A tennis-based health program for middle-aged men who are at risk for heart disease

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Abstract

The purpose of this study was to investigate the health effects of an adapted tennis-based exercise program, in middle-aged men with moderate risk of cardiovascular diseases (CVD). Fifteen men (44.9 ± 4.9 years) with two or more CVD risk factors, completed an eight-week health program that included a continuous tennis-based exercise protocol with constant and controlled intensity between 70%-85% of maximum heart rate and a standard nutritional orientation. Participants were assessed at the beginning and end of the exercise program for standard parameters related to CVD and the Framingham risk score, motor skills and cardiopulmonary fitness, peripheral inflammation (neopterin levels), oxidative stress (nitric oxide and carbonylated protein content), and mitochondrial function (lymphocyte complex I activity). Exercise improved muscle strength and power, speed, agility and flexibility, VO2 max and metabolic equivalents. The participants experienced decreases in body mass index, waist circumference, total and LDL-cholesterol, and reduction in the Framingham Risk Score. Exercise also decreased peripheral oxidative stress and production of nitric oxide. In addition, the increased VO2 at ventilatory threshold 1 correlated with enhanced mitochondrial activity in blood lymphocytes. The tennis-based exercise program adapted to sustaining moderate and constant intensity represents a valuable non-pharmacological intervention to prevent CVD.

Introduction

Cardiovascular diseases (CVD) are a major cause of mortality [1], while physical exercise reduces mortality from all causes [2]. A healthy lifestyle – including regular physical activity, a balanced diet, and weight management – is the best way to prevent and treat CVD [3]. The American College of Sports Medicine (ACSM) recommends that most adults engage in moderate-intensity cardiorespiratory exercise training for ≥ 30 min/day-1 on ≥ 5 days.week-1, or a vigorous-intensity cardiorespiratory exercise training for ≥ 20 min.day-1 on ≥ 3 days.week-1, or a combination of both [4]. Adults should also perform resistance exercises for each of the major muscle groups, and neuromuscular exercises involving balance, agility, and coordination on a 2-3 days.week-1 basis, according to ACSM [4].

According to the ACSM’s recommendations [4], a large number of studies demonstrate significant improvements in CVD risk factors in overweight and/or sedentary individuals [5]. Keep yourself physically active has been shown to be a successful strategy in preventing CVD. Many studies have focused on traditional exercise approaches, i.e., running, walking, resistance training or a combination of those. The effectiveness of more complex models of exercise, such as sports, still lacks evidence. Moreover, many people do not enjoy walking or running, which results in low adherence to physical activity [6]. In addition, it might be necessary to practice more than one of these exercises to improve physical and mental health, and specifically to reduce CVD risk factors including enhanced exercise capacity, muscular strength, and reduced body fat mass [7]. Sports might be a good option to promote good health, since most of them require complex physical functions, such as motor coordination, agility and speed, muscle power and endurance, among others. Outdoor sports are motivating and enjoyable with high adherence levels [6].

Tennis is a sport that improves motivation, strength and muscular endurance, recovering capacity and motor coordination [8]. A tennis match satisfies the ACSM’s recommendations [4] for quantity and quality of exercise involved for the development and maintenance of cardiovascular fitness in healthy adults, independent of the individual’s tennis proficiency [9]. Evidence indicates that regular tennis training decreases cardiovascular risk factors in healthy subjects [9,10]. Middle-aged tennis players displayed an improved VO2 max, decreased body fat mass, and greater grip strength [11]. Thus, tennis sport modality can be suggested as an attractive alternative to health orientated sports programs [9,10,12]. In agreement, a recent published cohort performed in a Scottish and English population showed a robust inverse association between participation in racquet sports and and mortality, with a substantial reductions in all-cause and CVD mortality [13]. Although, there is little evidence of the molecular benefits of these programs for subjects at risk for heart diseases. The purpose of this study was to investigate the effects of an adapted tennis-based exercise programs for subjects at risk for heart diseases.
program for health promotion in middle-aged sedentary men with moderate risk of CVD.

