EFFECTS OF A SINGLE BOUT OF EXERCISE AND EXERCISE TRAINING ON STEROID LEVELS IN MIDDLE-AGED TYPE 2 DIABETIC MEN: RELATIONSHIP TO ABDOMINAL ADIPOSE TISSUE DISTRIBUTION AND METABOLIC STATUS

P. BOUDOU, E. DE KERVILER, P. VEXIAU, J. FIET, G. CATHELINEAU, J.F. GAUTIER

SUMMARY - Lower androgen levels have been suggested to be associated with type 2 diabetes and central obesity and are probably involved into the development of atherosclerosis. The present study investigates the effect of acute and chronic exercise on Dehydroepiandrosterone (DHEA) levels in relation to abdominal fat distribution and metabolic status in type 2 diabetes. Twenty weight-stable, middle-aged males with type 2 diabetes were enrolled in the study and participated in a submaximal (VO2 peak) and moderate (50% VO2 peak) exercise bout. The subjects were randomly assigned either to a trained or a control group, respectively. Physical training consisted of an 8 week program of aerobic exercise (75% VO2 peak, 45 min), twice a week and intermittent exercise, once a week, on a bicycle ergometer. Acute exercise significantly increased DHEA and Testosterone (T) levels. Physical training increased VO2 peak (42%, p < 0.001), insulin sensitivity index (KITT) (57.5%, p < 0.02), and basal DHEA levels (38%, p < 0.05), and decreased HbA1c (29%, p < 0.001), visceral adipose tissue (VAT) (44%, p < 0.01) and subcutaneous adipose tissue (SAT) levels (18%, p < 0.01). Body weight, BMI and insulin, T levels were not modified. Changes in DHEA levels were not correlated with changes in insulin sensitivity and abdominal fat distribution. In conclusion, exercise training favourably affects DHEA levels independently of improvements of metabolic status and abdominal fat distribution in patients with type 2 diabetes.

Key-words: type 2 diabetes, exercise training, dehydroepiandrosterone, adiposity.

ORIGINAL ARTICLE

Diabetes & Metabolism (Paris) 2000, 26, 450-457

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Prospective studies have noted that low levels of dehydroepiandrosterone sulfate (DHEAS) and DHEA were associated with high prevalence for cardiovascular heart disease (CHD) in men over age 50 [1-2]. In addition, dehydroepiandrosterone (DHEA) has been proposed as a “missing link” between atherosclerosis and hyperinsulinemia [3]. Lower endogenous androgen levels have been reported in men with type 2 diabetes and have been shown to be associated with obesity and central adiposity [4-10]. Furthermore, 5α-androstane-3α, 17β diol glucuronide, an end product of the androgen metabolism, has been suggested as a steroid correlate of visceral adiposity in men [11]. Decreases in sex-steroid levels and visceral fat accumulation contribute to the increased cardiovascular risk particularly in men with type 2 diabetes which are characterized by a high risk of morbidity and mortality from cardiovascular and ischemic heart disease [12-14]. In parallel, physical activity has been recognized to improve body fat distribution in type 2 diabetic patients [15-17]. Furthermore, clinical and epidemiological studies provide evidence that physical activity improves androgenic status and the prevalence of coronary heart disease in the entire sample population population [18-19]. In the present study, we propose to evaluate the effects of acute (moderate and submaximal) exercise and/or physical exercise training on androgen levels in middle-aged men with type 2 diabetes. We further investigate if potential changes in androgen levels are related to the exercise training program per se, or to improvements in body fat distribution and insulin-glucose homeostasis following the exercise training program.

