Duration of Exercise as a Key Determinant of Improvement in Insulin Sensitivity in Type 2 Diabetes Patients

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Exercise duration and intensity are important parameters in exercise prescription and play a major role in improving insulin sensitivity (including transient and persistent improvement effects following cessation of training) in patients with type 2 diabetes mellitus (T2DM). However, whether duration or intensity of exercise is the more important factor has yet to be established. Therefore, we aimed to determine whether exercise prescriptions differing in duration and intensity differ in their ability to aid T2DM patients to retain insulin sensitivity following the conclusion of a period of training. Sedentary T2DM patients (age 51.2 ± 1.3 years) were assigned to either a low-intensity (50% VO2peak, n = 27) or a high-intensity exercise group (75% VO2peak, n = 28), and followed a 12-week exercise program of 5 sessions/week and 240 kcal/session. Insulin sensitivity (oral glucose tolerance test, ISI) was measured when subjects were sedentary and at 16-24 h and 15 days after the final training bout. The low-intensity group spent more training time to training per exercise session than the high-intensity group (56.1 ± 3.0 min/session vs. 34.3 ± 2.4 min/session) (P < 0.01), but the total amount of energy expended was the same. ISI was increased in both groups 16-24 h after the final training session, but only the low-intensity group still had elevated ISI 15 days after the cessation of training. These findings suggest that in T2DM patients, the persistent training-induced improvements in insulin sensitivity may be more dependent on exercise duration than exercise intensity in regimens with the same level of energy expenditure.

Keywords: energy expenditure; exercise duration; exercise intensity; insulin sensitivity; type 2 diabetes mellitus


Type 2 diabetes mellitus (T2DM) is a metabolic disease characterized by insulin resistance, relative β-cell dysfunction, and resultant hyperglycemia that contribute to the development of both microvascular and macrovascular diseases. Given the increase prevalence of T2DM in adults over the past few decades in developed and developing countries (Fox et al. 2006; Yang et al. 2010), strategies aimed at the prevention and treatment of diabetes are needed.

Physical activity is an important component in the management of T2DM. Many studies have shown positive health benefits with low-intensity exercise (Dunstan et al. 1997; Poirier et al. 2002; Soman et al. 1979; Tessier et al. 2000). Greater intensities of exercise tend to yield even greater benefits (1996), particularly changes in hemoglobin HbA1c levels and aerobic capacity (Mourier et al. 1997).

Very high intensities of cardiorespiratory exercise (e.g., 75% of peak O2 consumption [VO2peak]) have been associated with considerable improvements in Hba1c and cardiorespiratory fitness levels, but such demanding exercise intensities may not be well tolerated by all patients with T2DM. Because the magnitude of the beneficial effects of exercise has been linked to total energy expenditure (Larsen et al. 1999), higher intensities of exercise might be recommended since they allow the same total energy expenditure to be achieved in shorter time periods, which may facilitate compliance. However, the possibility of differences in the beneficial effects of low-intensity and high-intensity exercise training programs in T2DM remains to be established.

Exercise-mediated improvements in insulin sensitivity have been demonstrated; unfortunately, these desirable
responses are typically short-lived because insulin sensitivity can decline significantly after as little as 38 h after the final exercise training bout (King et al. 1995; Goodyear and Kahn 1998). This transient response should be considered when designing physical activity programs for the general populace, because the magnitude of the improvement in insulin action, and the ability to retain this improvement when not exercising have to be delineated and optimized. To date, despite the importance of understanding the ramifications of these relationships, there is very little information on the long-term maintenance of exercise-induced improvements in patients with T2DM.

Several studies have indicated that training-related improvements in insulin action are retained after 10 days without physical activity in master athletes (Rogers et al. 1990), and after 15 days of inactivity in overweight/obese subjects (Bajpeyi et al. 2009). However, to our knowledge, there is no other information concerning the retention characteristics of insulin action after various exercise prescriptions in T2DM. Therefore, the purpose of the present investigation was to define the effects of low intensity (50% VO_{peak}) and high intensity (75% VO_{peak}) exercise performed with the same total energy expenditure on insulin sensitivity. More importantly, we aimed to determine whether there are differences in the effects of these 2 exercise regimes. Persistence of exercise-mediated improvement in insulin action could provide guidance for designing exercise-training regimens in the treatment of diabetes.