Methods

Fifteen sedentary middle-aged men (44.9 ± 4.9 years), from Florianópolis, Santa Catarina, Brazil participated in the exercise program. The subjects did not meet the minimum needs of physical activity recommended by ACSM [4] in the previous 6 months to the program; thus, they were considered sedentary individuals. The participants presented one to four of the following CVD risk factors: family history of acute myocardial infarction, dyslipidemia, overweight/obesity or hypertension. The baseline anthropometric, motor skills, body composition and anthropometric plasma lipid profile are described in Table 1. Clinical follow-up was performed on a weekly basis by a cardiologist, a psychologist and a nutritionist for the duration of the program. One subject received a beta blocker (propranolol), one subject received an angiotensin II receptor antagonist (losartan), two of them received antidepressants of the serotonin/norepinephrine reuptake inhibitor class (venlafaxine and paroxetine) and one subject received an alpha-adrenergic blocker for urinary retention associated with benign prostatic hyperplasia (doxazosin mesylate). All subjects were informed of the purpose and risks of the study, and provided informed written consent. The study protocol was approved by the local ethics committee (Comitê de Ética em Pesquisa com Seres Humanos - 846/11).

1. The tennis-based physical training protocol employed the following activities at the physiology exercise lab and the tennis court:

The incremental maximal cardiopulmonary test was performed on a horizontal treadmill starting at a speed of 4 km.h⁻¹, increasing at 1 km.h⁻¹.min⁻¹. Gas exchanges were measured using breath-by-breath equipment (Cosmed, Quark CPET, Rome, Italy) and the data was smoothed and averaged at 15-second intervals to measure heart rate (HR), peak oxygen consumption (VO₂ peak), minute ventilation (VE) and respiratory exchange ratio (RER; VCO₂ / VO₂). Ventilatory threshold (VT) 1 and 2 were identified according to Davis et al. [14].

2. The subjects performed motor skills tests and were also evaluated for anthropometric, physiological and cardiovascular risk measures as described below:

The physical training lasted 2 months, and it was performed 3 times.week⁻¹ on alternate days (Mondays, Tuesdays, and Fridays) in an outdoor tennis court (Centro de Ciências do Desporto, Universidade Federal de Santa Catarina, Florianópolis, Brazil). Each 1 h exercise session was divided into 10 min of warm up, 40 min of moderate-intensity training, and 10 min of cool down. Tennis exercise sessions were restlessly continuous in the individual 70-85% maximal heart rate (HR max). The HR was monitored and recorded at 5-second intervals during the exercise (Polar FT1, Kempele, Finland). Some modifications were included to control the exercise intensity (70-85% HR max) during the tennis sessions. In a traditional tennis lesson, the coach feeds a set of balls to one student at a time (Figure 1A, player 1), and the others passively recover at the corner of the court (Figure 1A, player 2-4). In the tennis-based physical training proposed by Garcia et al. [12], subjects are allowed to let the ball bounce twice at the beginning, playing mainly doubles rather than singles games (Figure 1B, players 1-2). In addition, four different on-field movements are performed simultaneously for players 3-6 while they are not involved in the tennis specific activities, in order to maintain the effort within the HR (Figure 1B, players 3-6). We adapted Garcia’s program [12], as shown in Figure 1C, by adding extra motor skill activities which are performed around the court and allow the players to maintain exercise intensity. The activities consisted of running on an agility ladder, skipping drills, varied displacements with cones, and tennis strokes simulation with a medicine ball.

3. An educational nutrition program was conducted to change the subjects’ lifestyle and to control coronary risk factors.

The physiological and cardiovascular risk measures involved rest blood pressure (BP) and HR and they were measured in the morning (8:00 AM) using the automatic Oregon, Scientific BPW120. The anthropometry measures body mass (kg), height (m), waist-to-hip ratio (WHR, cm), and skinfold thickness (mm) were evaluated also for calculation of body fat percentage [15]. The cardiovascular risk measures included body mass index (BMI, kg/m²), WHR, and Framingham risk score. Framingham risk score for men considers age, blood total- and HDL-cholesterol levels, smoking status and rest systolic BP [16].

