Effects of Nandrolone Decanoate on $\dot{V}O_{2\text{max}}$, Running Economy, and Endurance in Rats

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ABSTRACT

GEORGIEVA, K. N., and N. P. BOYADJIEV. Effects of Nandrolone Decanoate on $V\dot{O}_{2\text{max}}$, Running Economy, and Endurance in Rats. Med. Sci. Sports Exerc., Vol. 36, No. 8, pp. 1336–1341, 2004. Purpose: The aim of the present study was to determine the effects of treatment with an anabolic androgenic steroid (AAS), nandrolone decanoate, on the submaximal running endurance (SRE), maximum oxygen consumption ($V\dot{O}_{2\text{max}}$), running economy ($V\dot{O}_{2\text{submax}}$), and blood oxygen carrying capacity of endurance trained rats.

Methods: Forty male Wistar rats were randomly allocated into two groups: a sedentary group and an exercising group training on treadmill for 8 wk. Half of the trained and half of the sedentary rats received weekly either nandrolone decanoate (10 mg·kg$^{-1}$) or placebo (Pl) for the last 6 wk of experiment. SRE and $V\dot{O}_{2\text{max}}$ tests were performed several times for all four groups ($N = 10$ each). Red blood cells parameters were measured at the end of the experiment. Results: The trained rats had increased their SRE compared with sedentary rats throughout the experiment. At the end of the trial, the trained rats receiving nandrolone decanoate ran 46% longer than trained rats receiving Pl during the SRE test ($P < 0.05$). At the end of the experiment, trained rats had greater maximal time to exhaustion and higher $V\dot{O}_{2\text{max}}$ than those of the sedentary rats but there were no differences in $V\dot{O}_{2\text{max}}$, $V\dot{O}_{2\text{submax}}$, and red blood cells parameters between the trained rats receiving nandrolone decanoate and those receiving Pl. Conclusions: Nandrolone decanoate has no effect on the SRE, $V\dot{O}_{2\text{max}}$, and $V\dot{O}_{2\text{submax}}$ of untrained rats. AAS treatment combined with submaximal training enhances SRE more than training alone but exerts no additive effects on $V\dot{O}_{2\text{max}}$, running economy, and oxygen carrying capacity of blood. The results suggest that this improvement in SRE of trained rats is due to the impact of AAS on other factors involved in exercise adaptation. Key Words: MALE RATS, ANABOLIC ANDROGENIC STEROIDS, OXYGEN CONSUMPTION, RED BLOOD CELLS, SUBMAXIMAL RUNNING ENDURANCE

Anabolic androgenic steroids (AAS), synthetic derivatives of the sex hormone testosterone, are used in various sports to enhance athletic performance. The majority of users are strength athletes in professional and amateur sports (8); however, there have also been reports of steroids taken by endurance athletes (e.g., swimmers, cyclists, runners) (14,29). Although there is much research on the effect of AAS on strength and power, there are few studies on their effect on endurance exercise performance. Several potential mechanisms have been suggested to cause the ergogenic effects of AAS treatment on endurance trained subjects. One of the possible mechanisms is improvement of skeletal muscle function by increasing protein synthesis (23,27). Another suggested mechanism is that AAS treatment counteracts the catabolic action of high circulating corticosteroid concentrations resulting from training (15). Another possibility is that AAS act through the central nervous system, allowing the subjects to train harder (3,26).

Endurance exercise training improves maximum oxygen consumption ($V\dot{O}_{2\text{max}}$) and running economy ($V\dot{O}_{2\text{submax}}$), which are considered key parameters in the improvement of submaximal running performance (17). AAS enhance erythropoiesis and increase blood volume (21). As oxygen carrying capacity is one of the factors determining $V\dot{O}_{2\text{max}}$ and submaximal running endurance (SRE) (4,11), it seems to be a likely mechanism through which SRE can be increased by AAS (20). The combined effect of endurance training and AAS treatment on these parameters is not well defined.