## MATERIALS AND METHODS

**Subjects and study design**

Twenty Caucasian male patients with type 2 diabetes mellitus, aged 46.8 ± 7.7 years (m ± SD), with moderate glucose control (HbA1c: 8.0 ± 1.7%), and stable body weight (1.27 ± 1.20 kg (m ± SD), 95% CI 0.70 ± 1.83 for at least 4 months prior to the study) were recruited from a population attending our department for diabetes care. All subjects underwent a medical examination, including medical history, physical examination and had a negative electrocardiogram at rest and during a maximal exercise test. None had a previous history or evidence of diabetic micro- or macroangiopathy, renal, liver or heart disease. The known duration of diabetes for each subject did not exceed 120 months. All were normotensive and non-smokers. All were previously sedentary subjects (no participation in regular moderate and or intensive exercise for at least 6 months before the study). The level of physical activity was assessed before and after the study using Baecke’s questionnaire [20]. All but seven were treated with hypoglycemic agents (metformin alone (n = 6) or metformin and glibenclamide (n = 7)). Antidiabetic medication, if any was continued during the program. This investigation was approved by the local ethics committee, in accordance with the guidelines published in the Declaration of Helsinki, and informed consent was obtained from all patients.

**Anthropometric measurements and magnetic resonance imaging**

Weight, height, waist and hip circumferences were determined for each subject using conventional procedures [21], and body mass index (weight/height²: kg/m²) and waist to hip circumference ratio calculated. Truncal (subcapular and suprailiac sites) and peripheral (biceps and tricep regions) skinfold thicknesses were measured using a Harpenden skinfold caliper (Serita, East Rutherford, NJ) [22]. The means of two repeated measurements at each skinfold site by the same experienced investigator were used. The overall mean values were averaged and used to estimate % body fat. Subcutaneous and visceral fat depots were quantified by magnetic resonance imaging (MRMax, General Electric, Milwaukee, WI), as previously described [23]. Briefly, a series of three 7 mm-thick axial images with a 3 mm-gap were obtained at the level of the umbilicus. Two large regions of interest, each corresponding to the largest cross-sectional area of subcutaneous and visceral adipose tissue were drawn manually on each slice. Areas of the respective adipose tissue (AT) region defined in each slice were computed automatically by summing up AT pixel and multiplying by the pixel surface area. A reference tube allows one to determine the threshold of pixels corresponding to AT.

**Insulin sensitivity**

After an overnight fast, an intravenous insulin tolerance test (ITT) was performed. Subjects discontinued hypoglycemic medication, if any, either on the morning (Glibenclamide) or 48-h before (Metformin) the beginning of the test which consisted of a bolus of regular insulin (0.1 U/kg body weight). Briefly, plasma glucose concentrations were measured before and every 3 min for 15 min after the insulin injection, in order to calculate the constant rate of plasma glucose disappearance [24]. Fasting insulin levels and levels of steroids were measured prior to the ITT.

**Exercise evaluation**

**Single bouts of exercise**

Forty minutes after a standard breakfast (carbohydrate (40 g), lipid (15 g), protein (12 g), subjects...
exercised between 09.00h and 09.30h on a bicycle ergometer (Monark-Crescent, Valberg, Sweden) according to an incremental protocol used for VO$_2$ max determination. This test was performed in non fasting state, as usual, in order to obtain the best performance of the subjects [25]. Exercise was performed at a constant rate of 60 rpm throughout the 30 min duration of the session. The test initially set at 30 w for a 2-min warm-up period was followed up by a 30 w workload increase every 2-min until the subject could no longer continue, and was adjusted to attain maximal effort for 15-20 min. VO$_2$$_{2max}$ was achieved when VO$_2$ demonstrated a plateau during an increase in power output and/or the respiratory exchange ratio was higher than 1.1, the blood lactate concentration was higher than 8 mmol/L, and the subject exhibited motor deficiencies and visual tiredness [25], because VO$_2$ did not reach a plateau in most patients, the highest VO$_2$ values obtained during incremental exercise corresponded to a VO$_2$ peak rather than VO$_2$$_{2max}$. Measurements of $O_2$ consumption, CO$_2$ production (Sensor Medics Corporation, Yorba Linda, CA), heart rate, minute ventilation, respiratory exchange ratio and power (watt) were recorded [16]. Two days after the VO$_2$ peak determination (submaximal exercise), a 30-min. moderate exercise bout was performed between 08.00 and 08.30h after an overnight fast at 50% of the previously determined VO$_2$ peak.

