Aerobic Exercise Capacity and Pulmonary Function in Athletes With and Without Type 1 Diabetes

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OBJECTIVE — To compare the aerobic exercise capacity and pulmonary function between athletes with and without type 1 diabetes.

RESEARCH DESIGN AND METHODS — Fifty-one adult age-matched individuals were assessed in random order to the maximum volume of O_2 consumption ($V_{O_{2peak\ max}}$) (ml/kg/min), anaerobic threshold (ml/kg/min), peak pulmonary ventilation (V_E), heart rate (beats per min), time to exhaustion (min), forced vital capacity (FEV) (%), forced expiratory volume in the first second (FEV1) (%), total lung capacity (TLC) (%), and lung diffusion capacity for carbon monoxide (DL_{CO}) (%). Individuals were 27 with type 1 diabetes: 15 athletes (ADM) and 12 nonathletes (NADM); and 24 healthy individuals: 12 ADM and 12 NADM. Duration of diabetes was 14.6 ± 6.2 and 15.2 ± 6.7 years in ADM and NADM, respectively.

RESULTS — $V_{O_{2peak\ max}}$ was higher in ADM than in NADM ($P < 0.001$). The anaerobic threshold was lower in subjects with type 1 diabetes than in control subjects ($P < 0.001$). FEV1 was lower in ADM than in other groups (NADM, athletes control, and nonathletes control, $P < 0.001$).

CONCLUSIONS — Aerobic capacity in subjects with type 1 diabetes with programmed exercise is similar to the capacity of normal athletes despite lower anaerobic threshold and FEV1.

Diabetes Care 33:2555–2557, 2010

Individuals with type 1 diabetes may have some impaired fitness-related components and alterations in their cardiorespiratory responses to exercise. The association of reduced lung function with diabetes has been described for many years (1). The lung may be another organ adversely affected by diabetes. Patients with diabetes demonstrate a restrictive (8–20%) ventilatory defect. However, some of these data are controversial (2–5).

In a previous study, we demonstrated that nonathletic type 1 diabetic patients had decreased lung ventilation associated with decreased maximal O_2 consumption and exercising capacity (6). It is currently unclear, however, whether the observed lower aerobic performance is a function of a reduced level of habitual activity or impairment in cardiorespiratory or skeletal muscle function (7). In this article, we will verify whether our previous findings in lung ventilation, maximum volume of O_2 consumption ($V_{O_{2peak\ max}}$), and exercising capacity are also present in type 1 diabetic patients who engage in regular or systematic exercise.

RESEARCH DESIGN AND METHODS

We studied 51 adult age-matched individuals. The experimental group included 12 athletes (ADM) and 12 nonathletes (NADM) with type 1 diabetes (8). The control group included 12 healthy athletes and 12 healthy nonathletes. The diabetic groups did not have clinical chronic diabetes complications or anemia. The study was approved by the Ethics Committee of the Federal University of São Paulo (number 0604/06). All subjects or respective relatives gave written, informed consent. Anthropometric, laboratory (A1C; high-performance liquid chromatography-normal range: 3.5–6.0%), and microalbuminuria (12-h overnight immunoturbidimetric method; normal up to 20 μg/min) test results were recorded at each visit. All individuals were in a nonfasting state and were in the diabetic group two hours after their last routine insulin injection.

Aerobic exercise capacity was conducted using a motorized Life Fitness treadmill model 9100 HR (Schiller Park, IL). A metabolic system model assembled together with a CO_2/O_2 gas analyzer (Vista Mini CPX; VacuMed Turbofit, Ventura, CA) was used to determine the subject's aerobic power (assessed as peak $V_{E_{max}}$).

The test ended (subject showed signs or symptoms of fatigue) at what we considered the $V_{O_{2peak\ max}}$ (ml/kg/min) of oxygen consumption (aerobic exercise capacity). O_2 consumption, CO_2 production, and pulmonary ventilation (V_E) were monitored at 30-s intervals. The $V_{O_{2peak\ max}}$ was used to define the subject's aerobic capacity. Anaerobic threshold was measured when V_E and V_{CO_2} began to increase nonlinearly as compared with V_{O_2} (9).

