Effects of Different Exercise Strategies to Improve Postprandial Glycemia in Healthy Individuals

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ABSTRACT

BELLINI, A., A. NICOLÒ, I. BAZZUCCHI, and M. SACCHETTI. Effects of Different Exercise Strategies to Improve Postprandial Glycemia in Healthy Individuals. Med. Sci. Sports Exerc., Vol. 53, No. 7, pp. 1334–1344, 2021. Purpose: We systematically investigated the effects of different exercise strategies on postprandial glycemia. Methods: Six randomized repeated-measures crossover studies were performed. Study 1 compared the effects of 60 min of brisk walking started at 30, 60, or 90 min after breakfast on postbreakfast and postlunch glycemic responses. Study 2 investigated the effects of 30 min of different exercise types (aerobic vs resistance vs combined). Study 3 compared the effects of 30 min of different aerobic exercise types (walking vs cycling vs elliptical). Study 4 evaluated the effects of 30 min of brisk walking performed 45 min before or 15 and 30 min after breakfast. Study 5 compared 30 with 45 min of postprandial brisk walking. Study 6 compared the effects of a total of 30 min brisk walking exercise fragmented in bouts of 15, 5, or 2.5 min performed every 15 min. Results: Postprandial but not preprandial exercise improved glycemic response (studies 1 and 4). The glycemic peak was attenuated only when exercise started 15 min after the meal (study 4). A similar reduction of the postprandial glycemic response was observed with different exercise types (studies 2 and 3). Thirty and 45 min of brisk walking provided a similar reduction of the postprandial glucose response (study 5). When performing activity breaks, 10 and 20 min of cumulative exercise were sufficient to attenuate postprandial glycemia in the first hour postmeal (study 6). Conclusion: Our findings provide insight into how to choose timing, type, duration, and modality for postprandial exercise prescription in healthy individuals. Key Words: ACTIVITY BREAKS, BREAKFAST, POSTPRANDIAL EXERCISE, AEROBIC EXERCISE, RESISTANCE EXERCISE, HYPERGLYCEMIC PEAK

Postprandial hyperglycemia and large postprandial glycemic fluctuations may enhance the risk of developing cardiometabolic disorders not only in people with diabetes but also in healthy individuals (1,2). Indeed, postprandial glucose dysmetabolism is associated with increased oxidative stress (3), expression of proinflammatory factors (4), endothelial dysfunctions (5,6), and increased risk of developing cardiovascular diseases (7). Importantly, exercise plays a relevant role in improving postprandial glycemic response and, therefore, preventing the negative effects of excessive glucose excursions (8–10). However, relevant exercise parameters related to postprandial metabolic control are overlooked by the current guidelines on physical activity for people with diabetes and healthy individuals (11–14).

The timing of exercise in relation to the previous or next meal is of great relevance in metabolic control. Performing exercise after the consumption of a meal seems to attenuate postprandial glycemic response more than exercising in fasting conditions (8,9,15). Furthermore, a correct exercise timing may attenuate the postmeal glycemic peak, thus preventing the negative cardiometabolic effects associated with it. However, there is a paucity of studies on the relationship between exercise timing and glucose peak reduction in healthy individuals. Therefore, further research is needed to establish the optimal time lapse between exercise and meal.

The effect of different exercise types on glycemic response is another relevant factor to be taken into account. Both aerobic and resistance exercise effectively improve fasting and postprandial glycemic control in individuals with diabetes (16,17). In addition, the combination of aerobic and resistance exercise had greater effects than the two types alone on chronic glycemic control in patients with type 2 diabetes (18,19), whereas no differences were found in acute glycemic responses (20). However, there is limited evidence on the effects of different exercise modalities on postprandial glycemic control in healthy individuals, and further work in this area is required.

