

Three 15-min Bouts of Moderate Postmeal Walking Significantly Improves 24-h Glycemic Control in Older People at Risk for Impaired Glucose Tolerance

LORETTA DiPIETRO, PHD¹
ANDREI GRIBOK, PHD²
MICHELLE S. STEVENS, MS²

LARRY F. HAMM, PHD¹
WILLIAM RUMPLER, PHD²

OBJECTIVE—The purpose of this study was to compare the effectiveness of three 15-min bouts of postmeal walking with 45 min of sustained walking on 24-h glycemic control in older persons at risk for glucose intolerance.

RESEARCH DESIGN AND METHODS—Inactive older (≥ 60 years of age) participants ($N = 10$) were recruited from the community and were nonsmoking, with a BMI $< 35 \text{ kg m}^{-2}$ and a fasting blood glucose concentration between 105 and 125 mg dL⁻¹. Participants completed three randomly ordered exercise protocols spaced 4 weeks apart. Each protocol comprised a 48-h stay in a whole-room calorimeter, with the first day serving as the control day. On the second day, participants engaged in either 1) postmeal walking for 15 min or 45 min of sustained walking performed at 2) 10:30 A.M. or 3) 4:30 P.M.. All walking was on a treadmill at an absolute intensity of 3 METs. Interstitial glucose concentrations were determined over 48 h with a continuous glucose monitor. Substrate utilization was measured continuously by respiratory exchange (VCO_2/VO_2).

RESULTS—Both sustained morning walking (127 ± 23 vs. $118 \pm 14 \text{ mg dL}^{-1}$) and postmeal walking (129 ± 24 vs. $116 \pm 13 \text{ mg dL}^{-1}$) significantly improved 24-h glycemic control relative to the control day ($P < 0.05$). Moreover, postmeal walking was significantly ($P < 0.01$) more effective than 45 min of sustained morning or afternoon walking in lowering 3-h postdinner glucose between the control and experimental day.

CONCLUSIONS—Short, intermittent bouts of postmeal walking appear to be an effective way to control postprandial hyperglycemia in older people.

Insulin secretion (relative to increased insulin resistance) declines with older age, and an impaired β -cell compensation for any existing aging- or disuse-related insulin resistance will accelerate the risk of postchallenge hyperglycemia and consequent type 2 diabetes (1). There also is evidence that glycemic exposure is a continuous risk factor for cardiovascular disease, with no apparent threshold (2–4). Therefore, higher levels of glycemia

(even in the nondiabetic range) have significant clinical implications. Most of the glycemic exposure among people who develop micro- and macrovascular complications from diabetes comprises isolated postprandial hyperglycemia (i.e., a 2-h postchallenge glucose concentration $\geq 200 \text{ mg dL}^{-1}$, but a fasting level $< 126 \text{ mg dL}^{-1}$) (5,6). Isolated postprandial hyperglycemia is particularly common in older people (7,8). Data from

epidemiologic studies suggest that post-challenge glucose concentrations increase with age by $\sim 6\text{--}9 \text{ mg dL}^{-1}$ per decade (9), whereas fasting glucose levels increase by $1\text{--}2 \text{ mg dL}^{-1}$ per decade (10). Thus, postprandial hyperglycemia may represent a key factor in the progression from impaired glucose tolerance (IGT) toward frank type 2 diabetes and cardiovascular disease in older people (1).

Both insulin and exercise (muscle contractions) stimulate the translocation of GLUT-4 transporter proteins to the plasma membrane in skeletal muscle, and the effects of these processes are at least to some degree additive (11,12). Muscle contractions per se will initiate glucose uptake independent of insulin secretion, however, and therefore exercise can supplement peripheral insulin action in the presence of an aging-related low or blunted insulin secretory response. Time of day may influence how effective exercise is on subsequent glycemic control, however, as there is evidence that insulin secretion is markedly lower in the afternoon compared with the early morning in older people (13), and the benefits of exercise may be best realized the closer that exposure is to the time it is needed (i.e., postprandially). Thus, timing of the exercise exposure (as well as volume [frequency \times duration]) is an important element to consider with regard to treating those older people vulnerable to postprandial hyperglycemia, especially later in the day. We investigated whether smaller bouts of walking performed after each meal (during the period of absorption) would have a greater benefit for postprandial, as well as for 24-h, glucose control than would a single large bout of walking performed once per day. We are not aware of any study comparing the benefits of several intermittent bouts of postmeal exercise with a single sustained bout (of similar daily volume) on continuous measures of glucose homeostasis in older people.