Motor evaluation was performed in the physiology exercise lab. Lower limb flexibility was assessed with the traditional sit-and-reach test.

Table 1. Fitness measures, body composition and lipid profile at baseline (PRE) and after (POST) the tennis-based program.

<table>
<thead>
<tr>
<th>Fitness measures</th>
<th>PRE</th>
<th>POST</th>
<th>ES</th>
<th>Body and lipid profiles</th>
<th>PRE</th>
<th>POST</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ peak (mL.kg⁻¹.min⁻¹)</td>
<td>36.8 ± 5.8</td>
<td>38.9 ± 6.4*</td>
<td>0.34</td>
<td>Body mass (kg)</td>
<td>95.1 ± 12.0</td>
<td>92.8 ± 11.1*</td>
<td>0.20</td>
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<tr>
<td>MET</td>
<td>10.50 ± 1.65</td>
<td>11.11 ± 1.82*</td>
<td>0.35</td>
<td>Lean body mass (kg)</td>
<td>63.6 ± 6.8</td>
<td>64.8 ± 6.6</td>
<td>0.18</td>
</tr>
<tr>
<td>Rest HR (bpm)</td>
<td>70.7 ± 10.8</td>
<td>68.5 ± 9.3</td>
<td>0.22</td>
<td>Fat body mass (kg)</td>
<td>31.4 ± 7.0</td>
<td>27.9 ± 6.0***</td>
<td>0.54</td>
</tr>
<tr>
<td>HR max (bpm)</td>
<td>173.8 ± 10.3</td>
<td>173.1 ± 10.7</td>
<td>0.07</td>
<td>Body mass index (kg.m⁻²)</td>
<td>31.3 ± 5.2</td>
<td>36.5 ± 4.7*</td>
<td>0.16</td>
</tr>
<tr>
<td>V̇E (L.min⁻¹)</td>
<td>110.3 ± 18.7</td>
<td>115.8 ± 19.0*</td>
<td>0.29</td>
<td>Percentage of body fat (%)</td>
<td>32.8 ± 4.2</td>
<td>29.7 ± 3.8*</td>
<td>0.54</td>
</tr>
<tr>
<td>HR at VT1 (bpm)</td>
<td>114.0 ± 13.6</td>
<td>118.7 ± 14.9</td>
<td>0.33</td>
<td>Waist circumference (cm)</td>
<td>102.5 ± 11.0</td>
<td>99.2 ± 9.5*</td>
<td>0.32</td>
</tr>
<tr>
<td>HR at VT2 (bpm)</td>
<td>151.9 ± 12.1</td>
<td>155.6 ± 12.2*</td>
<td>0.30</td>
<td>Hip circumference (cm)</td>
<td>110.4 ± 7.8</td>
<td>107.8 ± 6.8</td>
<td>0.23</td>
</tr>
<tr>
<td>VO₂ at VT1 (mL.kg⁻¹.min⁻¹)</td>
<td>17.9 ± 4.5</td>
<td>20.4 ± 6.9</td>
<td>0.44</td>
<td>Waist:Hip ratio</td>
<td>0.93 ± 0.05</td>
<td>0.91 ± 0.05</td>
<td>0.40</td>
</tr>
<tr>
<td>VO₂ at VT2 (mL.kg⁻¹.min⁻¹)</td>
<td>31.7 ± 5.9</td>
<td>34.6 ± 6.5*</td>
<td>0.47</td>
<td>Fast glucose</td>
<td>87.3 ± 13.3</td>
<td>88.4 ± 12.2</td>
<td>0.09</td>
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<tr>
<td>Rest systolic BP (mmHg)</td>
<td>132.07 ± 15.8</td>
<td>131.4 ± 14.9</td>
<td>0.04</td>
<td>Triglycerides</td>
<td>118.53 ± 56.3</td>
<td>124.47 ± 62.1</td>
<td>0.10</td>
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<tr>
<td>Rest diastolic BP (mmHg)</td>
<td>90.2 ± 14.0</td>
<td>87.8 ± 14.7</td>
<td>0.17</td>
<td>Total cholesterol</td>
<td>219.8 ± 65.4</td>
<td>185.4 ± 53.8*</td>
<td>0.58</td>
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<tr>
<td>RER (VCO₂ / VO₂)</td>
<td>1.10 ± 0.09</td>
<td>1.10 ± 0.07</td>
<td>0.00</td>
<td>HDL-cholesterol</td>
<td>48.9 ± 9.5</td>
<td>46.7 ± 8.9</td>
<td>0.27</td>
</tr>
<tr>
<td>Sit and reach (cm)</td>
<td>18.0 ± 8.5</td>
<td>22.2 ± 7.4**</td>
<td>0.53</td>
<td>LDL-cholesterol</td>
<td>152.2 ± 64.7</td>
<td>116.9 ± 50.4*</td>
<td>0.61</td>
</tr>
<tr>
<td>Handgrip strength (kg)</td>
<td>24.3 ± 10.6</td>
<td>29.4 ± 8.2**</td>
<td>0.54</td>
<td>MET: metabolic equivalent. HF: heart frequency. VE: minute ventilation. VO₂: oxygen consumption. BP: blood pressure. VT1/2: ventilatory threshold 1/2. RER: resting metabolic rate. ES: effect size. ES: effect size. *P &lt; 0.10, †P &lt; 0.05, ‡P &lt; 0.01.</td>
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</table>
test (cm). Lower body muscle power was assessed with the horizontal jump test (m) and upper body muscle power with the medicine ball (2 kg) throw test (m). Abdominal strength was assessed as a maximum number of repetitions during one min. The shuttle run test evaluated agility (5 × 5 m). The sprint fitness test over 20 meters measured running speed and acceleration. Participants performed each test twice and the highest scores were considered.