Other parameters potentially affecting postprandial glycemia are intensity, duration, and fragmentation (by performing activity breaks) of an exercise bout. The influence of exercise intensity has been investigated, and no differences were found.
on postprandial glycemic response when exercising at low, moderate, or vigorous intensities (21,22). Conversely, only a few studies have assessed the effect of exercise duration on postprandial glycemic response (23,24). Likewise, there is no clear evidence on the effects of activity breaks performed during the first hours postmeal, whereas this exercise strategy improves glycemic response when activity breaks are performed over several hours (25–27). Hence, further research is needed to assess if the modulation of exercise duration and activity breaks may improve postprandial glycemic responses. The purpose of the present investigation was to provide insight into the effects of different exercise strategies on postprandial glycemic response by systematically manipulating key parameters of exercise prescription, i.e., timing, type, duration, and modality of exercise. Six separate studies were performed to assess the effect of each of these parameters on postprandial glycemic response, and homogeneous groups of healthy individuals were tested to facilitate between-study comparisons. The recruitment of healthy individuals has allowed us to investigate the effect of exercise per se, thus excluding the confounding factors of pathological conditions and drug therapies. Collectively, the results of the six studies were expected to shed further light on how to prescribe exercise for improving postprandial glycemic response.

**METHODS**

**Participants.** Seventy-seven healthy, adult (19–40 yr old), and physically active (at least 150 min of moderate-intensity or 75 min of vigorous-intensity activity per week) volunteers were recruited for this investigation. They participated in one of six studies, as described below. Seventy-three individuals completed all the experimental procedures of the study in which they participated. A summary of participants’ characteristics is given in Table 1. All participants provided written informed consent for the study. The investigation was conducted in accordance with the Declaration of Helsinki, and ethical approval was provided by the Local Ethical Committee.

**Studies overview.** All the studies had a randomized repeated-measures crossover design. During each study, participants attended the laboratory four or five times. The first visit was the same for all the studies and consisted of a familiarization session. In addition, all the studies involved a control session (CON) during which no exercise was performed. A standardized breakfast high in carbohydrate content was provided at the same time of the day in all studies, but a standardized lunch was also supplied only in study 1. An overview of the protocols of the studies is schematically shown in Figure 1.

**Familiarization session.** During the first visit, participants were familiarized with the exercise protocols and the other experimental procedures. Furthermore, recommendations on nutritional and physical activity behaviors to assume before the experimental sessions were made. More specifically, participants were asked to refrain from vigorous exercise during the 48 h preceding each experimental session and, since the evening before, from the consumption of alcohol and caffeine. Moreover, subjects were invited to register their diet during the 24 h and activities during the 48 h preceding the first experimental trial of each study, and these behaviors were replicated before the subsequent visits.

**Experimental sessions.** During the remaining sessions of each study, participants attended the laboratory at 8:00 AM after an overnight fasting period (at least 10 h). A standardized mixed breakfast was provided at 9:00 AM. During each study, participants completed three (study 5) or four (studies 1, 2, 3, 4, and 6) experimental conditions in a randomized order and spaced 1 wk apart. All the studies included a control session (CON), during which participants remained seated for the whole experimental period. The other visits differed between studies, as explained below and represented in Figure 1.

**Study 1—the effect of exercise timing on postbreakfast and postlunch glycemia.** Participants completed four visits lasting 6 h and 30 min each. They performed a control trial (CON) and three experimental conditions involving 60 min of brisk walking performed 30 min (POST-30), 60 min (POST-60), or 90 min (POST-90) after the beginning of the breakfast (Fig. 1). Furthermore, an additional standardized mixed meal was consumed at 1:30 PM in all the conditions. Brisk walking was conducted at 120 steps per minute on a 50-m track. The walking cadence was rhythmically established using a digital metronome (Soundbrenner, Berlin, Germany). It was selected as a surrogate of walking intensity because cadence can easily be controlled and reproduced in free-living conditions. Furthermore, the cadence has been previously proposed as a valid estimate of the metabolic cost of walking pace (28). Walking speed was also measured to assure between-condition consistency (5.25 ± 0.22 km h⁻¹). The same exercise type was also performed in studies 2, 4, 5, and 6.