From the ¹Department of Exercise Science, The George Washington University School of Public Health and Health Services, Washington, DC; and the ²Beltsville Human Nutrition Research Laboratory, United States Department of Agriculture, Beltsville, Maryland.

Corresponding author: Loretta DiPietro, ldpl@gwu.edu.

Received 11 January 2013 and accepted 2 April 2013.

DOI: 10.2337/dc13-0084

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RESEARCH DESIGN AND METHODS

Study subjects

Subjects were recruited by advertisement from senior centers and from senior residential communities within the Washington, DC metropolitan area. Potential subjects were older (≥ 60 years of age) otherwise healthy men and women who were at risk for IGT based on a fasting glucose concentration 105–125 mg dL⁻¹. In addition, they were nonsmoking, were without uncontrolled hypertension, had a BMI < 35 mg kg⁻², and reported no regular physical activity (i.e., fewer than two 20-min bouts of exercise per week during the previous 6 months). None of the study subjects were on a glucose-lowering agent, and only one of the female study subjects was on hormone replacement therapy. Eligible study subjects gave written informed consent prior to their participation, and all protocols were approved by the institutional review board of the George Washington University Medical Center.

Screening

All interested older subjects underwent an initial screening visit at the Beltsville Human Nutrition Research Center (BHNRC) at the U.S. Department of Agriculture (USDA), during which time, a fasting blood sample was obtained in order to rule out cases of undiagnosed type 2 diabetes and to identify those at risk for IGT (i.e., a fasting blood glucose concentration 105–125 mg dL⁻¹). On a separate day, all eligible older subjects underwent a submaximal treadmill walking protocol (14) in the Clinical Exercise Physiology Laboratory at the George Washington University in order to identify any electrocardiographic abnormalities related to undiagnosed cardiovascular disease and to determine the heart rate corresponding to an absolute intensity level of 3 METs [MET value = VO_2 (mL (kg min)⁻¹)/3.5] (15). Heart rate was continuously recorded using a 12-lead electrocardiograph (Cardiac Science, Seattle, WA) while blood pressure was measured by auscultation. Oxygen consumption (VO_2) was determined by sampling expired gas fractions of CO_2 and O_2 from a mixing chamber (ParvoMedics, Sandy, UT). These measurements were corrected to standard conditions and used to determine VO_2 at 20-s intervals throughout the test.

Study protocols

After screening and body composition assessments, eligible subjects underwent three study protocols, which were randomly ordered and spaced 4 weeks apart. Each of the study protocols comprised a 48-h stay in a whole-room calorimeter, with the first day serving as the control day and the second as the exercise day. On the first day of a study protocol, subjects reported to the BHNRC at 7:30 A.M. in the fasted state. The sensor portion of a continuous glucose monitor (CGM) first was placed subcutaneously at the level of the umbilicus and was allowed to initialize for 5 min prior to being connected to the CGM monitor. During CGM initialization, an intravenous catheter was placed in an antecubital vein, and a fasting blood sample (5 cc) was obtained for determination of basal glucose and insulin concentrations. Subjects then entered the calorimeter where they stayed for the remaining 48 h. Subjects were instructed to remain inactive. Each calorimeter contained a bed, toilet, sink, treadmill, television, and personal computer, and since there was little room leftover, subjects had virtually no opportunity to move around, except to use the toilet. An observation window to the calorimeter also allowed investigators to monitor behavior over the entire 48 h. Standardized meals (~ 32 kcal [kg day]⁻¹; 53% carbohydrate/31% fat) were provided for subjects by the BHNRC metabolic kitchen.