The effect of AAS on submaximal running performance is difficult to study in training athletes because of methodological limitations and ethical considerations. One of the first animal studies investigating the effect of AAS on submaximal running performance was conducted on spontaneously running male rats (28). This study showed that combining AAS treatment with voluntary wheel running delays fatigue during submaximal exercise and improves SRE by 41% compared with wheel running. The improvement in SRE could not be explained by either the increased voluntary training distance, the increased skeletal muscle oxidative capacity, or the increased skeletal muscle glycogen concentration compared with trained controls. It was also found that AAS treatment had no effect on maximal sprint speed and on SRE of untrained rats (28). These data suggest that the increase of submaximal running performance is due to the impact of AAS on factors involved in exercise adaptation and not to the fact that subjects train harder. Voluntary wheel running is a type of exercise train-
ing which improves SRE, running economy and $V_O^{2\max}$ of rats similarly to the changes induced by the submaximal training on a treadmill (19). Treadmill-trained rats have been employed by other researchers to study the effects of AAS on running performance, but the training and the tests have been performed using high exercise intensity (with additional weight, high speed and high grade of the treadmill belt) and changes in the SRE, $V_O^{2\max}$, running economy, and blood oxygen carrying capacity have not been investigated (25).

Studying the changes these parameters undergo under the combined influence of AAS treatment and endurance treadmill training will provide new evidence about the AAS effect on endurance exercise performance and the possible mechanisms causing this effect. To our knowledge, the effects of AAS treatment on these parameters in submaximal treadmill-trained rats have not been studied. Therefore, the aim of the study was to evaluate the separate and combined effects of treatment with an anabolic androgenic steroid and submaximal treadmill training on submaximal running endurability, maximum oxygen consumption, running economy, and oxygen carrying capacity in rats. We hypothesized that AAS can increase SRE, running economy, and oxygen carrying capacity of endurance treadmill-trained rats.

**MATERIALS AND METHODS**

**Animals.** Forty male Wistar rats (200–220 g) were used in this study. They were housed in individual metabolic cages and fed standard rat chow and water *ad libitum*. Body mass and the amount of food intake were measured daily. The rats were maintained at an ambient temperature of 21–24°C with a 12/12-h dark-light cycle. The experimental protocol was approved by the Ethical Committee on Human and Animal Experimentation of Medical University, Plovdiv. Rat care and all experimental procedures employed were in accordance with the policy statement of the American College of Sports Medicine on research with experimental animals.

**Training program and steroid treatment.** As running on a treadmill is a skilled activity for rats, before the experiment all rats were exercised on the treadmill (Columbus Instruments, Columbus, OH) at a speed of 27 m min$^{-1}$ and 5° elevation for 5 min d$^{-1}$, 3 d wk$^{-1}$ for 2 wk. Such workload induces no training adaptations (18) but familiarizes the rats with treadmill running and allows selection of rats that run spontaneously. About 15% of the rats refused to run on the treadmill and were discarded from the study at the end of the preliminary stage.

Compliant rats were randomized into two groups, sedentary ($N = 20$) and trained ($N = 20$). Trained animals exercised on the treadmill at a speed of 27 m min$^{-1}$, 5° elevation (which is about 70–75% $V_O^{2\max}$), 5 d wk$^{-1}$ for 8 wk. The duration of the exercise was 20 min d$^{-1}$ on the first day and was increased by 5 min every second day. By the end of the second week, it reached 40 min d$^{-1}$ and remained so until the end of experiment. The untrained rats were exercised on the treadmill 3 d wk$^{-1}$ for 5 min at the same speed and elevation as the training group to ensure familiarization with treadmill running. The treadmill speed was calibrated before each training session.

At the beginning of week 3, half of the trained and untrained rats were given an anabolic androgenic steroid, nandrolone decanoate (ND) 10 mg kg$^{-1}$ wk$^{-1}$ intramuscularly (Retabolil®, Gedeon Richter, Hungary) over a time period of 6 wk. The other half of the rats were given placebo (Pl, oil vehicle) in the same dose and duration. The injection was administered after the day’s training in m. gluteus medius, applying it alternatively each week in the contralateral side. Nandrolone decanoate is a long acting steroid ester and the recommended therapeutic dose of ND in humans falls within 50–100 mg (i.m.) range every 3–4 wk (about 0.4 mg kg$^{-1}$ wk$^{-1}$) (26). The supraphysiological dose of 10 mg kg$^{-1}$ wk$^{-1}$ as used in the present study is consistent with the high doses of AAS reported to have been used by athletes (14,20).

As it is assumed that the AAS effect is more pronounced in pretrained individuals (10), treatment with AAS started after the daily exercise reached a duration of 40 min. To differentiate exactly the effect of AAS, the absolute intensity and duration of the exercise of both training groups was kept constant from the beginning of treatment with ND/Pl until the end of experiment. Thus, four experimental groups were formed ($N = 10$): sedentary rats + Pl (SP, controls); sedentary rats + ND (SND); trained rats + Pl (TP), and trained rats + ND (TND). Two rats of the TP group dropped from experiment because of injuries and training protocol violation.