**Study 2—the effect of aerobic, resistance, and combined exercise types.** Participants performed a CON session and three experimental conditions lasting 3 h. During the three experimental visits, participants performed 30 min of brisk walking (A30), 30 min of resistance exercises (R30), or

| Table 1. Summary of the characteristics of participants in all the studies. |
|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
| **Study** | **1** | **2** | **3** | **4** | **5** | **6** |
| **Sample size (M/F)** | 14 (8/6) | 10 (6/4) | 10 (4/6) | 13 (8/5) | 12 (8/4) | 14 (7/7) |
| **Age** | 25 ± 5 | 24 ± 5 | 24 ± 5 | 23 ± 1 | 24 ± 5 | 23 ± 5 |
| **Weight (kg)** | 72 ± 12 | 65 ± 12 | 65 ± 9 | 73 ± 16 | 73 ± 18 | 70 ± 13 |
| **Height (m)** | 1.74 ± 0.07 | 1.71 ± 0.05 | 1.73 ± 0.09 | 1.72 ± 0.10 | 1.75 ± 0.13 | 1.68 ± 0.09 |
| **BMI (kg m⁻²)** | 23.9 ± 2.5 | 22.4 ± 1.8 | 21.9 ± 2.7 | 24.5 ± 2.9 | 23.6 ± 3.2 | 24.7 ± 2.8 |

Data are expressed as mean ± SD.

M: male; F: female; BMI: body mass index.
15 min of brisk walking combined with 15 min of resistance exercises (COMB). All the exercise sessions started 30 min after the beginning of the breakfast (Fig. 1). Aerobic exercise was performed as described in study 1. Walking speed was $5.16 \pm 0.16$ km·h$^{-1}$. Resistance exercise consisted of a circuit training divided into four or two sets (in R30 and COMB, respectively) composed of five resistance exercises (push-up, box squat, dynamic plank, rowing using elastic bands, and lunges) sequentially performed. Each exercise lasted 30 s and was followed by 60 s of passive rest (work–rest ratio, 1:2). Exercise conditions were matched for duration. In addition, RPE was measured through the Borg’s 6–20 RPE scale.
at 15 and 30 min from the beginning of exercise. Thirty minutes after the end of the exercise period, participants were also asked to answer to the question “How hard was your workout?,” assigning a value on the Category Ratio 10 (CR-10) scale (29).

**Study 3—the effect of different aerobic exercise types.** Participants performed a CON session and three experimental conditions lasting 3 h. During the three experimental visits, participants completed 30 min of walking on a treadmill (WALKING), 30 min of cycling on a cycle ergometer (CYCLING), or 30 min of elliptical exercise (ELLIPTICAL) (Fig. 1). Exercise sessions started 30 min after the beginning of the breakfast. Exercise intensity was set at 70% of age-predicted maximum heart rate (208 – [0.7 × age]) for all the modalities (WALKING, 133 ± 5 bpm; CYCLING, 133 ± 6 bpm; ELLIPTICAL, 135 ± 7 bpm).

**Study 4—the effect of pre- versus postprandial exercise (timing).** Participants performed a CON session and three experimental conditions lasting 3 h and 45 min. During the three experimental visits, participants completed 30 min of brisk walking starting 45 min before (45-PRE), 15 min after (15-POST), or 30 min after (30-POST) the beginning of the breakfast (Fig. 1). Exercise was performed as described in study 1. Walking speed was 5.19 ± 0.31 km·h⁻¹.

**Study 5—the effect of exercise duration.** Participants performed a CON session and three experimental conditions lasting 3 h. During the three experimental visits, participants completed 30 min of brisk walking starting 15 min after the beginning of the breakfast (Fig. 1). Exercise was performed as described in study 1. Walking speed was 5.19 ± 0.39 km·h⁻¹.

**Study 6—the effect of exercise fragmentation on postprandial glycemia.** Participants performed a CON session and three experimental conditions lasting 3 h. During the three experimental visits, participants completed a total of 30 min of brisk walking fragmented into activity breaks. The 30 min of exercise was spread within the postprandial period as follows: (i) 2 × 15 min of walking separated by 15 min of rest (AB15), (ii) six intervals of 5 min of walking separated by 10 min of rest (5 min of walking every 15 min) (AB5), and (iii) 12 intervals of 2.5 min of walking separated by 12.5 min of rest (2.5 min of walking every 15 min) (AB2.5). The first bout of exercise started 15 min after the beginning of the breakfast. Exercise was performed as described in study 1. Walking speed was 5.08 ± 0.30 km·h⁻¹. Figure 1 shows a graphical representation.