A food frequency questionnaire was used to evaluate dietary habits before the health promotion program. Participants received nutritional orientation, according to the food pyramid of Brazilian Society of Functional Nutrition.

For the biochemical measurements 20 mL of blood were collected by venous puncture in 3.2% citrate-conditioned tubes. Ten mL were used for lymphocytes isolation according to Golzari et al. [17] and the final pellet was suspended in 4.4 mM potassium phosphate buffer pH 7.4, containing 0.3 M sucrose, 5 mM MOPS, 1 mM EGTA and 0.1% bovine serum albumin. This preparation was used to measure mitochondrial complex I activity. The other 10 mL of blood were centrifuged at 900 × g for 20 min and the plasma was used for measuring the lipid profile, glucose levels, and the content of neopterin, nitrites and carbonylated proteins.

The lipid profile, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and glucose were determined in the biochemical autoanalyser Dimension RxL (Siemens Healthcare Diagnostics Inc., EUA).

Neopterin concentrations were determined by high-performance liquid chromatography (HPLC) coupled to fluorescence detection as previously reported by our group [18-20], and values were expressed as nmol.L⁻¹. The oxidant status was assessed by measuring the content of carbonylated proteins based on the reaction with dinitrophenylhydrazine, results were expressed as nmol of carbonyl groups per mg protein and calculated by using the extinction coefficient of 22,000 × 10⁶ nmol/mL for aliphatic hydrazones. Nitric oxide formation was estimated spectrophotometrically as the content of nitrites and values were expressed as nmol.mL⁻¹.mg protein⁻¹. Mitochondrial activity was assessed by measuring the activity of complex I based on the rate of NADH-dependent ferricyanide reduction at 420 nm and results were expressed as nmol.min⁻¹.mg
protein. The enzyme activity was measured using a Varian Cary 50 spectrophotometer with temperature control (Varian Inc., Palo Alto, CA, USA). These methodologies were performed as previously reported by our group [18,21].

Data is described as mean ± standard error of the mean (SEM). The normally distributed variables were analysed using the paired Student t test and non-normally distributed variables using the Wilcoxon rank test. Effect sizes (ES) were calculated. ES values > 0.2 indicate a small change; > 0.5 a moderate change; and > 0.8 a large A P value <0.05 was considered to be statistically significant.

