A physical activity programme and its effects on insulin resistance and oxidative defense in obese male patients with type 2 diabetes mellitus

G Lazarevic¹, S Antic¹, T Cvetkovic², P Vlahovic², I Tasic³, V Stefanovic²

Summary

Aim: The aim of this study was to investigate the effects of regular aerobic exercise on glycaemic control, insulin resistance, cardiovascular risk and oxidative stress-defense parameters in overweight and obese type 2 diabetic patients.

Methods: Changes from baseline to 3 and 6 months of aerobic exercise in total of 30 type 2 diabetes were assessed for physical activity index (PAI), fasting glycaemia (FG), glycated hemoglobin (HbA1c), median blood glucose (MBG), insulin resistance (HOMA), triglycerides (TG), cholesterol, the Ashwell Shape Chart Health Risk, SCORE risk, body mass index (BMI), waist and hip circumference, systolic (SBP) and diastolic (DBP) blood pressure, plasma and erythrocyte malondialdehyde (MDA), glutathione, sulphhydryl groups and catalase (CAT) and were compared to the results of 30 healthy control subjects.

Results: At baseline, significant differences were recorded between the control and diabetes group for FG (P<0.001), HOMA (P<0.001), SBP and DBP (P<0.001), TG (P<0.01), MDA pl (P<0.05), HbA1c (P<0.05), CAT (P<0.01) and SCORE risk (P<0.001). Significant changes within the diabetes group were found for PAI (P<0.05), FG (P<0.001), MBG (P<0.05), HbA1c (P<0.05), HOMA (P<0.01), SBP and DBP (P<0.001) from baseline to 3 months, as well as for FG (P<0.01), HOMA (P<0.001), SBP and DBP (P<0.05) from 3 to 6 months. Significant (P<0.05) correlations were found for FG and PAI (R=0.432), as well as for HOMA and both HbA1c (R=0.412) and SCORE risk (R=-0.387) in the diabetes group.

Conclusion: Regular aerobic exercise has beneficial effects on glycaemic control, insulin resistance, cardiovascular risk, oxidative stress-defense parameters in overweight and obese type 2 diabetics.

Key-words: Diabetes mellitus type 2 · Aerobic exercise · Insulin resistance · Cardiovascular risk · Oxidative stress.

Résumé

Effets d’un programme d’activité physique sur la résistance à l’insuline et les défenses anti-oxydantes chez des hommes obèses atteints de diabète de type 2

Ce travail avait pour objectifs d’examiner les effets de la pratique régulière d’un exercice aérobie sur l’équilibre glycémique, la résistance à l’insuline, le risque cardiovasculaire et les paramètres de défense contre le stress oxydatif chez des obèses atteints de diabète de type 2.

Méthodes: Après 3 à 6 mois d’exercice aérobie chez 30 patients atteints de diabète de type 2, les variations de l’index d’activité physique (IAP), de la glycémie à jeun (GJ), de l’hémoglobine glyquée (HbA1c), de la glycémie moyenne (GSM), de la résistance à l’insuline (RI), des concentrations de triglycérides et de cholestérol, des réponses à un questionnaire d’evaluation des facteurs de risque, de l’indice de masse corporelle (IMC), des circonférences taille et hanches, de la pression artérielle systolique (PAS) et diastolique (PAD), des concentrations plasmatiques (MDA pl) et érythrocytaires en malondialdehyde, du glutathione, des groupes sulphhydryl et de la catalase (CAT) ont été déterminées. Les résultats ont été comparés avec ceux obtenus chez 30 sujets témoins en bonne santé.

Résultats: À l’état basal, des différences significatives ont été constatées entre les témoins et les diabétiques pour les paramètres suivants: GJ (P<0.001), RI (P<0.001), PAS et PAD (P<0.001), concentration de triglycérides (P<0.01), MDA pl (P<0.01), CAT (P<0.01) et score des facteurs de risque (P<0.001). Des variations significatives chez les diabétiques ont été trouvées pour IAP (P<0.05), GJ (P<0.001), GSM (P<0.05), HbA1c (P<0.05), RI (P<0.01), PAS et PAD (P<0.05) entre l’état basal et le 3e mois, GJ (P<0.01), RI (P<0.01), PAS et PAD (P<0.05) entre le 3e et le 6e mois. Des corrélations significatives (P<0.05) ont été observées entre GJ et IPA (r=0.432) ainsi que entre RI et, à la fois, HbA1c (r=0.412) et l’indice de facteur de risque (r=0.387) dans le groupe des diabétiques.