Energy expenditure (EE) and substrate utilization were measured continuously in the calorimeter by respiratory exchange (VCO_2/VO_2). VO_2 and CO_2 production (VCO_2) were determined by analyzing the mass and composition of airflow into and out of the calorimeter by standard mass spectrometry procedures (16,17). The minute-by-minute values of VO_2 and VCO_2 were then calculated using the technique developed at BHNRC (18).

Blood samples (5 cc each) were collected through a portal in the calorimeter prior to and 30 min after the start of each standardized meal for the determination of postmeal responses in insulin. Due to the difficulties in maintaining the intravenous catheter over 48 h in the calorimeter, insulin samples were collected over the control day only in order to document any diurnal patterns in secretion over the course of the day. Interstitial glucose readings from the CGM were calibrated against whole-blood glucose readings four times per day using a OneTouch

UltraLink glucometer (LifeScan, Inc., Milpitas, CA). After the postdinner blood draw, the intravenous catheter was removed.

On the second (exercise) day of the testing protocol, subjects awoke in the calorimeter at $\sim 7:00$ A.M. and a fasting blood sample (finger stick) was obtained for determination of basal concentrations of blood glucose. At $\sim 8:00$ A.M., subjects ate a standardized breakfast, which was consumed by 8:30 A.M. After breakfast, subjects performed one of three exercise protocols: 1) postmeal exercise (performed 30 min after completion of each meal for 15 min duration), 2) sustained morning exercise (performed at 10:30 A.M. for 45 min duration), or 3) sustained afternoon exercise (performed at 4:30 P.M. for 45 min duration). All exercise was performed under supervision on the calorimeter treadmill at an absolute intensity of 3.0 METs. The total volume of exercise (45 min/day at 3.0 METs) approximates recently published physical activity recommendations for older adults (19). Upon completion of the exercise protocol(s), subjects remained inactive in the calorimeter for the rest of the day and night. Similar to the previous day, the CGM was calibrated four times with blood glucose readings taken from the glucometer. The next morning, the CGM sensor was removed, and after breakfast, subjects left the calorimeter and returned home.

Continuous glucose monitoring

Whole-day interstitial glucose profiles were collected over 48 h using a CGM (MiniMed, Sylmar, CA). The device provides glucose pattern and trend data up to 288 times per day for 3 days (20,21), thus providing near continuous details of the direction, magnitude, duration, and frequency of fluctuation in glucose concentrations (22). The CGM system measures interstitial glucose by converting glucose at a glucose oxidase interface to hydrogen peroxide, which is then oxidized to produce an amperometric signal (20). This signal is proportional to the interstitial glucose concentration and is stored in the monitor. The stored amperometric data are then transferred and converted to glucose concentrations after data collection is completed using an infrared link to a personal computer and are analyzed using the CGM systems solution software (version 3.0B). Accuracy of the CGM appears higher in the euglycemic compared with the hypoglycemic range and is highest in hyperglycemia (23,24). Various

summary data over 24-h periods can be generated by the CGM and we considered the following: 1) 24-h averaged glucose concentrations, 2) averaged 3-h postlunch glucose concentrations, and 3) averaged 3-h postdinner glucose concentrations in our analysis. Because the CGM takes ~ 2 h to calibrate properly after sensor placement, we did not have 3-h postbreakfast values on the control day to compare with measured postbreakfast values on the exercise day.

Insulin analyses

All blood samples were placed in prechilled test tubes. Samples were centrifuged at 4°C and the plasma stored at -20°C until analyzed in the Core Laboratory of the USDA laboratories. Serial samples for each subject were analyzed in duplicate within a single assay. Plasma insulin concentrations were determined with an ELISA (Millipore Corporation, Billerica, MA). Observed inter- and intra-assay coefficients of variation were 2.98 and 5.33%, respectively.