**Resting time.** At the end of each exercise session and during the CON sessions, participants remained seated until the end of the visit. During sitting time, participants were asked to limit as much as possible any movement; however, they were allowed to read, listen to music, using PC, etc., with the recommendation to carry out the same activities in all the experimental visits.

**Meal composition.** During all the studies, an identical standardized breakfast high in carbohydrate content was provided (~75% of total energy intake). Food consisted of cornflakes, partially skimmed milk, and sugar to provide a total of 1 g of carbohydrate per kilogram of body weight. During study 1, in addition to breakfast, a standardized high-carbohydrate lunch (pasta, extra-virgin olive oil, and parmesan) was provided. The energy derived from the lunch was 30% of the individual total daily energy requirement, calculated using the Harris–Benedict equation and multiplied to a physical activity factor of 1.4. Participants were allowed to consume water ad libitum, but no other food or beverages were permitted.

**Glycemic assessment.** Fingertips capillary blood samples were collected every 15 min (studies 2–6) or 30 min (study 1) (see Fig. 1) and analyzed using reactive strips and a glucose monitor (Contour®Next; Bayer HealthCare S.p.A., Milan, IT) (30). All precautions were taken to avoid unprecise glucose measurements due to external factors (31). Blood glucose was measured in duplicate, and the average of the two measures was considered. A third additional measurement was performed when the two values differed more than 10%.

**Statistical analysis.** Statistical analysis was performed using the software IBM SPSS statistics version 23.0 (SPSS Inc., Chicago, IL). The statistical analyses performed were the same for all the studies. Data were checked for normality using the Shapiro–Wilk test. A two-way repeated-measures ANOVA (condition–time) was used for comparing the time course of glycemia across conditions. When significant interactions were found, a one-way repeated-measures ANOVA was performed for analyzing the simple main effect of condition at each time point. Mean blood glucose concentration was calculated for the period 0–180 min after breakfast in all the studies and for 0–90 min after lunch in study 1. Total area under the curve (AUC) was calculated using the trapezoid rule (32) and divided by the period of observation to obtain a time-averaged value. AUC values were obtained for the 0- to 60-min period after breakfast in studies 2–6 and for the 0–180 min (after breakfast) and 0–90 min (after lunch) periods in study 1. A one-way repeated-measures ANOVA was used to analyze differences between conditions in mean glucose and time-averaged glucose AUC. In study 2, a two-way repeated-measures ANOVA was used to compare the Borg’s 6–20 RPE scale scores between conditions, whereas a one-way repeated-measures ANOVA was used to compare the CR-10 scale scores. When significant differences were found, the Bonferroni correction was used for multiple comparisons. Sphericity was assessed using the Mauchly’s test. In case of violation of the sphericity assumption, the Greenhouse–Geisser adjustment was performed. For all statistical tests, the level of significance was set at P < 0.05. Partial eta squared (η²p) effect sizes were determined, considering η²p ≥ 0.01 as small, η²p ≥ 0.059 as medium, and η²p ≥ 0.138 as large (33). Data are presented as mean ± SD in Table 1 and in the Results section and mean ± SEM in figures.

**RESULTS**

**Study 1—the effect of exercise timing on postbreakfast and postlunch glycemia.** When the time course of glucose concentration was compared between conditions, a significant interaction (condition–time) (P < 0.001, η²p = 0.221) was
found. Figure 2A shows where a simple main effect of condition was found. Glucose AUC showed no between-condition differences at 0–180 min postbreakfast or 0–90 min postlunch (Fig. 2B and C).

Mean glucose concentration showed no significant between-condition differences neither at 0–180 min after breakfast (CON, 98.97 ± 6.92 mg·dL⁻¹; POST-30, 95.69 ± 7.67 mg·dL⁻¹; POST-60, 97.85 ± 7.57 mg·dL⁻¹; POST-90, 97.32 ± 6.06 mg·dL⁻¹) nor at 0–90 min after lunch (CON, 109.13 ± 7.79 mg·dL⁻¹; POST-30, 108.96 ± 8.83 mg·dL⁻¹; POST-60, 109.98 ± 7.40 mg·dL⁻¹; POST-90, 108.64 ± 10.44 mg·dL⁻¹).