Conclusion: Un exercice aérobie régulier produit des effets bénéfiques sur l’équilibre glycémique, la résistance à l’insuline, le risque cardiovasculaire et les paramètres de défense contre le stress oxydatif chez des obèses atteints de diabète de type 2.

Mots-clés : Diabète sucré de type 2 · Exercice aérobie · Résistance à l’insuline · Risque cardiovasculaire · Stress oxydatif.

Address correspondence and reprint requests to:
V Stefanovic. Institute of Nephrology and Hemodialysis, Faculty of Medicine, Bul. Zorana Djindjica 81, 18000 Nis, Serbia.
stefan@ni.ac.yu

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Introduction

Diabetes mellitus is an increasing problem worldwide and, along with obesity and aging, sedentary lifestyle seems to be one of the key contributing factors [1]. Exercise training has long been known as an important non-pharmacological tool in the treatment of diabetes, but unfortunately, up to one third of adults with diabetes is completely sedentary, and only a third exercises regularly [2].

The importance of tight glycaemic control, as the critical factor in reducing the risk of chronic diabetic complications, has been convincingly demonstrated in the United Kingdom Prospective Diabetes Study (UKPDS) [3]. Regular physical activity has been shown to improve glycaemic control, as measured by glycated haemoglobin and fasting glucose levels [4].

Insulin resistance is an important factor in the pathogenesis of type 2 diabetes mellitus, hypertension, coronary heart disease, dyslipidemia and other metabolic disturbances, also known as metabolic syndrome [5]. Therapeutic reduction of insulin resistance has the potential to improve glycaemic control [6] and has been shown to favorably modify other components of the metabolic syndrome, thereby reducing long-term cardiovascular complications. Aerobic exercise was found to enhance insulin sensitivity acutely [7] and with chronic training [8] in type 2 diabetic patients. High leisure-time physical activity is associated with reduced total and cardiovascular mortality among patients with diabetes or impaired glucose tolerance [9]. Exercise improves cardiovascular risk factors such as hypertension and dyslipidemia [10], as well as enhancing insulin action [11]. Even moderate weight loss in combination with increased activity, which results in preferential loss of fat from central regions, can improve insulin sensitivity and glycaemic control [12].

Oxidative stress is closely associated with aging and is suggested as a contributory factor for initiation and progression of a number of diseases including cardiovascular diseases, impaired glucose tolerance, diabetes and its complications, as well as some other comorbidities [13]. Although acute exhaustive exercise increases oxidative stress, regular exercise training has been shown to upregulate antioxidant protection.

Subjects and methods

Subjects

A total of 30 male, previously sedentary, type 2 diabetic patients, identified from outpatient clinics, aged 54 ± 7.32 years, of the mean BMI 30.8 ± 3.04 kg/m², underwent a regular aerobic exercise for 6 months. Diabetes mellitus type 2 was defined according to the revised National Diabetes Data Group / World Health Organisation criteria and controlled by diet and, if necessary, oral hypoglycaemic agents. Previously prescribed therapy was maintained and participants were instructed not to change their usual diet habits during the study period. The control group consisted of 30 healthy blood bank donors of the same sex, age (48 ± 6.37 years) and BMI (29.6 ± 2.1 kg/m²), with no personal history of, or first degree relatives with diabetes and receiving no medications. Informed written consent was obtained from all subjects before participation.

Procedures

A detailed medical evaluation was performed at baseline, including neurological and ophthalmological examination, paying particular attention to underlying diabetic complications, such as autonomic neuropathy and proliferative retinopathy. An exercise stress test was performed to assess cardiorespiratory fitness and help identify, if present, abnormal heart rate and blood pressure responses, as well as previously undiagnosed ischaemic heart disease. After an overnight fast of 12 hours, urine and blood samples were taken in order to determine glycaemia, glycated haemoglobin, insulin, triglycerides, total, HDL and LDL cholesterol, glutathione, sulphhydryl groups, catalase, plasma and erythrocyte malondialdehyde. Blood pressure, body height and weight, body fat percentage, waist and hip circumference were measured. Body mass index, waist-to-hip ratio, median blood glucose, HOMA and Physical Activity Index were calculated. The absolute 10-year probability of developing a fatal cardiovascular event and the health risk according to the Ashwell Shape Chart were estimated. All outcome measures and procedures carried out at baseline were repeated after 3 and 6 months of regular exercise, with the exception of oxidative stress and oxidative defense parameters, which were not determined after 3 months of physical activity programme.