**Exercise training session**

Subjects were randomly divided into two equal groups. The trained group was first enrolled in a thrice-a-week graded program (pre-exercise training) on a bicycle ergometer. This program was maintained for about 2 weeks until each subject was able to work for 45 min at 75% of their previously determined VO$_2$ peak. Then, the trained group was assigned to a 8-week training program (3 times/week) consisting of two different kinds of exercise: firstly, a continuous exercise bout performed twice a week for 45 min at 75% of their VO$_2$ peak; secondly an intermittent exercise consisting of five 2 min exercise bouts at 85% VO$_2$ peak separated by 3 min exercise at 50% VO$_2$ peak performed once a week [16]. All exercises were performed on an ergocycle and supervised in our department. Subjects from the control group were seen weekly to exercise on the bicycle ergometer at a constant rate of 60 rpm for 20 min at low intensity (30 W). Medication levels were not modified during the study, and subjects were asked to maintain their usual diet. At the end of the exercise training session, all subjects repeated the entire investigation exactly as upon entry into the study. The insulin tolerance test was performed 3-5 days after the last exercise session to make sure that effects of a single bout of exercise on insulin sensitivity had subsided [26]. Among the 20 patients enrolled into the baseline investigation (including submaximal and moderate exercises), four (two of the trained, and two of the control group) dropped out for personal constrains at different moment of the training period and did not have the follow-up investigation.

**Endocrine investigation**

In each patient one blood sample (=10 ml) was collected from an antecubital vein before and immediately after the VO$_2$ peak and 50% VO$_2$ peak determination while patients remain seated on their ergocycle. For a given parameter, samples from each patient were processed in the same assay run.

Glucose levels were measured using the glucose oxidase method. Insulin levels were measured by RIA, using the insulin INSIK-5 kit (Sorin Biomedica, Antony, France). Serum levels of dehydroepiandrosterone sulfate (DHEAS), cortisol (F), and sex-hormone binding globulin (SHBG) were measured by RIA using the $^{[125]}$DHEAS kit (Immunotech, Marseille, France), the Gamma Coat $^{[125]}$IF (Incstar Corp., Stillwater, MN) and $^{[125]}$SBP Coat RIA (bioMérieux, Lyon, France), respectively. The intra- and interassay coefficients of variation were from 3.2-7.4%, 6.6-7.7%, 2.5-5.2% and 3.4-10.6%, 8.8-9.8%, 4.1-5.5% for DHEAS, F and SHBG assays, respectively. Circulating testosterone (T), DHEA, 5α-androstan-3α, 17β-diol glucuronide (3α-AdiolG) levels were determined. Briefly, glucuronon conjugated steroids were previously hydrolyzed to unconjugated steroids by incubating samples, adjusted to pH 6.4 with 1M phosphate buffer, for 24h at 45°C with highly purified β-glucuronidase (10.000 Fishman units). Then, samples were mixed with tracer doses of tritiated steroids (3.000 cpm, each) to monitor losses occurring during the extraction, chromatography and redissolution steps. After 30 min-incubation with intermittent shaking at room temperature, samples were extracted with cyclohexane: ethyl acetate (1: 1, v: v). The upper organic layer containing steroids was evaporated and the dried extract dissolve in isooctane was used for separation by cellulose partition column chromatography. Steroids were eluted by sequential addition of set volumes of solvent (isooctane: dichloromethane, v: v) mixtures of increasing polarity as follows: 95: 5 for DHEA, 91: 9 for T, 64: 36 for 3αAdiol and 70: 30 for 17OHpreg. Each specific steroid fraction was collected evaporated, redissolved in phosphate gelatine buffer and assayed by RIA methods. Percent recoveries ranged from 85.25 - 91.10, 77.15 - 81.05, 81.00 - 85.00 and 74.85 - 78.30 for DHEA, T, 3α-AdiolG and 17OHpreg, respectively. Intra- and inter-assay coefficients of variations were from 4.3-7.2% (T, 17OHpreg, DHEA), 4.3-8.5% (3α-AdiolG) and 4.4-9.6% (T, 17OHpreg, DHEA), 7.2-12.0% (3α-AdiolG), respectively [27-28].
Statistical analyses