Body composition

Height and weight were measured on a stadiometer and balance-beam scale, and the BMI ($\text{BMI} = \text{kg m}^{-2}$) was used as an indicator of relative weight and overall "obesity." Overall body composition (whole-body muscle [kg] and fat mass [kg]) scans also were obtained using dual energy X-ray absorptiometry (Prodigy; General Electric Health Care, Pittsburgh, PA). Percent body fat (overall and site-specific) obtained from the dual energy X-ray absorptiometry scan also served as an indicator of obesity.

Statistical analysis

Univariate statistics (mean \pm SD and frequencies [%]) first were generated on all study variables. Mean pre- and 30-min postmeal insulin values were compared across the control day using ANOVA. Within-subject change (simple difference and percent change [% Δ]) in the mean values of the CGM summary variables between the control day and the experimental day first were determined within each exercise condition. We also calculated changes in the 24- and 3-h postmeal, areas under the curve (AUCs) from the CGM data using the trapezoidal method within each condition. Mean differences in these changes then were compared across the three experimental conditions using repeated-measures ANOVA. Specific comparisons of interest among

each of the three exercise conditions were tested using orthogonal contrast statements.

RESULTS—On average, subjects were nearly 70 years of age (69 ± 6 years) with class I obesity ($\text{BMI } 30 \pm 5 \text{ mg kg}^{-2}$) and $>40\%$ ($40.8 \pm 7.2\%$) of their body fat distributed around the truncal region. Subjects had impaired fasting glucose ($109 \pm 6 \text{ mg dL}^{-1}$) and fasting insulin values ($9.8 \pm 4.5 \mu\text{U mL}^{-1}$) in the normal range for this age-group (25). Resting systolic ($130 \pm 17 \text{ mmHg}$) and diastolic ($81 \pm 6 \text{ mmHg}$) blood pressures were also in the normal range for older people. The treadmill speed necessary to achieve an exertion level of 3.0 METs ranged from 2.1 to 3.5 mph, with an average speed of 3.0 ± 0.4 mph. Averaged values for the respiratory exchange ratio over 24 h did not differ between the control and the exercise day for any of the exercise conditions (0.85 ± 0.05 vs. 0.86 ± 0.06 for the 45-min morning walking condition, 0.81 ± 0.10 vs. 0.82 ± 0.07 for the 45-min afternoon walking condition, and 0.86 ± 0.01 vs. 0.87 ± 0.04 for the postmeal condition, respectively). Average EE between the control and exercise days were 1.96 ± 0.42 vs. $2.36 \pm 0.53 \text{ kcal min}^{-1}$ for the morning walking condition, 1.94 ± 0.39 vs. $2.28 \pm 0.50 \text{ kcal min}^{-1}$ for the afternoon condition, and 1.91 ± 0.60 vs. $2.39 \pm 0.67 \text{ kcal min}^{-1}$ for the postmeal condition ($P < 0.05$).

These differences in EE between the control and the exercise days were not significantly different across the three exercise conditions, however ($P < 0.20$).

Insulin values over the control days were similar for all three trials and, therefore, the data were pooled. Both pre- and 30-min postmeal insulin values also appeared in the normal range, although we observed a significantly blunted β -cell response at midday. Insulin concentrations 30 min postlunch were significantly lower than 30-min postbreakfast values (37.9 ± 33.4 vs. $59.4 \pm 53.5 \mu\text{U mL}^{-1}$; $P < 0.001$), and predinner insulin concentrations were significantly lower than prelunch concentrations (7.4 ± 3.4 vs. $14.2 \pm 13.4 \mu\text{U mL}^{-1}$; $P < 0.001$). Indeed, the 0–30-min change in insulin concentrations in response to each of the meals was 60% from breakfast, 40% from lunch, and 54% from dinner ($P < 0.01$).