Study 2—the effect of aerobic, resistance, and combined exercise types. When the time course of glucose concentration was compared between conditions, a significant interaction (condition–time) \((P = 0.012, \eta_p^2 = 0.250)\) was found. All the exercise types proposed (A30, R30, and COMB) similarly lowered postprandial glycemia compared with CON at 45 min, but only COMB significantly lowered glycemia compared with CON at 60 min. Figure 3A shows where a simple main effect of condition was found.

Glucose AUC at 0–60 min after breakfast showed significant differences \((P = 0.002, \eta_p^2 = 0.407)\). Post hoc analysis revealed significantly lower values in R30 \((P = 0.035)\) compared with CON (Fig. 4A). In addition, a statistical trend was found between COMB and CON \((P = 0.066)\) but not for A30 compared with CON \((P = 0.093)\).

Mean glucose concentration showed significant between-condition differences at 0–180 min \((P = 0.045, \eta_p^2 = 0.253)\), although the post hoc pairwise comparisons did not reveal significant differences (CON, 100.98 ± 8.19 mg·dL⁻¹; A30, 94.19 ± 9.21 mg·dL⁻¹; R30, 93.97 ± 8.53 mg·dL⁻¹; COMB, 93.19 ± 6.19 mg·dL⁻¹).

No significant differences between conditions were found between the Borg’s 6–20 RPE scale scores at 15 min (A30, 9.5 ± 1.3; R30, 11.2 ± 1.1; COMB, 9.7 ± 1.0) and those at 30 min (A30, 10.7 ± 1.5; R30, 12.2 ± 1.2; COMB,
11.1 ± 1.0). Similarly, no significant differences between conditions were found for the CR-10 scale scores (A30, 3.2 ± 0.8; R30, 3.8 ± 0.8; COMB, 3.4 ± 0.5).

**Study 3**—the effect of different aerobic exercise types. When the time course of glucose concentration was compared between conditions, a significant interaction (condition–time) \((P < 0.001, \eta_p^2 = 0.443)\) was found. All the aerobic exercise types (WALKING, CYCLING, and ELLIPTICAL) similarly lowered glucose concentration compared with CON. Figure 3B shows where a simple main effect of condition was found.

Glucose AUC at 0–60 min after breakfast showed significant differences \((P < 0.001, \eta_p^2 = 0.592)\). *Post hoc* analysis revealed significantly lower values in WALKING \((P = 0.008)\), CYCLING \((P = 0.004)\), and ELLIPTICAL \((P < 0.001)\) compared with CON (Fig. 4B).

Mean glucose concentration showed significant between-condition differences at 0–180 min \((P = 0.001, \eta_p^2 = 0.461)\). The *post hoc* pairwise comparisons revealed significantly lower values in CYCLING \((100.54 ± 7.73 \text{ mg·dL}^{-1})\) and ELLIPTICAL \((100.97 ± 7.68 \text{ mg·dL}^{-1})\) compared with CON \((109.37 ± 10.87 \text{ mg·dL}^{-1})\) \((P = 0.036\) and \(P = 0.010,\)

**FIGURE 3**—Glycemic response over time for study 2 (A) (effect of exercise types) and study 3 (B) (effect of different aerobic exercise types). (A) e, \(P < 0.05\) vs A30; f, \(P < 0.05\) vs R30; g, \(P < 0.05\) vs COMB. (B) h, \(P < 0.05\) vs WALKING; i, \(P < 0.05\) vs CYCLING; j, \(P < 0.05\) vs ELLIPTICAL. The boxes in the graphs indicate the exercise period, and the arrows indicate meals. Values are presented as mean ± SEM.
Study 3—the effect of different aerobic exercise types. A significant interaction (condition × time) \((P = 0.006, \eta^2_p = 0.241)\) was found. W30 and W45 similarly reduced postmeal glucose concentration compared with CON. Figure 5B shows where a simple main effect of condition was found.