**Methods**

**Subjects**

Sixty sedentary adults with T2DM, aged between 40 and 60 years, were recruited from diabetes outpatient clinics in the Metabolic Disease Hospital of Tianjin Medical University. Diabetes was defined according to established criteria (Yang et al. 2010) and was controlled by diet and/or oral hypoglycemic agents. Exclusion criteria were current insulin therapy; participation in exercise two or more times per week on treadmill (SportsArt 6300, SportsArt Fitness, Shenzhen, China) at the Rehabilitation and Sports Center of Tianjin Medical University. A 5-min warm-up and cool-down period was included before and after the exercise session, which included light aerobic exercise and appropriate stretching. Training gradually progressed in duration and intensity. In the first week all exercise group subjects performed 15 min treadmill exercise in one session at an intensity of 50% VO_{peak}. In the second week, all subjects performed 2 × 15 min bouts of treadmill exercise at an intensity of 50% VO_{peak}. In the 3rd and 4th week, the low-intensity exercise group subjects performed 2 × 20 min bouts at an intensity of 50% VO_{peak}, and the high-intensity exercise group subjects performed 2 × 15 min bouts at an intensity of 65% VO_{peak}. During sessions in the 5th-12th weeks, the low-intensity group and high-intensity exercise group subjects performed 2 × 120 kcal energy expenditure bouts of treadmill exercise at an intensity of 50% VO_{peak} and 75% VO_{peak}, respectively. Heart rate monitors (RS800, Polar Electro Oy, Kempele, Finland) were used to adjust workload to achieve the target heart rate and energy expenditure. The target heart rate corresponding to the targeted intensity achieved during the VO_{peak} test. The exercise regimes (1,200 kcal/week, 240 kcal/session × 5 sessions/week) were consistent with recommendations of the Surgeon General’s report. After the 12 weeks of exercise training, the subjects were asked to cease all exercise and perform only normal daily activities for the subsequent two weeks; they were offered the incentive that they could reenter the exercise training program for a further 3 months at no cost after adhering to the withdrawal period.

Background physical activity was assessed and verified by using pedometers (Walking Style HJ-301, Omron Healthcare Co., Ltd, Kyoto, Japan) of each subject during all experimental period (including 2 weeks of baseline). The measurement was made for the average daily steps of a full week, but the devices were removed when showering, sleeping, or stepping during scheduled exercise sessions. All subjects were required to not change their daily normal activities between enrollment and the 14-weeks of the experimental period. When the average daily steps was increased > 2,000 steps/day compared with that in the baseline, it might be considered clinically significantly changed of background physical activity (Bravata et al. 2007). All subjects indicated that they had abstained from structured exercise and had not changed their dietary habits during the course of the study.

**Clinical examination**

**Oral glucose tolerance test (OGTT) and blood sample collection:** Clinical data were obtained at baseline, after 16-24 hours and 14-16 days after the final exercise training bout in 2 exercise groups at baseline, week 12, and week 14. All subjects arrived at the laboratory after a 12-hour overnight fast, and after a brief rest period, underwent an OGTT. This involved consumed a drink containing 75 g of anhydrous glucose in 300 mL of water; with blood samples being taken at 30-minute intervals for 120 minutes. Blood samples were collected into potassium EDTA tubes (Vacutainer; BD, Oxford, United Kingdom) and immediately placed on ice. Within 15 minutes of the samples collection, they were centrifuged for 15 minutes at 3,000 rpm. Plasma was then dispensed into 0.5-mL aliquots and stored at −80°C until analysis. A Roche Modular P (Roche Diagnostic
Pulse wave velocity (PWV) and blood pressure: Subjects lay supine for at least 30 minutes before all PWV measurements. Blood pressure was measured using an automated blood pressure monitor (Omron HEM-70801c; Omron Healthcare, Japan). On each occasion, three measurements of blood pressure were taken; and the lowest of these values was used for analysis. Once blood pressure measurements were conducted, carotid-femoral PWV was performed using the Omron to provide an index of arterial stiffness. Pulse transit time was determined using pressure transducers placed over the carotid and femoral pulses with the Complior software (Artech Medical). Measurements of PWV were conducted, carotid-femoral PWV was performed using the Omron to provide an index of arterial stiffness. Pulse transit time was determined using pressure transducers placed over the carotid and femoral pulses with the Complior software (Artech Medical) establishing the propagation time from the carotid to femoral artery. The transit distance was measured as the superficial distance between the two pressure transducers. Thus, the PWV was calculated as the transit distance divided by the transit time.