Continuous mean data on glucose kinetics from CGM over the exercise day only (Fig. 1) show substantial variability in postmeal glycemia by exercise condition. Postmeal exercise (broken line) appeared to halt a further increase in postprandial glucose levels, resulting in a blunted glucose excursion that was particularly evident after the evening meal at 6:00 P.M. When compared with sustained afternoon (predinner) exercise (dotted line), the benefits of postmeal exercise in the evening continued overnight

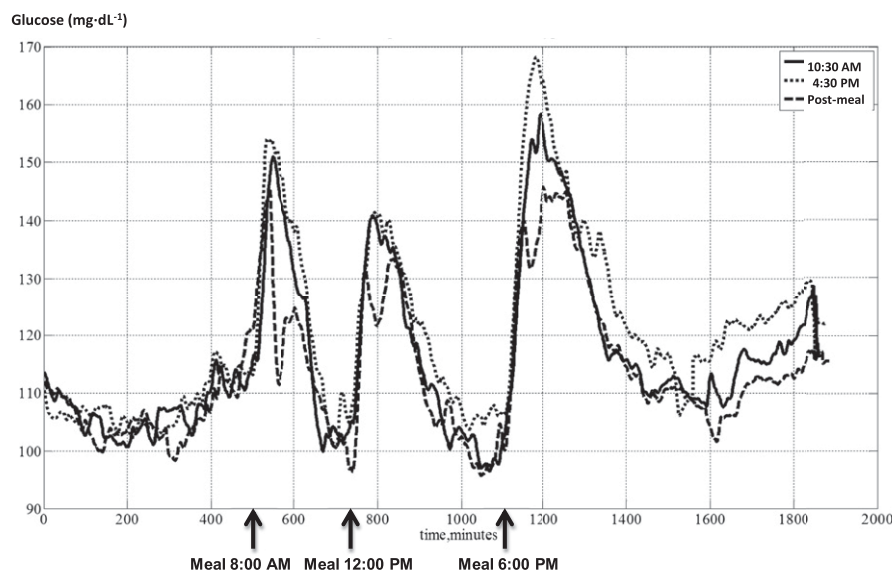


Figure 1—Mean continuous glucose concentrations from CGM over the exercise day according to exercise condition. Data represent exercise at 10:30 A.M. for 45 min (solid line), at 4:30 P.M. for 45 min (dotted line), and postmeal (broken line). To convert to Système International units ($\mu\text{mol/kg min}^{-1}$), multiply glucose values by 5.5.

until 7:00 A.M. when the sensor was removed. Between 1,300 and 1,600 min (~9:40 P.M. to 2:40 A.M.), the slope of decline in postdinner glucose values appeared comparable between the postmeal and the sustained morning exercise (solid line) conditions; however, the relative benefits of postmeal exercise increased thereafter.

Differences in averaged 24-h glucose concentrations between the control and exercise days according to exercise trial are presented in Fig. 2. Three 15-min bouts of postmeal walking ($P < 0.03$) and one 45-min bout of sustained morning walking ($P < 0.05$) were equally effective in significantly reducing 24-h glucose concentrations between the 2 days ($\% \Delta = 10\%$ for postmeal and 8% for sustained morning exercise, respectively). In contrast, 45 min of sustained walking in the afternoon had a negligible impact on averaged 24-h glycemic control. The corresponding improvements in glycemic control as expressed by the 24-h AUC reflect a similar trend (Table 1).

Postmeal walking and 45 min of sustained morning walking both appeared to lower 3-h postlunch glucose concentrations (measured at ~3:30 P.M.), as well as the 3-h AUC for lunch, although these trends were not statistically significant (Fig. 3A and Table 1). Since sustained afternoon walking was performed at 4:30 P.M., it was not possible for it to impact 3-h postlunch glucose values. Thus, the marked increase in postlunch glucose concentrations observed under

this condition between days 1 and 2 reflects nearly 30 h of total sedentary behavior in the calorimeter. On the other hand, postmeal exercise was the only exercise prescription to significantly reduce 3-h postdinner glucose levels (measured at ~9:30 P.M.) ($P < 0.01$) and the 3-h AUC for dinner ($P < 0.03$) relative to the control day (Fig. 3B and Table 1). Of note in Fig. 3B is the degree of hyperglycemia that extends up to 3 h postmeal. Sustained exercise at 10:00 A.M. had a negligible effect on subsequent postdinner glycemia, and exercise at 4:30 P.M. (i.e., premeal) appeared to increase 3-h glycemic responses to the subsequent dinner meal compared with the control day. We observed that improvements in 24-h glucose values were significantly correlated with improvements in 3-h postdinner values ($r = 0.88$; $P < 0.001$), but not with improvements in 3-h postlunch values ($r = 0.44$; $P = 0.21$) or with fasting blood glucose ($r = 0.30$; $P = 0.40$).