Glucose AUC at 0–60 min after breakfast showed significant differences \((P < 0.001, \eta^2_p = 0.509)\). Post hoc analysis revealed significantly lower values for W30 \((P = 0.023)\) and W45 \((P = 0.001)\) compared with CON, whereas no significant differences were found between W45 and W30 (Fig. 6B).

Mean glucose concentration showed significant between-condition differences at 0–180 min \((P = 0.024, \eta^2_p = 0.288)\), although the post hoc pairwise comparisons did not reveal significant differences \((P = 98.25 \pm 8.78 \text{ mg·dL}^{-1}; \text{W30}, 92.45 \pm 5.86 \text{ mg·dL}^{-1}; \text{W45}, 94.78 \pm 8.07 \text{ mg·dL}^{-1}).

Study 6—the effect of exercise fragmentation on postprandial glycemia. When the time course of glucose concentration was compared between conditions, a significant interaction (condition × time) \((P < 0.001, \eta^2_p = 0.256)\) was found. AB5 and AB2.5 presented lower postprandial glycemia compared with those of CON and AB15. Figure 5C shows where a simple main effect of condition was found. Glucose AUC at 0–60 min after breakfast showed significant differences \((P = 0.011, \eta^2_p = 0.300)\). Post hoc analysis revealed significantly lower values in AB5 \((P = 0.028)\) and AB2.5 \((P = 0.027)\) compared with CON, whereas no significant differences were observed between AB15 and CON (Fig. 6C).

Mean glucose concentration showed significant between-condition differences at 0–180 min \((P = 0.021, \eta^2_p = 0.256)\). The post hoc pairwise comparisons revealed a statistical trend \((P = 0.062)\) between CON \((103.44 \pm 8.33 \text{ mg·dL}^{-1})\) and AB2.5 \((96.68 \pm 8.32 \text{ mg·dL}^{-1})\), whereas no differences were observed for AB15 \((99.22 \pm 8.87 \text{ mg·dL}^{-1})\) and AB5 \((97.86 \pm 7.72 \text{ mg·dL}^{-1})\).

**DISCUSSION**

Current exercise guidelines do not specifically address the issue of how to prescribe exercise for improving postprandial glycemic response. To partially overcome this problem, we provided a systematic and extensive assessment of the effect of the main exercise parameters affecting postprandial glucose response in healthy individuals. Six studies were performed with the following main findings: (i) postprandial but not preprandial exercise reduced the glycemic response to a standardized meal; (ii) the hyperglycemic peak was only attenuated when exercise started at 15 min after the meal; (iii) a similar reduction of the postprandial glycemic response was observed with different exercise types; (iv) when performing continuous exercise, 30 min was effective for improving postprandial glycemia and no additional benefits were observed for longer...
bouts of exercise; and (v) when fragmenting exercise in activity breaks, 10 and 20 min of cumulative exercise were sufficient to improve postprandial glycemic response in the first

**FIGURE 6** — Time-averaged glucose AUC at 0–60 min for study 4 (A) (effect of exercise timing), study 5 (B) (effect of exercise duration), and study 6 (C) (effect of exercise fragmentation). a, \( P < 0.05 \) vs CON; b, \( P < 0.05 \) vs 45-PRE. Values are presented as mean ± SEM.

**FIGURE 5** — Glycemic response over time for study 4 (A) (effect of exercise timing), study 5 (B) (effect of exercise duration), and study 6 (C) (effect of exercise fragmentation). (A) a, \( P < 0.05 \) vs CON; b, \( P < 0.05 \) vs 45-PRE; c, \( P < 0.05 \) vs 15-POST; d, \( P < 0.05 \) vs 30-POST. (B) e, \( P < 0.05 \) vs W30; f, \( P < 0.05 \) vs W45. (C) g, \( P < 0.05 \) vs AB15; h, \( P < 0.05 \) vs AB5; i, \( P < 0.05 \) vs AB2.5. The boxes in the graphs indicate the exercise period, and the arrows indicate meals. Values are presented as mean ± SEM.
hour postmeal. These findings have important implications for effective postprandial exercise prescription.