Intervention

A structured and supervised aerobic exercise programme was conducted following previously published guidelines [14-16] and lasted for 6 months. The programme consisted of at least 3-5 sessions under direct supervision of continuous and moderate aerobic exercise per week, for an average duration of 45-60 minutes and at a workload corresponding to 50-75% of maximal heart rate. Intensity, duration and number of sessions were gradually increased during the follow-up period. Intensity rate was prescribed and monitored on the basis of heart rate. Each session began with a warm-up period, which consisted of 5-10 minutes of mild aerobic activity and 5-10 minutes of stretching, and ended with a cool-down period of 5-10 minutes of less intense activity. The main part of each session consisted of 10-15 minutes of brisk walking at the beginning of the programme, its duration gradually increased for 5-10 minutes each week until the desired duration of 40-50 minutes had been attained.
Outcome measures

Physical activity outcomes

The current physical activity level was assessed using a structured interview from the Framingham Study, following a standard set of questions considering time spent in sleep, rest, occupational and extracurricular activities over a typical 24-hour period and considering the intensity of each of the activities (sleep, sedentary behavior, light, moderate and heavy physical activity). Physical activity index (PAI) was calculated [17] by summing the number of hours spent in each activity intensity level and multiplying by a respective weight factor derived from the estimated oxygen consumption requirement for each intensity level (1.0, 1.1, 1.5, 2.4 and 5.0 respectively). The step counter (HJ-105 Step counter, OMRON) was used to display the number of steps, walking distance and burned calories during one of the usual sessions.

Glycaemic control

Fasting glycaemia was determined directly using an enzymatic method. Glycated hemoglobin (HbA1c) was measured by the immunoinhibition method using latex agglutination inhibition assay and 4 reagents (total hemoglobin R1, HbA1c R2 antibody reagent, HbA1c R3 agglutinin reagent and hemoglobin denaturant) (OLYMPUS AU400e® Chemistry Immuno Analyser). Median blood glucose was calculated as an average value of 5 daily measures of glycaemia.

Insulin resistance

According to the method described by Matthews et al. [18], insulin resistance was estimated by HOMA, which was calculated as [fasting glycaemia (mmol/l) x fasting insulin (mU/l)] / 22.5. Fasting glycaemia was measured directly using an enzymatic method. Fasting insulin was measured using radioimmunoassay (Inep, Zemun).

Cardiovascular risk factors

Total cholesterol (enzymatic color test using cholesterol esterase), triglycerides (enzymatic color test based on the series of coupled enzymatic reactions) and HDL cholesterol (immunoinhibition method, enzymatic color test using antihuman HDL-antibodies) were measured on an OLYMPUS AU400e® Chemistry Immuno Analyser, while LDL cholesterol was calculated using the Friedewald formula [19], except when triglycerides exceeded 3.96 mmol/l (in that case, data were treated as missing). Blood pressure was measured from the left arm in the seated position, after a five minutes rest, three measurements were taken and the average value recorded. BMI was calculated as weight (kg) / height (m)².

Table I

<table>
<thead>
<tr>
<th>Control group (n=30)</th>
<th>Diabetes group (n=30)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>At baseline</td>
</tr>
<tr>
<td>Age (years)</td>
<td>48±6.37</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>92.0±6.89</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.6±2.07</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>103.9±6.24</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>106.3±6.90</td>
</tr>
<tr>
<td>Waist to hip ratio</td>
<td>0.98±0.04</td>
</tr>
<tr>
<td>Physical activity index</td>
<td>34.6±4.37</td>
</tr>
</tbody>
</table>

Values are means ±S.D; ⁸ P<0.05 vs. controls; ⁰ P<0.05 vs. diabetes at baseline; ⁸ P<0.001 vs. diabetes at baseline.

Oxidative stress and oxidative defense parameters

The activity of catalase was determined by the spectrophotometric method, based on the ability of hydrogen peroxide to form a stable stained complex with molybdenum salts [22]. Plasma malondialdehyde was determined by a modified thiobarbituric acid (TBA) method and the products of the reaction were measured at 535 nm after FeSO₄ administration [23]. The determination of erythrocyte malondialdehyde was performed with washed erythrocytes, previously resuspended into 0.1 M phosphate buffer (pH=7.4), kept on ice for 1 hour after 30% trichloroacetic acid administration and centrifuged. Supernatant was transferred into another tube, 1% TBA was added, boiled and
color product read at 532 nm [24]. Reduced glutathione was determined by the modification of the method of Ellman [25], based on the formation of the color product, monitored at 412 nm after Ellman reagent (5,5’-dithiobis-2-nitrobenzoic acid) was added. Sulphydryl groups were determined using Ellman reagent [26], as well.