CONCLUSIONS—We observed that 15 min of walking performed 30 min after each meal was equally effective as 45 min of sustained morning walking in significantly improving 24-h glycemic control in older people at risk for IGT. Moreover, postmeal exercise was effective in significantly lowering 3-h postdinner glucose levels, whereas the other exercise prescriptions were not. All walking was performed at an absolute intensity of 3.0 METs, with a volume of 45 min/day; the differences between the three treatments

were the frequency (one sustained vs. three intermittent bouts) and the timing of the exercise (A.M., P.M., or 30 min postmeal). Moreover, the exertion level of the exercise was barely of moderate intensity, suggesting that the timing of exercise may be as important (if not more) as volume and intensity in determining the best exercise prescriptions for glycemic control in older people. Similar to some pharmacologic treatments, a smaller exercise dose repeated several times per day may provide greater overall benefits than a single large dose taken once per day. This may be especially so if older people are more tolerant of smaller exercise doses and are better able to adhere to multiple frequencies of such smaller doses on a regular basis. Finally, improvements in 24-h glucose values were strongly correlated with improvements in 3-h postdinner values, suggesting that an after-dinner walk may have the greatest relative benefits for overall daily glucose homeostasis.

Other studies have investigated the role of exercise timing on glycemic responses to meals, primarily among younger or middle-aged people with type 2 diabetes and with the exercise timing ranging from premeal (26,27), immediately postmeal (25,28), 15–45 min postmeal (29,30), and 2 h postmeal (31). Studies of premeal (i.e., postabsorptive) exercise generally report either no effect on postchallenge glycemia (29,30) or an increase in the glycemic effect of a meal (26), which corroborates our findings regarding the effects of exercise performed at 4:30 P.M. on the subsequent dinner meal. Studies of postmeal exercise report improvements in postprandial glycemic control for as long as 4 h postchallenge, but not in response to the next meal (30), suggesting that these benefits are short-lived. We timed our three 15-min bouts of exercise to occur at 30 min postmeal, which would correspond to the period of absorption, thereby maximizing the potential for glucose uptake by the contracting muscles and for glucose oxidation due to its being the prevailing fuel source (32). Also, we repeated the dose after each meal in an effort to extend the benefits of exercise for as long as possible over 24 h. Thus, what the postmeal exercise lacked in duration (15 vs. 45 min), it made up for in frequency (three times per day vs. once per day). Our use of CGM enabled us to study glucose kinetics across the control and the entire exercise day, which is a limitation to the previous studies of postmeal exercise.

24-h averaged glucose (mg·dL⁻¹)

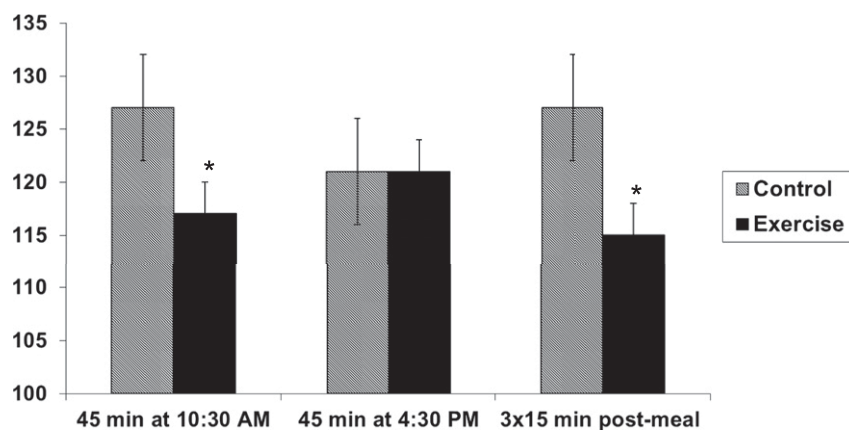


Figure 2—Differences in mean 24-h averaged glucose concentrations between the control day (hashed bars) and the exercise day (solid bars) (N = 10). All walking was performed on a treadmill at a 3.0 MET exertion level. * $P < 0.05$ between control and exercise day. To convert to Système International units ($\mu\text{mol}/\text{kg min}^{-1}$), multiply glucose values by 5.5.