Our findings show that timing is one of the most relevant parameters affecting postprandial glycemia. When directly comparing exercise performed before or after breakfast (study 4), we found that exercise performed after the meal is superior to exercise performed before the meal in reducing postprandial glycemia. Likewise, exercise performed after breakfast showed a reduction in the glycemic response after breakfast but not after lunch (study 1). These results are in line with previous findings showing a more effective postprandial glycemic response when exercise is performed after the meal in both people with diabetes and healthy individuals (8,9). However, different timing strategies of postprandial exercise may affect the glycemic response in different ways, especially within the first 2 h after the meal. This matters because the glycemic fluctuations observed in this time window are predictors of cardiometabolic risk (1,3,6). The present investigation adds insight into this issue.

Our findings show that the choice of the correct timing of exercise is crucial for the reduction of the hyperglycemic peak. In healthy individuals, the hyperglycemic peak is reached between 30 and 45 min after the beginning of the meal, as documented by the present and previous results (34). This is why we assessed the effect of starting exercise at 15 min (15-POST) compared with 30 min (30-POST) after the meal (study 4). We found that a relatively small difference in timing (15 min) determines large differences in the reduction of the hyperglycemic peak. A substantial decrease in the glucose peak was only found in the 15-POST condition, whereas no changes were observed in the 30-POST condition. The same conclusion can be drawn when comparing the other studies with a timing of 15 min (studies 5 and 6) to those with a timing of 30 min (studies 2 and 3). In study 1, we further tested the effect of timing on postprandial glycemia and found that the glucose reduction was time shifted by 30 min when comparing timings of 30, 60, and 90 min. Hence, while timing affected the postprandial glucose response up to 90 min postmeal, no effect on the hyperglycemic peak was found in any of the three conditions. These findings shed some light on the prescription of the optimal exercise timing, suggesting that an early start after the meal (i.e., 15 min for healthy individuals) is essential for the reduction of the hyperglycemic peak.

The present investigation also provides insight into the volume of exercise to perform after the consumption of a meal. In most of the studies presented here, we used 30 min of continuous exercise as a practical solution to improve postprandial glycemic response. When comparing 30 and 45 min of exercise directly (study 5), we found no additional benefits of a 15-min increase in exercise duration on the postprandial glucose response. Likewise, similar mean glucose concentration values were observed when 30 min (studies 2, 3, and 4) or 60 min (study 1) of exercise was performed. Conversely, 15 min of exercise appears to be a suboptimal exercise duration to reduce the postprandial hyperglycemic peak, as suggested by the glucose response observed in the AB15 condition of study 6. These findings extend previous observations showing a higher effect of 40 min versus 15 min (24) or 20 min (23) of postprandial exercise in improving postprandial glycemic response in healthy individuals (24) or in those at high risk of developing type 2 diabetes (23). Collectively, the present and previous findings (23,24) suggest that 30 min of continuous moderate-intensity exercise is a practical and effective dose to improve postprandial glycemic response in healthy individuals.

When exercise is fragmented in activity breaks, the recommendations on the volume of exercise may differ compared with the above recommendations for continuous exercise. Moreover, a recent study suggested that accumulating 30 min walking in 10-min bouts after each meal leads to a similar effect on postprandial glucose response compared with a single 30-min continuous bout (35). Our results revealed that when exercise was structured into short bouts of 5 min (AB5) and 2.5 min (AB2.5) of activity repeated every 15 min, a substantial reduction in the glucose peak was observed in both exercise conditions. Furthermore, the glucose AUC response was significantly improved after 1 h from the beginning of the meal. These responses were similar to those observed when 30 min of continuous exercise was performed (studies 4 and 5). These findings are even more interesting if we consider that an improvement of glycemic response was observed in the first hour after the meal with only 20 and 10 min of cumulative exercise in the AB5 and AB2.5 conditions, respectively. This suggests that a low dose of exercise is sufficient to improve postprandial glycemia when exercise is structured in activity breaks. Furthermore, an improvement in glycemic response was observed throughout the 3-h postmeal when activity breaks were performed over the course of this time frame (i.e., in the AB2.5 condition). The efficacy of short-duration activity breaks has important implications for individuals with poor exercise capacity, hence requiring further studies in patients.