**Submaximal running endurance test.** Before the start of training, after weeks 2 and 6, and at the beginning of week 9 all groups were subjected to a submaximal running endurance test. SRE was determined in rats by having them run at 27 m min$^{-1}$, with 5° elevation of the belt until they could no longer maintain their position on the treadmill belt. The time taken to reach this stage was assessed as SRE.

**$V_O^{2\max}$, $V_O^{2\submax}$ and maximum time to exhaustion assessment.** At baseline, after week 6, and at the beginning of week nine all groups were subjected to $V_O^{2\max}$ test according to Bedford et al. (5). The parameters were measured using the Oxymax gas analyzing system for small animals (Columbus Instruments). The test was always carried out after a 1-d recovery period. The volume of the supplied air was 4.5 L min$^{-1}$. The gas analyzers were calibrated with a reference gas mixture before each test. The $V_O^{2\max}$ test protocol involved stepwise increasing of the treadmill speed and elevation as follows—I: 15 m min$^{-1}$, 5° elevation; II: 19 m min$^{-1}$, 10°; III: 27 m min$^{-1}$, 10°; IV: 27 m min$^{-1}$, 15°; V: 30 m min$^{-1}$, 15°; VI: 35 m min$^{-1}$, 15°; and VII: 40 m min$^{-1}$, 15°. Each step of exercise was 3 min long, and oxygen consumption ($V_O$) was measured at every minute. Each rat was placed in the chamber 10 min before exercising, and the lowest value of those measured during the last 4 min was taken to be the rest consumption. The highest $V_O$ measured at each workload was taken as a measure of each rat’s running economy ($V_O^{2\submax}$) for that...
workload, and at the last step, as $\dot{V}O_2_{\text{max}}$. Rats were removed from the test either when they could no longer maintain their position on the treadmill belt or in the plateau phenomenon (5). The time taken to reach $\dot{V}O_2_{\text{max}}$ was defined as the maximum running time to exhaustion of each rat (18).

**Blood analysis.** At the beginning of week 9, 10 d after the last administration of ND/Pl and 23–25 h after the last $\dot{V}O_2_{\text{max}}$ test, the rats were sacrificed under narcosis with 10 mg·kg$^{-1}$ thiopental i.p. Mixed blood was collected into heparinized tubes for investigation for amount of erythrocytes, mean corpuscular volume, hemoglobin and hematocrit. All parameters were determined by a hematological analyzer Coulter T-660 (Coulter Electronics, Inc., Hialeah, FL).

**Statistical analysis.** To test for the two main effects (exercise training and AAS administration) and for the interaction between them, the results of all tests, which were repeated several times during the experiment, were assessed by a two-way factorial analysis of variance for repeated measures. The results obtained only at the end of the experiment were analyzed with a two-way factorial analysis of variance. The level of significance was set at $P < 0.05$. Tukey post hoc test was applied for intergroup differences. Data are presented as mean ± SEM.

**RESULTS**

**Body mass and food intake.** At baseline, all rats in the groups had a similar body mass. During the experiment, the body weight of all rats gradually increased and at the end of the experiment the analysis failed to find any significant differences in their body mass: SP, 346.10 ± 10.70 g; SND, 323.30 ± 10.70 g; TP, 323.25 ± 11.96 g; TND, 340.40 ± 10.70 g ($P > 0.05$). There were no significant differences between the groups in the amount of food taken during the experiment (not shown).

**Submaximal running performance.** At baseline, the rats of all groups had similar SRE ($P > 0.05$). Endurance training had a significant main effect on SRE measured during the study period (Fig. 1). The trained rats improved significantly their SRE compared with baseline values ($P < 0.01$ at week 2, $P < 0.001$ at weeks 6 and 8) and compared with the untrained rats SRE measured after 2 wk (91.97 ± 6.85 min vs 35.33 ± 6.46 min, $P < 0.001$), 6 wk (107.78 ± 8.39 min vs 34.97 ± 7.91 min, $P < 0.001$), and 8 wk (128.39 ± 7.28 min vs 35.10 ± 6.87 min, $P < 0.001$) of training. We found no significant differences between the groups in the amount of food taken during the experiment (not shown).