The time, flow-volume (lung dynamic and static volumes curves [Collins spirometer DS11a; Warren E. Collins, Braintree, MA]), and lung diffusion capacity for carbon monoxide (DL_{CO}) were obtained following the American Thoracic Society Pulmonary Function Test Guidelines (10). Forced vital capacity (FVC), expiratory volume in the first second (FEV1), functional residual capacity (FRC) (open-circuit method of N_2 washout), and DL_{CO} (corrected for individual hemoglobin concentrations) measurements were reported. Inspiratory volumes were considered acceptable when subjects achieved an inspired volume above 90% of their vital capacity in all tests. All spirometric, N_2 washout, and DL_{CO} data were reported as percentages of the normal predicted values by age, height, and sex.

Table 1—Anthropometrics, clinical characteristics, physical capacity parameters, and lung function in the subjects studied

	ADM	NADM	AC	NAC
n	15	12	12	12
Age (years)	27.4 ± 5.1	26.5 ± 5.6	27.7 ± 4.0	24.1 ± 5.0
Weight (kg)	70.4 ± 9.2	63.1 ± 9.0	68.0 ± 11.3	65.1 ± 11.2
Height (cm)	174.5 ± 8.2*	164.4 ± 5.8*∞	172.6 ± 7.1∞	171.0 ± 7.5
BMI (kg/m²)	23.0 ± 1.7	23.3 ± 2.8	22.6 ± 2.4	22.1 ± 2.6
TDDM (years)	14.6 ± 6.2	15.2 ± 6.7	—	—
A1C (%)	7.5 ± 6.2*†Ω	9.0 ± 1.3*∞×	4.4 ± 0.8†∞	4.4 ± 0.8Ω×
Microalbuminuria (μg/min)	10.46 ± 7.2†Ω	11.69 ± 7.53∞×	2.75 ± 3.01†∞	4.44 ± 3.19Ω×
$V_{O_{2peak\,max}}$ (ml/kg/min)	42.4 ± 5.5*†	34.8 ± 3.3*∞	44.2 ± 5.0∞°	37.8 ± 2.7†°
Anaerobic threshold (ml/kg/min)	31.7 ± 4.4*†Ω	24.6 ± 3.3*∞×	35.9 ± 4.9†∞°	28.3 ± 3.0Ω×°
Peak $V_{E_{max}}$ (btps)	101.8 ± 21.8	91.2 ± 14.3∞	115.8 ± 13.3∞	100.4 ± 8.8
Peak heart rate (beats per min)	194.2 ± 7.1	190.1 ± 6.4∞	198.0 ± 4.9∞°	192.5 ± 6.9°
Time to exhaustion (min)	13.3 ± 1.5Ω	12.4 ± 1.0∞	13.6 ± 1.2∞°	12.1 ± 0.8Ω°
FVC (%)	89 ± 0.74	94 ± 0.86	99 ± 0.98	87 ± 0.24
FEV1 (%)	89 ± 0.51†Ω	96 ± 0.12	102 ± 0.68†	97 ± 0.47Ω
TLC (%)	100 ± 0.77	91 ± 0.78	101 ± 0.14	93 ± 0.99
DL_{CO} (%)	101 ± 0.13	94 ± 0.76	103 ± 0.12	96 ± 0.64

Data are means ± SD. TDDM, time from diabetes diagnosis. Height: *ADM × NADM, ∞NADM × AC. A1C: *ADM × NADM, †ADM × AC, ΩADM × NAC, ∞NADM × AC, ×NADM × NAC. Microalbuminuria: †ADM × AC, ΩADM × NAC, ×NADM × NAC. $V_{O_{2peak\,max}}$: *ADM × NADM, ΩADM × NAC, ∞NADM × AC, °AC × NAC. Anaerobic threshold: *ADM × NADM, †ADM × AC, ∞NADM × AC, °AC × NAC, ΩADM × NAC, ×NADM × NAC. Peak $V_{E_{max}}$: ∞NADM × AC. Peak heart rate: ∞NADM × AC, °AC × NAC. Time to exhaustion: ΩADM × NAC, ∞NADM × AC, °AC × NAC. FEV1: †ADM × AC, ΩADM × NAC. The mean difference is significant at the 0.05 level.