**Statistical analysis**

Data were analyzed using the statistical software Jandel SigmaStat® for Windows Version 2.0. Student’s t-test and non-parametric tests (Mann-Whitney Rank Sum Test and Kruskal-Wallis ANOVA on Ranks test) were used when appropriate and data were expressed as means ± SD. Parameters were correlated using the simple linear regression test. A P value of less than 0.05 was considered statistically significant.

**Results**

**Baseline characteristics**

The diabetes group consisted of 30 previously sedentary, overweight (n=17) and obese (n=13) type 2 diabetic patients, aged 54±7.32 years, with a mean BMI of 30.8±3.04 kg/m² and previously prescribed diet or oral hypoglycaemic agents therapy. The control group consisted of 30 healthy men of the same age (48±6.37 years) and BMI (29.6±2.07 kg/m²) (table I).

**Physical activity outcomes**

The diabetes and control groups were of the same usual physical activity level, expressed as physical activity index (PAI), at baseline and after 3 months of the study period. However, PAI significantly increased in the diabetes group from baseline to 3 (P<0.05) and 6 months (P<0.001), significantly affecting the between-group difference (P<0.05) at the end of the study period. The average number of 4918.57±686.59 steps, 220.17±31.66 kcal of energy consumption and 3.62±0.30 km of walking distance were measured using the step counter during one of the sessions.

**Glycaemic control**

Fasting glycaemia was significantly over the control level in diabetics during the whole follow-up period (P<0.001), even though it significantly decreased from baseline to 3 (P<0.001) and 6 months (P<0.001), as well as from 3 to 6 months (P<0.01) (table II) and it significantly (R=0.432, P<0.05) correlated with PAI (figure 1). Regular exercise significantly lowered median blood glucose from baseline to 3 (P=0.001) and 6 months (P=0.001) (table II), as well. Just a single bout of aerobic exercise significantly (P<0.001) decreased glycaemia, as measured before and after one of the sessions. Regularly performed exercise significantly decreased HbA1c from baseline to both 3 (P<0.05) and

**Table II**

<table>
<thead>
<tr>
<th>Glycaemic control and insulin resistance.</th>
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<tbody>
<tr>
<td><strong>Control group</strong> (n=30)</td>
</tr>
<tr>
<td><strong>Fasting glycaemia (mmol/l)</strong></td>
</tr>
<tr>
<td><strong>Fasting insulin (mU/l)</strong></td>
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<tr>
<td><strong>MBG (mmol/l)</strong></td>
</tr>
<tr>
<td><strong>HbA1c (%)</strong></td>
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<tr>
<td><strong>HOMA</strong></td>
</tr>
</tbody>
</table>

Values are means ± S.D;<sup>A</sup> P<0.001 vs. controls;<sup>B</sup> P<0.001 vs. diabetes at baseline;<sup>C</sup> P<0.01 vs. diabetes after 3 months;<sup>D</sup> P<0.01 vs. diabetes at baseline;<sup>E</sup> P<0.001 vs. diabetes after 3 months;<sup>F</sup> P<0.01 vs. diabetes at baseline;<sup>G</sup> P<0.05 vs. diabetes at baseline;<sup>H</sup> P<0.05 vs. controls.
6 months (P<0.01), without significant changes from 3 to 6 months (table II).

**Insulin resistance**

In spite of significant decrease of HOMA from baseline to both 3 (P<0.01) and 6 months (P<0.001), as well as from 3 to 6 months (P=0.001) of regular exercise (table II), it was significantly higher in the diabetes group during the whole study period (P<0.001). Within the diabetes group HOMA significantly (P<0.05) correlated with both HbA1c (R=0.412) and SCORE index (R=-0.387).

**Cardiovascular risk factors**

A statistically significant between-group difference was found for triglycerides at baseline (P<0.01). Its within-group change from baseline to 6 months (P=0.05), was significant as well (table III). Even though regular exercise tended to decrease total and LDL-C, as well as to increase HDL-C in the diabetes group, neither between-group nor within-group changes were significant during the whole follow-up period.