Changes in steroid levels following moderate and submaximal exercise bouts, and differences in steroid responses (expressed for each subject as (t_after/t_before exercise) between moderate and submaximal exercise bouts were assessed using the non-parametric Wilcoxon matched pairs signed rank test. Before training, comparisons of the levels of the overall descriptive variables between the trained and the control group were performed using the Mann-Whitney U test. The effect of exercise training was evaluated by comparison of changes (expressed for each subject as t_after/t_before exercise) in exercise capacity and metabolic and hormonal parameters between the trained and the control group, using the Mann-Whitney U test. The effect of training on steroid response to acute exercise (moderate or submaximal) was analyzed using the Mann-Whitney U test on (t_after/t_before exercise) after training - (t_after/t_before exercise) before training, determined for each subject. A two-tailed p value < 0.05 was considered significant. Spearman rank correlation coefficients were computed to assess the association between sex-steroid levels and anthropometric characteristics, VO2 peak and KITT measurements (baseline values, whole group). The same test was applied to evaluate the association between changes in each of these parameters in the trained group. Values are expressed as mean ± SD otherwise stated.

RESULTS

Descriptive data

Age, duration of diabetes, anthropometric, metabolic and physical characteristics of the entire population (n = 20) are summarized in Table I. Subjects on oral hypoglycemic agents (i.e. metformin alone or in association with glibenclamide) had similar mean KITT values compared with those not on medication (2.40 ± 0.80 vs 2.25 ± 0.75 vs 1.90 ± 0.60% min⁻¹, p > 0.3).

Correlation between endocrine and anthropometric data

Basal testosterone levels were negatively correlated with fasting insulin levels (r = -0.51, p < 0.04) and a trend was observed between levels of 3α-AdiolG and WHR (r = -0.39, p: 0.09). In addition, T levels and fasting insulin levels were not related to anthropometric variables.

Effect of acute exercises

Acute (intensive and moderate) exercise increased serum levels of T and DHEA (Table II). No significant variations in DHEAS and 3α-AdiolG levels were noted and changes in DHEA level were greater during moderate compared to submaximal exercise (Table II).

Effect of exercise training

No significant differences were observed in patient variable values before training except for HbA1c and DHEAS values (p < 0.05) between the trained and the control group (Table III). The training program increased VO2 peak (42%, p < 0.001), maximal workload levels (41%, p < 0.001), KITT (57.5%, p < 0.02), 17OHPreg (37.4%, p < 0.02), DHEA (36%, p < 0.05) and F levels (15.3%, p < 0.01) compared with the control group (Table III). These changes were not modified after adjustment for age, duration of diabetes, and anthropometric characteristics. Furthermore, exercise training induced decreases in HbA1c (29%, p < 0.001), visceral (44%, p < 0.001) and subcutaneous (18%, p < 0.001) adipose tissues in the trained compared with the control group. In contrast, no significant changes were noted in body weight, BMI,