Results

The 8-week tennis program improved various fitness measures in the subjects shown in Table 1. The effect size was small (ES=0.2) for \( \dot{V}_\text{O}_2 \) peak [t (13) = 2.62; P<0.05], MET [t (13) = 2.78; P<0.05], VO\(_\text{AT2}\) [t (13) = 2.19; P<0.05], medicine ball throw (t (14) = 2.33; P<0.05) and the 20 m sprint (t (14) = 2.38; P<0.05). However, the effect size was moderate (ES>0.5) for lower limb flexibility [t (13) = 6.25; P<0.001], abdominal strength [t (13) = 4.46; P<0.001], horizontal jump [t (13) = 3.54; P<0.01] and the shuttle run test [z (13) = 3.30; P<0.001]. No significant changes were observed in the rest HR and HRmax, BP, maximum VE, HR, VT1 and VT2, VO\(_2\) at VT1, and RER. Exercise also improved the body composition and the lipid profile of the participants. As shown in Table 1, the effect size of exercise was small (ES>0.2) for body mass [t (14) = 2.81; P<0.05], WHR [t (14) = 3.45; P<0.01] and HDL-cholesterol levels [t (14) = 2.21; P<0.05]; but, it was moderate (ES>0.5) for fat mass [t (14) = 6.40; P<0.001], percentage of body fat [t (14) = 3.28; P<0.01], and the total cholesterol levels [t (14) = 2.18; P<0.05]. Exercise reduced plasma carbonylated protein content [t (9) = 7.23; P<0.001; ES=3.8], improved plasma NO levels [t (9) = 1.98; P<0.05; ES>0.8], and increased mitochondrial activity of complex I in blood lymphocytes [t (9) = 9.19; P = 0.01; ES=7.54] (Figure 2A, B and D; Panel I). The mitochondrial activity of complex I in lymphocytes positively correlated with VO\(_2\) (mL/kg/min) at VT1 [\( r^2 \) = 0.66, P<0.05]. No changes were observed in plasma neopterin levels. Finally, all these changes decreased Framingham risk score [t (14) = 2.25; P<0.05; Figure 2E; Panel II] and the percentage risk of developing coronary heart disease [z (14) = 2.06; P<0.05; Figure 2F; Panel II].

Discussion

The present study proposes a new approach for physical activities linked to health programs that aim to prevent primary CVDs. The exercise protocol applied was based on the modified tennis training method described initially by Garcia et al. [12]. In our adapted protocol, tennis training was performed at a constant intensity between 70% and 85% of HRmax, and various exercises were included around the tennis court in order to maintain the HRmax and to improve the motor skill performance. As a positive outcome of this novel protocol, participants enhanced agility, speed, flexibility, muscle power and abdominal strength that resulted in improved fitness, body composition and lipid profile and decreased plasmatic oxidative stress. Altogether, the

Figure 2. Panel I. Plasma biomarkers of protein oxidative damage (A), nitric oxide (NO, B), inflammation (neopterin levels; C) and lymphocyte mitochondrial activity (D) at baseline (PRE) and after (POST) the tennis-based program. Panel II. Framingham risk score (E) and risk of heart disease (F) at baseline (PRE) and after (POST) the tennis-based program. Results are expressed in mean ± SEM. *P<0.05; ***P<0.001 (paired Student t test).
A tennis-based exercise program adapted to sustaining moderate and constant intensity represents a valuable lifestyle modification to prevent primary CVDs in middle-aged subjects.

- The adapted tennis program improved fitness, body composition, plasma lipid profile, oxidative stress and vascular activity, and reduced the Framingham risk score in middle-aged subjects at risk for heart diseases.

- The tennis-based exercise program improved aerobic (maximal oxygen consumption, VO2 max) and anaerobic (lactate threshold) fitness indexes in middle-aged subjects.

- The tennis training program increased mitochondrial activity in peripheral lymphocytes, which correlated significantly with V̇O2 at VT1 (the onset of plasma lactate accumulation), suggesting this measure could be used as a marker of muscle metabolic capacity or mitochondrial oxidative power.
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Conflict of interest


References