Table 1—Areas under the glucose response curve (AUCs) between the control and exercise days according to exercise condition

AUC variable	Time of exercise	Control day	Exercise day
AUC ₂₄ (mg dL ⁻¹) 24 h 10 ³	Postmeal	176 ± 27	148 ± 17*
	10:30 A.M.	176 ± 28	147 ± 22*
	4:30 P.M.	175 ± 23	159 ± 24
AUC _{Dinner} (mg dL ⁻¹) 3 h 10 ³	Postmeal	26 ± 5	24 ± 4*
	10:30 A.M.	25 ± 5	25 ± 5
	4:30 P.M.	24 ± 3	26 ± 5
AUC _{Lunch} (mg dL ⁻¹) 3 h 10 ³	Postmeal	23 ± 5	22 ± 4
	10:30 A.M.	24 ± 4	23 ± 4
	4:30 P.M.	21 ± 5	24 ± 4

Data represent mean ± SD. AUC₂₄, 24-h AUC; AUC_{Dinner}, 3-h AUC in response to the evening meal; AUC_{Lunch}, 3-h AUC in response to the noon meal. *Significantly lower than control day at $P < 0.05$.

As stated previously, exercise can supplement peripheral insulin action in the presence of a low or blunted insulin secretory response (11,12). Insulin data collected on the control day only suggested a midday secretory response (i.e., 0–30-min change in lunchtime concentrations) that was significantly lower than the response to breakfast. A blunted insulin response at this time of day may have exacerbated the effects of ~30 h of inactivity in the calorimeter prior to the late afternoon exercise condition, as indicated by a significantly greater 3-h

postlunch glucose on the experimental relative to the control day. Other studies have documented this diurnal pattern in β -cell response, which appears more frequently in older age (13). To our knowledge, however, we are the first to investigate how exercise timing may interact with these secretory patterns in improving glycemic control. Unfortunately, we were not able to collect blood over the exercise day, which would have allowed us to interpret the exercise-related glycemic responses directly in relation to the prevailing insulin levels.

EE increased significantly between the control and the exercise day, whereas energy intake remained constant. We assume that this imbalance would only improve glycemic control on the exercise day. We held the volume of exercise (45 min/day at 3 METs) constant over the three trials and observed that the increase in EE between the control and the exercise day did not differ significantly across the three trials. We assume, therefore, that any effects of energy imbalance on glycemic control were nondifferential across the three trials, which would attenuate our effect sizes toward the null. We also note the relatively healthy and self-selected nature of our study sample. The small sample size may have compromised our statistical power, and the convenience sample no doubt precludes the generalizability of the findings to the general population of older people. On the other hand, each subject served as their own control, and the multiple crossover design of the study certainly enhanced both the internal validity and the reliability of these data. Furthermore, our findings in this study sample may have even greater implications for the manner in which exercise is prescribed for those less robust older adults already demonstrating frank clinical impairments in