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**Table I.** Age, duration of diabetes, anthropometric, and physical characteristics (mean ± SD, and range) of the entire sample population (n = 20).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>46.80 ± 7.70</td>
<td>31.00 – 59.00</td>
</tr>
<tr>
<td>Duration of diabetes (months)</td>
<td>62.70 ± 47.50</td>
<td>12.00 – 144.00</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>85.30 ± 14.10</td>
<td>55.00 – 108.00</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>28.10 ± 4.50</td>
<td>20.20 – 36.95</td>
</tr>
<tr>
<td>Waist to hip ratio</td>
<td>0.95 ± 0.06</td>
<td>0.83 – 1.02</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>97.80 ± 12.10</td>
<td>76.00 – 119.50</td>
</tr>
<tr>
<td>Subcutaneous adipose tissue (cm²)</td>
<td>239.00 ± 61.50</td>
<td>142.20 – 380.10</td>
</tr>
<tr>
<td>Visceral adipose tissue (cm²)</td>
<td>175.00 ± 60.70</td>
<td>93.00 – 347.70</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>25.80 ± 3.00</td>
<td>18.60 – 30.40</td>
</tr>
<tr>
<td>Workload (watt)</td>
<td>77.90 ± 10.50</td>
<td>60.00 – 90.00</td>
</tr>
<tr>
<td>VO2 peak (ml/kg⁻¹.min⁻¹)</td>
<td>22.70 ± 3.50</td>
<td>17.70 – 29.70</td>
</tr>
<tr>
<td>Glycemia (mmol/L)</td>
<td>8.85 ± 1.60</td>
<td>5.40 – 12.50</td>
</tr>
<tr>
<td>HbA1c %</td>
<td>8.00 ± 1.70</td>
<td>5.10 – 11.30</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>19.40 ± 7.10</td>
<td>8.40 – 34.10</td>
</tr>
<tr>
<td>KITT (%min⁻¹)</td>
<td>2.10 ± 0.90</td>
<td>0.61 – 3.48</td>
</tr>
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WHR, fasting insulin, glycemia, SHBG, T, 3α-AdiolG, DHEAS levels compared with the control group (Table III). Finally, exercise training significantly reduced the response of DHEA (20 ± 14 vs. 5 ± 27%, p < 0.03) to submaximal exercise in the trained compared with the control group. Changes in basal DHEA levels were not related to changes in glycated hemoglobin (HbA1c), changes in KFFT, changes in visceral adipose tissue and changes in DHEAS. In contrast, changes in DHEA levels were positively and significantly correlated with changes in 17OHPreg (r = 0.88, p < 0.02). Furthermore, changes in KFFT were negatively correlated with changes in visceral adipose tissue (r = −0.88, p < 0.02), and changes in HbA1c tended to be positively correlated with changes in VO2 peak (r = 0.67, p: 0.07) in the trained group.

**DISCUSSION**

The main findings of our study suggest that in men with type 2 diabetes:

1) fasting DHEAS and DHEA concentrations do not correlate with body weight, abdominal fat distribution and insulin levels and insulin sensitivity;
2) both moderate and submaximal exercises increase DHEA concentrations;
3) exercise training increases fasting DHEA concentrations.

The association between sex-steroids and obesity, body fat distribution and insulin levels in men has been previously investigated [7-10]. In non diabetic subjects neither DHEA nor DHEAS levels have been shown to be associated with anthropometric variables or insulin levels or insulin sensitivity [29-31], except in the study of Haffner et al. [32] for DHEAS and insulin levels. Accordingly, we did not find any relationship between these variables in our group of type 2 diabetic men. Fasting testosterone levels were negatively correlated with fasting insulin levels as it has been previously reported in non diabetic subjects [33]. In contrast with Tchernof et al. [11], we did not find any positive correlation between VAT areas and serum levels of 3α-AdiolG whether analysed in the entire sample population or when subjects were assigned to BMI ≤25 kg/m² vs BMI > 25 kg/m². In addition the association between 3α-AdiolG levels and WHR tended to be negative. The reasons which could account for the differences among the two studies are not readily explained. Our data concerning serum 3α-AdiolG in men [34] are very similar to those found by other groups [35-38]. The method (MRI) we used to estimate the abdominal fat distribution was closely related to computed axial tomography. Although abdominal fat distribution was determined at only one level by our MRI protocol, Ross et al. [39] showed that the results obtained by this method correlated well with total abdominal fat. The participants of the two studies were caucasians and non smokers. Although, subjects of the two studies have very similar degree of obesity, our patients were slightly older, diabetics and dietary intake could be different. In contrast, the lower levels of the main 3α-AdiolG steroid precursors (e.g. DHEA, androstenedione, and T) in Tchernoff’s obese subjects did not favor elevated levels of 3α-AdiolG levels as noted by this group [11]. Finally, the absence of gradient of conjugated steroids between adipose tissue and systemic circulation tended to demonstrate that there is no accumulation of conjugated steroids in adipose tissue [40].