Peak \( V_{\text{O2}} \) testing: \( V_{\text{O2peak}} \) was determined using a modified Bruce treadmill protocol exercise test (Silva et al. 2007) by a cardiorespiratory fitness analysis system (Metalyzer 3B, Cortex, Germany). An initial test was performed to screen for underlying cardiovascular disease and to acclimate the subject. If no evidence for cardiovascular disease was detected, a second test was conducted to determine the pretraining \( V_{\text{O2peak}} \). The first two stages of the Modified Bruce Test are performed at 1.7 mph and 0% grade and 1.7 mph and 5% grade, and the third stage corresponds to the first stage of the Standard Bruce Test protocol which continues as 1.7 mph 10% grade, 2.5 mph 12% grade, 3.4 mph 145 grade. Per stage takes 3 minutes. The last 30 s were averaged to determine \( V_{\text{O2peak}} \). The data of \( V_{\text{O2peak}} \) were normalized for body mass. \( V_{\text{O2peak}} \) tests were performed after the OGTT test and PWV measurement at the baseline, week 12 and week 14. In addition, two times additional \( V_{\text{O2peak}} \) tests were performed at the 4th-5th and 8th-9th week in order to modify the exercise workload.

Anthropometrics: Body mass was measured to the nearest tenth of a kilogram on a digital electronic scale before \( V_{\text{O2peak}} \) tests was determined. Height was measured to the nearest 0.5 cm with a stadiometer, and body mass index (BMI) was calculated as mass/height\(^2\). Total body fat was measured using a dual-energy X-ray absorptiometry scanner (Lunar Prodigy, GE Medical systems, USA).

Statistical Analysis

Data were compared with repeated-measures ANOVA and contrast-contrast comparisons. Relative changes were compared with between-groups ANOVA. Statistical significance was denoted at the \( P < 0.05 \) level. All data are presented as means ± s.e.

Results

Baseline characteristics

Five subjects \( (n = 3 \) in the low-intensity, \( n = 2 \) in the high-intensity group) dropped out of the study because of either loss of motivation, change of medication, or work commitments. The baseline characteristics of subjects who completed the study are shown in Tables 1 and 2. All the baseline characteristics were not statistically different between the two exercise groups.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Low-intensity ( (n = 27) )</th>
<th>High-intensity ( (n = 28) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (male/female)</td>
<td>15/12</td>
<td>15/13</td>
</tr>
<tr>
<td>Age (y)</td>
<td>52.0 ± 1.3</td>
<td>50.3 ± 1.2</td>
</tr>
<tr>
<td>Duration of diabetes (y)</td>
<td>5.1 ± 0.9</td>
<td>4.2 ± 0.6</td>
</tr>
<tr>
<td>Medications, ( n ) (%) Oral hypoglycemic agents</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>21 (78)</td>
<td>23 (82)</td>
</tr>
<tr>
<td>Metformin</td>
<td>19 (70)</td>
<td>20 (71)</td>
</tr>
<tr>
<td>Sulfonylurea</td>
<td>5 (19)</td>
<td>7 (25)</td>
</tr>
<tr>
<td>Meglitinide</td>
<td>3 (11)</td>
<td>5 (18)</td>
</tr>
<tr>
<td>( \alpha )-Glucosidase inhibitor</td>
<td>3 (11)</td>
<td>5 (18)</td>
</tr>
<tr>
<td>Thiazolidinedione</td>
<td>4 (15)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Antihypertensive agents</td>
<td>10 (37)</td>
<td>9 (32)</td>
</tr>
<tr>
<td>Lipid-lowering agents</td>
<td>5 (19)</td>
<td>7 (25)</td>
</tr>
<tr>
<td>Other</td>
<td>7 (27)</td>
<td>9 (32)</td>
</tr>
</tbody>
</table>
Exercise training

To achieve the prescribed energy expenditures (240 kcal/session), the low-intensity groups devoted significantly more time to training per exercise session than the high-intensity groups (56.1 ± 3.0 min/session vs. 34.3 ± 2.4 min/session) (*P < 0.01).