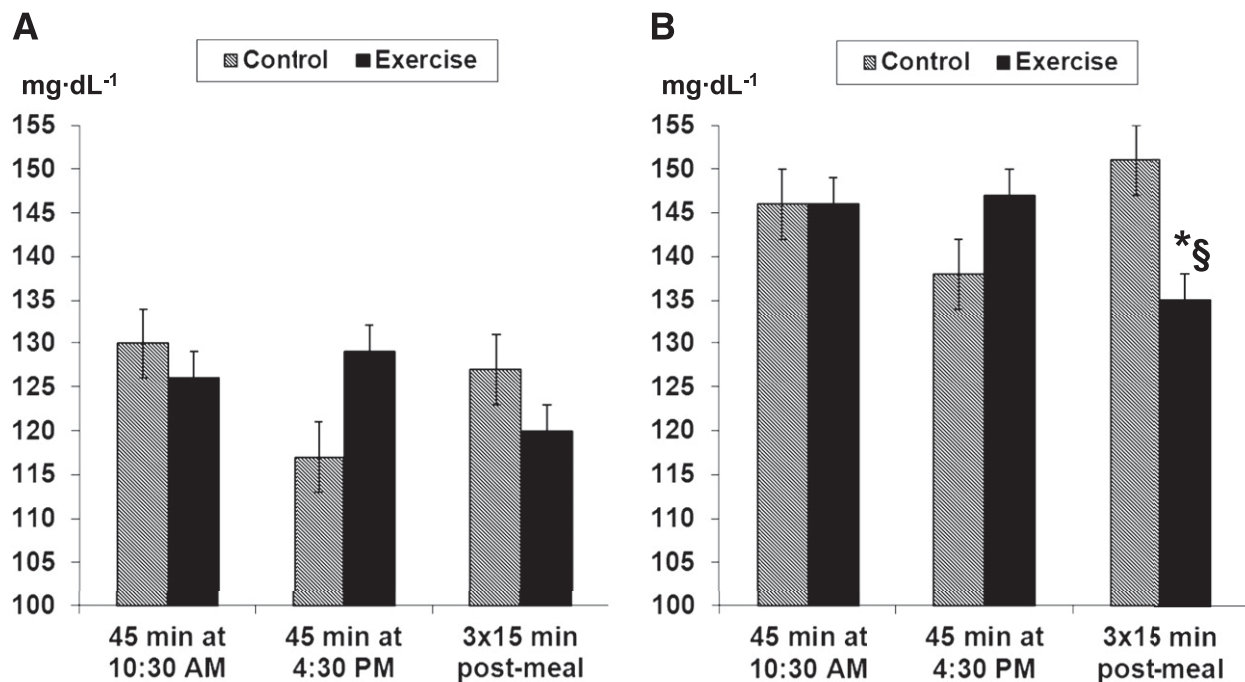


Figure 3—Differences in 3-h postlunch (A) and postdinner (B) glucose concentrations between the control day (hashed bars) and the exercise day (solid bars) ($N = 10$). All walking was performed on a treadmill at a 3.0 MET exertion level. * $P < 0.01$ between control and exercise day; § $P < 0.05$ compared with sustained exercise in the morning or afternoon. To convert to Système International units ($\mu\text{mol}/\text{kg min}^{-1}$), multiply glucose values by 5.5.

β -cell function and in postprandial glycemic control.

In short, intermittent bouts of postmeal walking appear to be an effective way to control postprandial hyperglycemia in older people. The clinical relevance of shorter, but more frequent, bouts of lower- to moderate-intensity walking (compared with the standard recommendation of 30–45 min of sustained moderate-intensity walking) is substantial, and this may be especially so among older people who may feel more capable of engaging in intermittent physical activity on a daily basis. In fact, this type of exercise can be coupled with common daily activities such as dog walking or performing short errands. Given the excess disease burden associated with hyperglycemia in older age, and the recognized value of non-communicable disease prevention, there are enormous public health benefits to designing exercise programs that are enjoyable and effective within the populations needing them the most.

Acknowledgments—This work was supported in part by National Institutes of Health/ National Institute on Aging Grant R21-AG-031550 (to L.D.P.) and by the BHNRC, Agricultural Research Service, USDA.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the USDA.

No potential conflicts of interest relevant to this article were reported.

L.D.P. designed and conducted the study, analyzed the data, and wrote the manuscript. A.G. analyzed data and contributed to the writing of the manuscript. M.S.S. assisted in data collection and performed insulin analyses. L.F.H. screened study subjects and contributed to the writing of the manuscript. W.R. assisted in conducting the study, analyzed data, and contributed to the writing of the manuscript. L.D.P. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

The authors thank the staff of the BHNRC metabolic kitchen and the study subjects for their commitment to the study protocol.

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