As noted in normal men [41-42], our findings show that acute exercise increases T and DHEA levels in type 2 diabetic subjects. In contrast, DHEAS and 3α-AdiolG levels were not modified which is consistent with their much longer half-lives compared to those of T and DHEA [43-45]. Changes in DHEA levels are greater during moderate compared with submaximal exercise. However, the submaximal exercise was performed in the postprandial state, as usually recommended [25]. The postprandial submaximal exercise could induce a relative hyperinsulinemia and higher levels of glucose compared to the fasting moderate exercise. Since hyperinsulinemia and hyperglycemia have been reported to decrease DHEA levels in healthy [46] and type 2 diabetic men [5], it could be

<table>
<thead>
<tr>
<th>Steroids</th>
<th>Moderate exercise before</th>
<th>after</th>
<th>Submaximal exercise before</th>
<th>after</th>
</tr>
</thead>
<tbody>
<tr>
<td>T (nmol/L)</td>
<td>16.26 ± 6.26</td>
<td>20.21 ± 6.52^b</td>
<td>14.17 ± 4.86</td>
<td>16.80 ± 5.34^c</td>
</tr>
<tr>
<td>DHEA (nmol/L)</td>
<td>9.15 ± 4.30</td>
<td>20.01 ± 10.80^a</td>
<td>10.50 ± 4.90</td>
<td>15.90 ± 7.50^a</td>
</tr>
<tr>
<td>DHEAS (µmol/L)</td>
<td>4.73 ± 2.63</td>
<td>4.92 ± 2.58</td>
<td>4.72 ± 2.33</td>
<td>5.01 ± 2.68</td>
</tr>
<tr>
<td>3α-AdiolG (nmol/L)</td>
<td>21.90 ± 8.90</td>
<td>22.70 ± 8.50</td>
<td>20.80 ± 7.50</td>
<td>21.70 ± 8.15</td>
</tr>
</tbody>
</table>

Significant changes following each exercise were as follows:^a p ≤ 0.0006, ^b p ≤ 0.002, ^c p ≤ 0.01.
assumed that the post-prandial state had blunted the submaximal exercise-induced DHEA increment, explaining why DHEA changes were lower during submaximal compared to moderate exercise. This could be directly addressed by investigating both exercises in the same post-prandial state with plasma insulin and glucose measurements.

Exercise training increases basal DHEA, 17OHpreg and F levels and does not modify basal T, DHEAS and 3αAdiolG levels. Some studies have already shown that exercise training does not modify [47-48] or significantly increase [18] T and DHEAS levels in non diabetic men. The differences in steroid responses to chronic exercise among non diabetic studies and our study could be related to the heterogeneity of the population studied (age, degree of physical condition), the exercise program (type, frequency, intensity, duration), the time of blood assessment (at the end of the session, few days or weeks after completing the exercise program). The mechanism by which exercise training favourably affects basal DHEA levels is unknown. Although our study involved small groups of subjects, HbA1c and insulin did not seem to have any direct influence on DHEA changes following exercise training in the trained group. Indeed, changes in HbA1c levels were not related to changes in DHEA levels, and exercise training has no effect on fasting insulin levels. Increases in 17OHpreg and F levels with no modifications in T and DHEAS levels following training suggest that changes