Insulin action

As the shown in Fig. 1, ISI was significantly increased in the low-intensity (16-24 h, 5.4 ± 0.6 vs. baseline, 3.4 ± 0.4; *P < 0.05) and high-intensity (16-24 h, 4.8 ± 0.5 vs. baseline, 3.6 ± 0.4; *P < 0.05) groups at 16-24 h after the final exercise training bout compared with that at baseline, respectively. After 15 days of training cessation, ISI in the high-intensity exercise group declined to a value that was not significantly different from the sedentary pre-training level. In contrast, in the low-intensity group’s insulin action (ISI) at 15 days remained significantly elevated compared with the sedentary pre-training condition (*P < 0.05).

Table 2. Characteristics of both groups at the three 3 points.

<table>
<thead>
<tr>
<th></th>
<th>Low-intensity (n = 27)</th>
<th></th>
<th>High-intensity (n = 28)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>16-24 h</td>
<td>15 days</td>
<td>Baseline</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.9 ± 0.6</td>
<td>25.4 ± 0.6*</td>
<td>25.3 ± 0.7*</td>
<td>26.1 ± 0.7</td>
</tr>
<tr>
<td>Total body fat (%)</td>
<td>28.0 ± 1.4</td>
<td>27.1 ± 1.3*</td>
<td>27.0 ± 1.3*</td>
<td>30.1 ± 1.4</td>
</tr>
<tr>
<td>HbA₁c (%)</td>
<td>6.7 ± 0.2</td>
<td>6.7 ± 0.2</td>
<td>6.7 ± 0.2</td>
<td>6.6 ± 0.2</td>
</tr>
<tr>
<td>Fasting glucose (mmol/l)</td>
<td>7.5 ± 0.4</td>
<td>7.3 ± 0.3</td>
<td>7.4 ± 0.3</td>
<td>7.1 ± 0.4</td>
</tr>
<tr>
<td>Fasting insulin (µU/ml)</td>
<td>14.3 ± 1.1</td>
<td>11.0 ± 0.9*</td>
<td>13.5 ± 1.0</td>
<td>13.7 ± 1.0</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>4.8 ± 0.3</td>
<td>3.6 ± 0.2*</td>
<td>4.5 ± 0.2</td>
<td>4.3 ± 0.2</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>5.2 ± 0.2</td>
<td>5.0 ± 0.2</td>
<td>4.9 ± 0.2</td>
<td>5.5 ± 0.3</td>
</tr>
<tr>
<td>HDL-C (mmol/l)</td>
<td>1.4 ± 0.1</td>
<td>1.5 ± 0.1</td>
<td>1.4 ± 0.1</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>LDL-C (mmol/l)</td>
<td>3.1 ± 0.2</td>
<td>3.2 ± 0.2</td>
<td>3.0 ± 0.3</td>
<td>3.3 ± 0.2</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>1.8 ± 0.2</td>
<td>1.9 ± 0.3</td>
<td>1.8 ± 0.2</td>
<td>1.9 ± 0.3</td>
</tr>
<tr>
<td>DBP</td>
<td>74 ± 3</td>
<td>73 ± 2</td>
<td>75 ± 3</td>
<td>76 ± 2</td>
</tr>
<tr>
<td>SBP</td>
<td>125 ± 3</td>
<td>120 ± 3*</td>
<td>125 ± 3</td>
<td>127 ± 3</td>
</tr>
<tr>
<td>PWV (m/s)</td>
<td>13.7 ± 0.4</td>
<td>13.6 ± 0.5</td>
<td>13.8 ± 0.5</td>
<td>13.8 ± 0.4</td>
</tr>
<tr>
<td>Background physical activity (steps/day)</td>
<td>5,391 ± 289</td>
<td>5,613 ± 307</td>
<td>5,480 ± 321</td>
<td>5,247 ± 325</td>
</tr>
</tbody>
</table>

Data are presented as means ± s.e. BMI, body mass index; HbA₁c, hemoglobin A₁c; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; DBP, diastolic blood pressure; SBP, systolic blood pressure; PWV, pulse wave velocity. *P < 0.05 vs. baseline in the same group. All data are expressed as means ± s.e.