ND treatment had a significant main effect ($P < 0.05$) on SRE measured at the end of the experiment, and there was a significant interaction ($P < 0.05$) of training and AAS treatment. ND treatment had no effect on untrained rats but improved the SRE of the trained group. At the beginning of week 9, TND group had significantly higher SRE than the other groups and TND rats ran 46% longer than TP rats during SRE test (152.15 ± 13.30 min vs 103.90 ± 11.77 min, $P < 0.05$). At the end of the experiment, the TP and TND groups had greater SRE than that of SP rats by 196% and 336%, respectively. Between weeks 2 and 9 the TP group improved their SRE by 18% ($P > 0.05$), whereas the TND group improved their SRE by 60% ($P < 0.01$). There were no significant differences in the SRE between both untrained groups throughout the experiment.

**Maximal running performance.** There were no differences in the maximum running time to exhaustion in the incremental maximal treadmill test at the beginning of experiment (Fig. 2). Endurance training had a significant main effect on maximum running performance during the study period. The trained groups increased their maximum running time to exhaustion in comparison both with the base-

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**FIGURE 1—Submaximal running endurance (min) of experimental groups measured at the beginning of experiment, 2, 6, and 8 wk later. *$P < 0.001$ (trained vs sedentary); **$P = 0.01$ (TND vs TP by 46%).**

**FIGURE 2—Maximum running time to exhaustion (min) in incremental running test of the experimental groups at the beginning of experiment and 6 and 8 wk later. *$P < 0.01$ (trained vs sedentary); **$P < 0.001$ (trained vs sedentary).**
Maximum oxygen consumption. Endurance training had a significant main effect on \( V_{O2_{max}} \). There were no differences between the groups in the \( V_{O2_{max}} \) measured at baseline \((P > 0.05)\) and after 6 wk \((P > 0.05)\) (Fig. 3). At the end of experiment the trained rats \( V_{O2_{max}} \) was higher than that of the sedentary rats by 4\% \((73.7 \pm 1.1 \text{ mL·kg}^{-1}·\text{min}^{-1} \text{ vs } 70.5 \pm 1.0 \text{ mL·kg}^{-1}·\text{min}^{-1}; P < 0.05)\). Between week 6 and the beginning of week 9, the TP and TND rats increased significantly their \( V_{O2_{max}} \) \((69.7 \pm 1.1 \text{ mL·kg}^{-1}·\text{min}^{-1} \text{ vs } 74.1 \pm 1.2 \text{ mL·kg}^{-1}·\text{min}^{-1}, P < 0.01\) and \(70.1 \pm 1.3 \text{ mL·kg}^{-1}·\text{min}^{-1} \text{ vs } 73.3 \pm 1.8 \text{ mL·kg}^{-1}·\text{min}^{-1}, P = 0.01)\). ND treatment had no effect on \( V_{O2_{max}} \) of both untrained and trained rats.

Running economy. At the end of experiment, the \( V_{O2_{submax}} \) measured in the first three steps during \( V_{O2_{max}} \) test showed no differences between the groups. There was a main effect of training on running economy during the fourth step \((27 \text{ m·min}^{-1}, 15° \text{ elevation})\) and the trained rats had significantly lower \( V_{O2_{submax}} \) than the sedentary rats \((65.3 \pm 0.9 \text{ mL·kg}^{-1}·\text{min}^{-1} \text{ vs } 68.0 \pm 0.9 \text{ mL·kg}^{-1}·\text{min}^{-1}; P < 0.05)\). The \( V_{O2_{submax}} \) measured during the fifth, sixth, and seventh steps of exercise intensity were not analyzed because of the small and unequal number of measured values in the groups. There was no effect of ND treatment on \( V_{O2_{submax}} \) of both untrained and trained rats in the different steps of exercise intensity.

There was a significant main effect of training on \( V_{O2_{submax}} \) when it was analyzed as %\( V_{O2_{max}} \) (Fig. 4). At the first step the trained rats had lower values of %\( V_{O2_{max}} \) than sedentary by 5\% \((P < 0.01)\), at the second step by 6\% \((P < 0.05)\), at the third step by 7\% \((P < 0.01)\), and at the fourth step by 9\% \((P < 0.01)\). There was no effect of ND treatment on %\( V_{O2_{max}} \) of both untrained and trained rats in the different steps of exercise intensity.