Statistical analysis

Statistical analysis was performed using the SPSS 17.0 (Chicago, IL). The variable curve normality was evaluated using the Kolmogorov-Smirnov test. The correlation (Spearman or Pearson coefficients) between variables was accomplished using the ANOVA test and a Tukey post hoc analysis. $P < 0.05$ was considered significant.

RESULTS — The anthropometric parameters were similar between the type 1 diabetes and control groups. A1C was higher in the NADM than in the ADM ($9.0 ± 1.3$ vs. $7.5 ± 6.2\%$; $P < 0.05$). There were no statistical differences in the microalbuminuria between diabetic groups.

$V_{O_{2peak\,max}}$ was higher in ADM ($42.4 ± 5.5$ ml/kg/min) and athletes control (AC) ($44.2 ± 5.0$) than in NADM ($34.8 ± 3.3$) and nonathletes control (NAC) ($37.8 ± 2.7$) ($P < 0.001$). Anaerobic threshold was lower in ADM ($31.7 ± 4.4$ ml/kg/min) and NADM ($24.6 ± 3.3$) than in AC ($35.9 ± 4.9$) and NAC ($28.3 ± 3.0$) ($P < 0.001$). FEV1 in AC ($102 ± 0.6\%$) and NAC ($97 ± 0.4\%$) was higher than in ADM ($89 ± 0.5\%$) ($P < 0.001$) (Table 1).

CONCLUSIONS — In this study, we have shown that athletes with type 1 diabetes have a $V_{O_{2peakmax}}$ similar to that of athletes without diabetes but a lower anaerobic threshold than that of athletes without diabetes.

In a previous study (6), we demonstrated that nonathletic type 1 diabetic patients have a lower $V_{O_{2peak\,max}}$ than healthy subjects. In the present study, we confirm these data in nonathletic type 1 diabetic patients, but the defect (low $V_{O_{2peak\,max}}$) was not found in athletes with type 1 diabetes. These data are in accordance with a study (11) that compared 128 patients with long-duration type 1 diabetes and 36 healthy individuals.

Metabolic issues (such as hyperglycemia and low lactic acid clearance during and after exercise) and respiratory problems (the ventilation reached at peak exercise was lower in proportion to the reduction in O_2 uptake) may relate to reduced anaerobic threshold and early fatigability or loss of performance (12). However, we did not find a difference in $V_{O_{2peak\,max}}$ between type 1 diabetic and healthy control athletes. Therefore, other factors besides ventilation may influence anaerobic threshold in this diabetic condition.

All of the individuals in this study went to heart rate max frequency during the test. However, the type 1 diabetes sedentary group had lower maximum heart rate than the control group, as expected. This was an interesting finding and one in accordance with our previous data (6) in which the diabetic group showed lower maximum frequency during exercise than normal control subjects. This defect could be corrected with regular exercise since the diabetic athlete was able to achieve the same maximum heart rate as a normal athlete.

In this study, we also found that FEV1 was decreased in type 1 diabetic athletes compared with other groups. This result conflicts with data found in type 1 diabetic nonathletes with the same clinical characteristics and duration of type 1 diabetes but higher A1C than type 1 diabetic athletes (13). However, this can suggest a difficult bronchial adaptation to air flow in the first second of expiration in these individuals. Abnormalities in lung elasticity behavior can be manifestations of widespread elastin and collagen abnormalities in type 1 diabetic patients (14). These alterations have been demonstrated in diabetes and are, in some respects, similar to those that occur during normal aging.

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