Blood pressure was significantly (P<0.001) above the control level in the diabetes group at baseline. Arterial hypertension was found in 28 (93.33%) diabetics at baseline, in 20 (66.67%) after 3 months and only 9 (30%) of them at the end of the study, i.e. blood pressure significantly decreased from baseline to both 3 and 6 months (P<0.001), as well as from 3 to 6 months (P<0.05) (table III) of exercise, contributing to significantly lower both SDP (P<0.01) and DBP (P<0.05) in diabetics at the end of the study period.

Although groups were matched at the beginning of the study, body weight, waist and hip circumference decreased in the diabetes group significantly under the control level (P<0.05) (table III) during the six months of moderate physical activity.

The absolute 10-year probability of developing a fatal cardiovascular event, expressed as SCORE risk, was significantly higher in the diabetes group (P<0.001) during the whole study period (table III), in spite of its significant (P<0.05) within-group decrease from baseline to 6 months of regular exercise. The decrease of the health risk according to the Ashwell Shape Chart within the diabetes group from baseline to the end of an exercise programme was significant (P<0.05), as well.

Body fat percentage was significantly higher in the diabetes group at baseline and after 3 months (P<0.001), as well at the end of the study (P<0.05) in spite of its significant decrease in diabetics from baseline to 6 months (P<0.05) (table III).

**Oxidative stress and oxidative defense parameters**

Glutathione markedly increased within the diabetes group from baseline to 6 months (P<0.001). Plasma MDA significantly decreased (P<0.001), although it was significantly above the control level at baseline (P<0.01), while catalase, although significantly (P<0.01) below the control level at the beginning of the study, significantly increased during the same period of time (P<0.05) (table IV).

**Discussion**

Regular exercise is appointed as an important non-pharmacological tool for the prevention and management of diabetes mellitus, usually demanding direct supervision in previously sedentary, but motivated elderly type 2
diabetics [27]. A significant increase of the current physical activity level, expressed as physical activity index, has been clearly demonstrated in the present study as well, suggesting the importance of the supervised nature of this physical activity programme.

Improvement in glycaemic control was found to be the critical factor in reducing the risk of chronic diabetic complications [28]. A significant decrease in fasting glycaemia, as demonstrated in the present study, confirmed that exercise alone, in the absence of any significant changes in body weight, was able to significantly enhance glucose homeostasis, corresponding to numerous already published results. The difference between pre- and post-exercise glycaemia was significant as well, in accord with the findings of Fritz and Rosenqvist [29]. Since HbA1c reflects mean glycaemia over the preceding 2-3 months [30], its assessment was used in the present study, as well, to determine glycaemic control and demonstrated a significant within-group decrease during the follow-up period, which is consistent with the findings of a meta-analysis [4].

Conflicting results have been published on the validity of HOMA for the assessment of insulin resistance in type 2 diabetics, with both positive [31-33] and negative [34,35] attitudes, since it is thought to mainly reflect hepatic insulin resistance. In the present study, HOMA was significantly over the control level during the whole follow-up period, in spite of its significant decrease in diabetics during the exercise period. Although not significantly, fasting insulin tended to decrease in the diabetes group during the study, suggesting that the decrease of HOMA was not only due to the decrease in fasting glycaemia, but also the decrease in fasting insulin. HOMA significantly correlated with both HbA1c and SCORE index within the diabetes group, as well.

The only significant change in lipid profile recorded in the present study was a significant within-group decrease in triglycerides from baseline to 6 months, although several other studies, combining regular exercise and diet [36] and more rigorous in terms of both volume and intensity [37] than those that have been evaluated in the present study, have reported greater improvements in other lipid profile parameters in type 2 diabetics. In the present study, aimed at regular, but moderate exercise, patients were instructed not to change their usual diet habits during the whole follow-up period, which may explain the disparity in the results.

Hypertension is closely related to being sedentary, but even low-intensity physical effort has been shown to decrease blood pressure, making exercise an especially appropriate intervention for elderly individuals, including those with type 2 diabetes. That has been convincingly demonstrated in the present study as well, since regular exercise significantly decreased blood pressure within the diabetes group, even below the level of healthy controls, probably due to the supervised nature of the intervention and the type of undertaken activity; this is confirmed the findings of a meta-analysis [38].

Insulin resistance and impaired glucose homeostasis are metabolic perturbations that increased abdominal adipose tissue is linked to [39]. The preferential loss of fat from the central regions during regular exercise is closely related to an improvement in insulin sensitivity [40]. A significant between-group difference for BMI has been found at the end of the present study, even in the absence of its significant change within the diabetes group during the whole follow-up period, which is consistent with previous findings that even moderate weight loss in combination with increased activity improves insulin sensitivity and glycaemic control in type 2 diabetics [41]. Even more important was a statistically significant between-group decrease in waist circumference after 6 months, although without significant changes in waist-to-hip ratios, as both waist and hip circumferences tended to decrease similarly.

