recommendations derived from these studies remain essentially an extrapolation for populations with rice-based diets.

This gap is compounded by the absence of post-meal walking studies conducted in Indonesian or, more broadly, Southeast Asian populations; all reviewed studies came from European, North American, or East/South Asian populations whose dietary patterns differ from the rice-dominant consumption in Indonesia. In addition, the sample size in most studies was small to moderate (9–64 subjects), and almost all evaluated only acute effects over one to several days of observation, so evidence on long-term effects on HbA1c and on patient adherence to this strategy on a routine basis remains very limited;

only Pahra et al. [23] evaluated HbA1c outcomes over a period longer than one month.

Based on these gaps, future research should be directed at randomized clinical trials that specifically use rice as the standard test meal, conducted in populations with type 2 diabetes mellitus in Indonesia or Southeast Asia, to ensure that the timing (15–30 minutes), duration (10–20 minutes), and intensity (light to moderate) recommendations summarized in this review truly suit the glycemic response characteristics of that population. Long-term designs evaluating the impact on HbA1c, adherence, and quality of life are also needed to complement the evidence, which is currently still dominated by short-term acute-effect studies.

CONCLUSION

This literature review was prepared to address the knowledge gap regarding the effectiveness of post-meal walking in people with type 2 diabetes mellitus, particularly in the context of rice-based, high-carbohydrate food consumption, which has so far received little specific study. Based on a synthesis of ten main studies, walking after meals has consistently reduced postprandial glucose responses, reflected by reductions in peak glucose, glucose excursion, and AUC/iAUC. Post-meal walking also contributes to longer-term glycemic control, including improvements in HbA1c. This effect is mediated partly by insulin-independent GLUT4 translocation, supporting the effectiveness of post-meal walking in individuals with insulin resistance. This benefit is most consistently obtained when walking is performed within 15–30 minutes after a meal, for 10–20 minutes per session at light to moderate intensity, and this modality remains the most applicable choice compared with resistance exercise, HIIT, cycling, and spot marching because it requires no equipment or special supervision.

In practical terms, adults with type 2 diabetes mellitus are advised to walk for 10–20 minutes, beginning 15–30 minutes after each main meal (breakfast, lunch, and dinner), at moderate speed or brisk walking (about 4.8–6.4 km/h, still allowing conversation but with slightly faster breathing), performed routinely following the daily meal schedule. Because white rice has a high glycemic index and produces glucose spikes equal to or potentially sharper than the test foods used in these studies (bread, cereal, biscuits), the timing and duration principles are rationally expected to remain relevant after rice consumption, but this is an inference based on similar glycemic characteristics, not a recommendation that has been directly tested in rice-based meals.

The above recommendations are essentially an extrapolation, because no RCT in this review directly tested the effectiveness of post-meal walking after rice consumption, or was conducted in Indonesian or Southeast Asian populations. Other limitations include generally small to moderate sample sizes

(9–64 subjects), a study design dominated by short-term acute-effect evaluation, and minimal data on long-term adherence. Therefore, future research needs to be directed at randomized clinical trials that specifically use rice as the test meal in populations with type 2 diabetes mellitus in Indonesia, accompanied by long-term designs evaluating the impact on HbA1c and patient adherence, in order to validate whether the timing, duration, and intensity recommendations summarized in this review truly suit the glycemic response characteristics of that population.

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