![Fig. 1. Insulin sensitivity index (ISI) of both groups at the 3 time points. All data are expressed as means ± s.e. *P < 0.05 vs. baseline values for the same group.](image-url)
Other components of insulin action, such as fasting insulin and HOMA-IR demonstrated an exercise training and subsequent detraining effect, with no differences between the two exercise regimes. As shown in Table 2, compared with the pre-training condition, fasting plasma insulin significantly decreased at 16-24 h after the final training bout but it subsequently returned to baseline in both groups. A similar pattern was evident for HOMA-IR (Table 2).

**Cardiorespiratory fitness**

As shown in Fig. 2, VO_{2peak} was significantly increased with 12-weeks exercise training in both the low-intensity and high-intensity groups ($P < 0.05$, 16-24 h vs. baseline in both groups). In the low-intensity group, VO_{2peak} was retained at trained levels even after 15 days of training cessation. In contrast, VO_{2peak} declined significantly at 15 days of training cessation compared with 16-24 h trained values in the high-intensity groups (~5%). However, the VO_{2peak} value 15 days of training cessation remained significantly elevated compared with baseline in both exercise groups ($P < 0.05$ in each).

**Background physical activity**

As the shown in Table 2, the background physical activity (average daily steps) was not significantly changed during the 14-weeks experimental period in either the low-intensity and high-intensity groups (Table 2), suggesting that no subjects had changed daily normal activities during all experimental period.

**BMI and total body fat**

BMI and total body fat were both significantly lower some 16-24 h after training and this decreased further even 15 days after training cessation in both exercise protocols. However, there were no statistically significant differences between the two exercise regimes (Table 2).

**Blood pressure and PWV**

Systolic blood pressure significantly decreased 16-24 h after the final training bout compared with baseline, but this effect was lost by 15 days, in both groups. However, PWV did not differ significantly from baseline at either 16-24 h or 15 days after the final training bout in either group.

**HbA$_1c$, fasting glucose and serum lipids**

As shown in Table 2, 12-weeks of exercise training did not significantly change the HbA$_1c$, fasting glucose, total cholesterol, HDL-C, LDL-C and triglyceride in either the low-intensity and high-intensity groups.

**Discussion**

The aim of the present study was to determine which, if any, clinically relevant exercise prescriptions retain training-induced improvement in insulin action a fortnight after training cessation in patients with T2DM. The main finding of the present study was that insulin action (ISI) remained above pre-training sedentary levels even 15 days after training cessation when the initial exercise intervention was 12-weeks at 50% VO$_{2peak}$ (ie, low-intensity), and that this was not observed when the same amount of energy was expended during a higher intensity intervention (75% VO$_{2peak}$ high-intensity exercise training at 240 kcal/session × 5 sessions/week; Fig. 1). To our knowledge, there is no other information concerning the retention characteristics of insulin action after various exercise prescriptions in T2DM patients.

In the present study, VO$_{2peak}$ significantly increased after 12 weeks of exercise training, and in both low-intensity and high-intensity groups, these benefits were retained at significantly higher levels 15 days after training.
cessation, in comparison with the pre-training levels (Fig. 2). Consistent with our results, other studies have also reported a 0 to –7% decline in maximal O2 consumption (\(\dot{V}O_{2\text{peak}}\)) 10-14 days after training ended (Heath et al. 1983; Coyle et al. 1984; Cullinane et al. 1986). These results suggested that the persistence of training-induced improvements in insulin action does not appear to be due to continuing physical activity by the subjects during the 15-day period following the conclusion of training.

Other studies have also observed a lasting influence of exercise training on insulin action after cessation of training. Results of one previous study demonstrated retention of training-related improvements in insulin action after 10 days without physical activity in master athletes (Rogers et al. 1990). Dela et al. (1995) reported a training-induced increase in insulin action that persisted after 6 days of inactivity in overweight subjects. Data in the present study thus support those of other studies that found a persistent improvement in insulin action beyond the early compensation phase of 2-4 days following a prolonged period of endurance-oriented (longer duration) low-intensity exercise training (Rogers et al. 1990; Dela et al. 1995). In the present study, although changes in insulin sensitivity after 15 days of training were clearly dissimilar to those evident within 16-24 h after the final training session, the fact that retention of benefits was observed at 15 days could be important clinically and may indicate a persistent effect of exercise training on insulin action in T2DM.