Although served as a proxy for obesity for many years, BMI was recognized not to differentiate between the over-muscular and the over-fat, nor between individuals with different types of fat distribution. Confirming previously reported results [42], a significant decrease in the health risk according to the Ashwell Shape Chart from baseline until the end of the study was of the utmost importance considering significant improvement in both insulin sensitivity and glycoregulation in type 2 diabetics.

The SCORE risk assessment, aimed at predicting a fatal cardiovascular event over a ten year period, is based on the personal data such as gender, age, smoking, systolic blood pressure and either total cholesterol or the cholesterol/HDL ratio and the threshold for high risk based on fatal cardiovascular events is defined as ≥5%. The SCORE risk significantly decreased from baseline to the end of the study but it

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**Table IV**

Oxidative stress and oxidative defense parameters.

<table>
<thead>
<tr>
<th></th>
<th>Control group (n=30)</th>
<th>Diabetes group (n=30)</th>
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<tbody>
<tr>
<td></td>
<td>At baseline</td>
<td>After 6 months</td>
</tr>
<tr>
<td>Glutathione (µmol/g Hb)</td>
<td>10.6 ± 3.74</td>
<td>9.06 ± 0.74</td>
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<tr>
<td></td>
<td></td>
<td>16.0 ± 5.14</td>
</tr>
<tr>
<td>Catalase (U/l)</td>
<td>102.8 ± 23.4</td>
<td>71.5 ± 26.9</td>
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<tr>
<td></td>
<td></td>
<td>99.5 ± 38.6</td>
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<tr>
<td>Sulphhydril groups (µmol/l)</td>
<td>19.4 ± 12.9</td>
<td>19.9 ± 12.8</td>
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<tr>
<td></td>
<td></td>
<td>20.1 ± 9.03</td>
</tr>
<tr>
<td>Plasma malondialdehyde (µmol/l)</td>
<td>11.6 ± 3.61</td>
<td>16.44 ± 4.73</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.2 ± 3.33</td>
</tr>
<tr>
<td>Erythrocyte malondialdehyde (nmol/ml er)</td>
<td>5.43 ± 0.79</td>
<td>7.46 ± 3.15</td>
</tr>
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<td></td>
<td></td>
<td>8.75 ± 7.01</td>
</tr>
</tbody>
</table>

Values are means ± S.D.  
A P<0.01 vs. controls;  
B P<0.001 vs. diabetes at baseline;  
C P<0.05 vs. diabetes at baseline.
was significantly above the control level in the diabetes group during the whole study period, as expected.

Oxidative stress, the imbalance between reactive oxygen species production and breakdown by endogenous antioxidants, has been implicated in the onset and progression of diabetes-associated cardiovascular disease [43] and its microvascular complications. Hyperglycaemia is meant to be a trigger of endothelial damage underlying these dysfunctions through multiple mechanisms, including increased oxidative stress, expression of atherogenic circulating adhesion molecules and inflammatory cytokines [44]. Exposure of skeletal muscle to an oxidative stress leads to impaired insulin signaling, suggesting endurance exercise training as an additional intervention that is effective in improving skeletal muscle insulin resistance in type 2 diabetics [45]. Although acute exhaustive exercise increases oxidative stress, exercise training has been shown to upregulate antioxidant protection [46], as shown for physically active older adults with reduced exercise-induced oxidative stress compared to their less active counterparts [47]. A significant decrease of endogenous antioxidants, including glutathione [48] and catalase [49], as well as significant increase of malondialdehyde, taken as a marker of oxidative stress [48], are the characteristics of diabetes. As demonstrated in the present study, regular exercise beneficially affected oxidative stress in type 2 diabetics, since it significantly increased plasma glutathione and decreased plasma MDA during the follow-up period.

Conclusions

The most important finding of the present study is that a regular and structured programme of moderate physical activity, maintained properly, is effective in improving glycaemic control, insulin resistance, status of cardiovascular risk factors, oxidative stress and oxidative defense parameters in overweight and obese type 2 diabetics. Group activities under direct supervision are appropriate and beneficial and support the implementation of physical activity into the lifestyle of type 2 diabetes mellitus patients.

References


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