Although exercise type is a classic factor affecting physiological responses to exercise, our findings showed no substantial effect of exercise type on postprandial glycemia. We observed similar attenuations in the postprandial glycemic responses when 30 min of aerobic, resistance, or combined exercise was performed (study 2). Contrasting results were found in patients with diabetes when evaluating the effects of different exercise types on acute and chronic glycemic control (17–20). However, it is difficult to compare our findings with the responses observed in patients with diabetes that are affected by confounding factors such as aging, metabolic disorders, and pharmacological treatments. We also showed a similar attenuation of postprandial glycemia when performing walking, cycling, or elliptical exercises (study 3). These results suggest that the recruitment of lower limb muscle masses provides a sufficient stimulus for improving the postprandial glycemic response and that the additional recruitment of upper limb muscle masses does not provide further benefits. Along this line, the increase in exercise intensity does not lead to improvements in postprandial glycemia (21,22). On the other hand, these findings are relevant from an exercise adherence perspective, as practitioners can select the exercise type that they prefer without impairing the effect of postprandial...
exercise. Exercise adherence is also favored by the fact that brisk walking is effective both in continuous and intermittent formats, and that does not require any tool or specific training structure to be performed.

We did not attempt to investigate the mechanisms underlying the effect of the exercise strategies proposed on postprandial glycemia. However, it is conceivable that timing, volume, and activity breaks are important factors affecting the rate of appearance and disappearance of blood glucose. These factors may affect insulin-independent (36) and insulin-dependent glucose uptake (37), endogenous glucose production, gastric emptying, and the appearance of glucose from the meal (38–40). Given the paucity of studies investigating the mechanisms underlying the effect of different postprandial exercise strategies on glycemic response in healthy individuals, further research is needed to address this issue.

This investigation has some limitations that should be acknowledged. First, the use of capillary blood samples (collected every 15 min in studies 2–6 and every 30 min in study 1) may have limited the possibility to precisely identify the timing corresponding to the glucose peak. Although the higher sampling frequency of the continuous glucose monitoring system might have addressed this issue, it was not used in this investigation because interstitial glucose concentration shows a delayed response compared with blood glucose concentration, especially in non–steady-state conditions such as those induced by meals and exercise (41). Hence, the use of the continuous glucose monitoring system may have some limitations when assessing the effect of relatively short exercise bouts within the early postprandial phase, but it offers valuable information for the evaluation of the late effect of exercise. Second, although the recruitment of healthy individuals has several advantages, as previously described, this population shows lower glycemic excursions compared with patients with diabetes. This factor may have contributed to limit the possibility to observe significant differences across exercise conditions, along with the relatively small sample sizes of our studies and the use of the Bonferroni correction. Hence, our findings should not discourage researchers to assess the effect of different exercise strategies on patients with glycemic dysmetabolism. These limitations have not allowed us to identify the most effective strategies for improving postprandial glycemia among those tested in this investigation. Nevertheless, our findings show that various exercise strategies enhance postprandial glycemic control, thus potentially satisfying the needs of different populations that may benefit from postprandial exercise.

CONCLUSION

This investigation aimed to systematically assess the effect of the manipulation of different parameters of the postprandial exercise to improve our understanding of how to prescribe exercise after the meal. Our findings show that the timing of exercise is crucial to improve postprandial glycemic response. Exercise should be performed as early as possible after the meal to decrease the glycemic peak, as we found in our healthy volunteers that a timing of 15 min is substantially more effective than that of 30 min, whereas premeal exercise showed no effects. When performing continuous exercise, 30 min is an effective exercise duration. Longer durations are not necessarily more beneficial, whereas an exercise duration of 15 min appears to improve postprandial glycemia less. Brisk walking is an effective exercise type, but other forms of aerobic or resistance training can also be used. When exercise is structured in activity breaks, even 10 min of brisk walking spread over the first hour postmeal is sufficient to effectively attenuate the glucose peak and improve postprandial glycemia. This result was achieved by performing 2.5 min of activity breaks every 15 min. When this format of activity breaks is prolonged up to 3 h postmeal, further benefits in glycemic response are observed. These findings have implications for structuring effective training plans aiming to improve postprandial glycemic response. Future studies should verify the long-term effects of these exercise recommendations on healthy individuals and patients with glucose dysmetabolism.

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