<table>
<thead>
<tr>
<th>Variables</th>
<th>trained group before</th>
<th>trained group after</th>
<th>untrained group before</th>
<th>untrained group after</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months)</td>
<td>42.90 ± 5.20</td>
<td>47.90 ± 8.35</td>
<td>88.90 ± 13.40</td>
<td>90.40 ± 11.50</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>86.90 ± 13.40</td>
<td>27.80 ± 4.30</td>
<td>30.85 ± 5.20</td>
<td>30.90 ± 5.00</td>
</tr>
<tr>
<td>WHR</td>
<td>0.94 ± 0.05</td>
<td>0.93 ± 0.06</td>
<td>0.97 ± 0.05</td>
<td>0.97 ± 0.05</td>
</tr>
<tr>
<td>VO2 peak (ml.kg⁻¹.min⁻¹)</td>
<td>23.45 ± 3.60</td>
<td>32.85 ± 4.00a</td>
<td>21.95 ± 3.55</td>
<td>22.20 ± 4.05</td>
</tr>
<tr>
<td>Maximal workload (Watt)</td>
<td>78.43 ± 10.25</td>
<td>110.93 ± 23.29a</td>
<td>74.68 ± 10.89</td>
<td>74.68 ± 12.27</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>21.30 ± 7.25</td>
<td>22.35 ± 8.20</td>
<td>21.60 ± 2.15</td>
<td>24.30 ± 14.00</td>
</tr>
<tr>
<td>Glycemia (mmol/L)</td>
<td>9.35 ± 1.20</td>
<td>9.70 ± 1.65</td>
<td>8.50 ± 2.15</td>
<td>8.55 ± 1.95</td>
</tr>
<tr>
<td>HbA1C (%)</td>
<td>9.00 ± 1.70*</td>
<td>6.25 ± 0.50a</td>
<td>7.30 ± 1.30</td>
<td>7.60 ± 1.40</td>
</tr>
<tr>
<td>SAT (cm²)</td>
<td>241.55 ± 49.55</td>
<td>198.00 ± 39.00a</td>
<td>262.50 ± 69.10</td>
<td>260.00 ± 70.40</td>
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<tr>
<td>SHBG (nmol/L)</td>
<td>19.65 ± 10.20</td>
<td>18.70 ± 9.50</td>
<td>15.70 ± 5.65</td>
<td>16.25 ± 6.55</td>
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<tr>
<td>17OHPreg (nmol/L)</td>
<td>4.60 ± 2.75</td>
<td>6.10 ± 3.25b</td>
<td>5.40 ± 3.85</td>
<td>4.45 ± 2.40</td>
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<tr>
<td>DHEA (nmol/L)</td>
<td>11.00 ± 3.10</td>
<td>14.25 ± 4.10c</td>
<td>11.35 ± 6.05</td>
<td>10.75 ± 7.30</td>
</tr>
<tr>
<td>F (nmol/L)</td>
<td>304.10 ± 103.40</td>
<td>360.10 ± 156.45b</td>
<td>336.50 ± 74.95</td>
<td>286.05 ± 65.20</td>
</tr>
<tr>
<td>T (nmol/L)</td>
<td>15.50 ± 5.90</td>
<td>16.85 ± 6.70</td>
<td>13.75 ± 4.95</td>
<td>17.20 ± 5.00</td>
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<tr>
<td>3αAdiolG (nmol/L)</td>
<td>23.80 ± 8.00</td>
<td>19.70 ± 6.30</td>
<td>18.50 ± 7.40</td>
<td>18.30 ± 7.30</td>
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<tr>
<td>DHEAS (µmol/L)</td>
<td>6.05 ± 2.70*</td>
<td>6.15 ± 3.10</td>
<td>3.30 ± 1.90</td>
<td>3.30 ± 2.10</td>
</tr>
</tbody>
</table>

Data comparing differences between changes expressed as t after /t before Exercise in the two groups are evaluated as noted in materials and methods section and are reported for each variable in the column “after training session” of the trained group as follows: a p < 0.001, b p < 0.02, c p < 0.05. Significant differences before training between the trained and untrained group were noted in the column “before training session” of the trained group (*p < 0.05).
crease in the cardiovascular risk remains to be estab-
lished. Type 2 diabetic male subjects contribute to a de-
crease in the activation of the sympatho-adrenal
system induced by training [49].

Improvement of DHEA levels could be of clinical
importance in the diabetic population. Indeed, it has
been shown that the prevalence of cardiovascular dis-
ease is inversely related to DHEA and DHEAS levels
[1-2]. Furthermore, a beneficial effect of DHEA on
insulin resistance has been suggested [3].

CONCLUSION

Our results show that acute exercise and exercise
training improve DHEA levels in men with type 2
diabetes independently of changes in metabolic and
anthropometric variables. Whether changes in DHEA
levels induced by acute exercise and exercise training
in type 2 diabetic male subjects contribute to a de-
crease in the cardiovascular risk remains to be estab-
lished.

Acknowledgements – This work was supported by: La Caisse
Nationale de Prévoyance, La Caisse Nationale d’Assurance
Maladie des Professions Indépendances, La Caisse Maladie
Régionale des Professions Industrielles et Commerciales d’Il
de France, and l’Assistance Publique des Hôpitaux de Paris.

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