Exercise regimens of 2 different intensity levels (low-intensity, 50% \(\dot{V}O_{2\text{peak}}\); high-intensity, 75% \(\dot{V}O_{2\text{peak}}\)) of the same frequency (5 sessions/week) and total energy expenditure (240 kcal/session) were used in this study to determine if variations in the exercise intensity led to differences in persistence of insulin sensitivity after training cessation. The exercise duration in the low-intensity groups (56.1 ± 3.0 min/session) was significantly longer than that in the high-intensity groups (34.3 ± 2.4 min/session, \(P < 0.01\)). As the only variable with a significant effect in the present study, exercise duration may be a critical factor to consider when designing exercise programs to optimize the effects of training-induced improvements in insulin action.

Although data are lacking to explain the possible cellular mechanisms by which the exercise duration prolonged the benefits of insulin action in the present study, several studies have indicated that skeletal muscle mitochondrial content may be an important factor governing insulin action (Kriketos et al. 1996; Bruce et al. 2003). Higher volumes of exercise (i.e., more weekly sessions) may induce more changes in the skeletal muscle mitochondrial content compared with lower-volume exercise regimens of the same intensity (65-85% \(\dot{V}O_{2\text{peak}}\)) 15 days after training cessation (Bajpeyi et al. 2009). However, there is a debate about the magnitude of exercise-induced changes in skeletal muscle mitochondrial content as a function of exercise intensity (40-55% vs. 65-85% \(\dot{V}O_{2\text{peak}}\)) so this deduction should be treated with caution (Bajpeyi et al. 2009). Because the mechanisms that govern insulin action are multifactorial and complex (Goodyear and Kahn 1998; Hawley 2004), further studies are needed to explain differences in retention of training-induced improvements in insulin action in exercise regimens of different intensities.

In the present study, significant improvements in fasting insulin levels and HOMA-IR scores, but not fasting glucose and HbA1c levels, were observed 16-24 h after the final training session compared with those at baseline in both groups. Previous studies have reported that exercise leads to improvements in metabolic control, as measured by HbA1c levels, blood glucose levels, or insulin sensitivity. However, studies in which these benefits were absent have utilized interventions of low intensity (Wing et al. 1988; Khan and Rupp 1995) or low volume (Brun et al. 2008) or have reported poor adherence to the intervention (Krousel-Wood et al. 2008).

The present study did not reveal any positive exercise-induced effects in the plasma concentrations of TC, HDL-C, LDL-C and TG. Indeed, Kraus et al. found that the benefits of aerobic exercise may not be derived so much from the improvement of lipid and lipoprotein levels measured in the clinical setting but rather from changes in the physical structure of protein particles that carry cholesterol through the bloodstream (Kraus et al. 2002).

The results of this study showed that SBP decreased significantly with training compared with that in the sedentary condition at 16-24 h in both exercise regimens, suggesting that training has the potential to counteract the development of hypertension. However, no significant change in PWV was observed with exercise training in the present study. Ciolac et al. reported that PWV in hypertensive patients decreased because of the reduction of SBP after aerobic interval training, whereas no significant reduction in PWV was observed after low-intensity continuous exercise training (Ciolac et al. 2010). In addition, the effect of exercise training on PWV in the diabetic population is equivocal, and variously reported as being ameliorated (Madden et al. 2009) or unchanged (Loimaala et al. 2009). The different results among the studies may be explained by differences in exercise mode and status of the subjects.

In summary, the present study found persistent training-induced improvements in insulin action 15 days after training cessation in patients with T2DM. This effect may be more dependent on exercise duration than on exercise intensity in regimens involving the same levels of energy expenditure. These data provide useful information for the design of exercise training regimens in sedentary populations at risk for the development of diabetes and cardiovascular disease.

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**Conflict of Interest**

The authors declare that they have no conflict of interest.

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