Oxygen carrying capacity. The results of the study of blood oxygen carrying capacity assessed by the amount of erythrocytes, hemoglobin, hematocrit, and the mean corpuscular volume of erythrocytes (MCV) are presented in Table 1. All values of the red blood cells parameters measured in the groups were within the physiologic reference values for male Wistar rats (6). There were no significant main effects of training and AAS treatment on the erythrocyte count, but there was a significant two-way interaction \((P < 0.05)\). AAS treatment for 6 wk elevated the erythrocytes only in the SND group in comparison with SP \((P < 0.05)\), and there was no effect on the TND group. Training resulted in higher red blood cell count only in TP in comparison with SP \((P < 0.05)\), but there was no difference in the erythrocyte count between the two trained groups \((P > 0.05)\). There was a significant main effect of training on hemoglobin concentration and trained rats had higher hemoglobin than untrained animals \((161.56 \pm 1.42 \text{ g·L}^{-1} \text{ vs } 157.25 \pm 1.39 \text{ g·L}^{-1}; P < 0.05)\). There was a significant main effect of training on hematocrit and trained rats had higher hematocrit than sedentary rats \((0.50 \pm 0.01 \text{ vs } 0.47 \pm 0.01; P < 0.05)\). Training had no main effect on MCV. ND treatment had a significant main effect on MCV \((P < 0.01)\), and there was a significant interaction between training and AAS administration \((P < 0.05)\). ND treatment had no effect on trained rats but SND group had a significantly lower MCV than the other groups at the end of experiment. The red blood cells parameters of SND group showed an increase of the number of erythrocytes but with reduced MCV (within the normal limits) and without a respective increase in the hemoglobin and hematocrit in comparison with the controls. There were no differences in red blood cells parameters between TP and TND group.

**DISCUSSION**

The fact that there were no differences in the body weight and the amount of food taken by the rats indicated that the
training used and/or the treatment with AAS did not impair the natural growth of the rats, and this is consistent with the results reported by Van Zyl et al. (28).

To our knowledge, this is the first study to show that anabolic androgenic steroid treatment combined with endurance treadmill training in rats delays fatigue during submaximal exercise. Our results have shown clearly that supraphysiological doses of nandrolone decanoate administered together with submaximal treadmill training improve the SRE of male rats better than the training alone does. This was proved by the different magnitude of SRE improvement of the two trained groups subjected to a stable training identical in intensity and duration during the treatment with AAS/placebo. These results corroborate the findings of a study on the combined effect of AAS and training on SRE of spontaneously running male rats (28).

In contrast to AAS effect on SRE, treatment with nandrolone decanoate did not increase the maximal running time to exhaustion to a greater extent than training alone. These different effects can be accounted for by the fact that performance SRE and maximal running time are dependent on different mechanisms. Davies et al. (9) report that sprint training in rats increases their maximal endurance and maximal sprint speed, whereas it has no affect on muscle oxidative capacity or SRE. Differences in the degree and rate of change of both parameters in rats have been reported also in submaximal training on treadmill (18).

These results, supporting previous research (1), show that AAS treatment does not increase the aerobic power either of untrained or endurance trained animals. The values of VO2max we measured in the study are consistent with the data about the Wistar male rats in the methodological study of Bedford et al. (5) and to the established time parameters of change in rats. No change of VO2max was found after 8 wk of treadmill training (18), but increases of 14–18% have been reported with 10–12 wk of training (18,22). The 4% increase of VO2max we established in this study at the beginning of week 9 of training suggests that initial changes have been registered in this parameter. We only observed the plateau phenomenon in three rats tested. There are other authors reporting similar plateau of plateau in the VO2max test in part (5) or in all studied rats (18).

The training protocol in this study exerted different effects on the rate and extent of improvement in SRE and VO2max. In the trained rats, SRE increased significantly as early as during the second week of training whereas the first significant differences in VO2max were found only at the beginning of week 9 of experiment. The slight increase of VO2max in comparison with the threefold increase of SRE in trained rats at the end of experiment suggests that changes in VO2max cannot serve as an accurate predictor of SRE improvement (18). Thus, the combined effect of submaximal training and AAS treatment on SRE cannot be explained by changes in VO2max.

Our results indicate that treatment with AAS does not change running economy to a greater degree than training alone. Higher running economy (lower VO2 at a specific intensity of submaximal exercise) is considered an advantage in endurance sports as it leads to utilization of lower percentage of VO2max (17). This increases the running speed at equal %VO2max and improves sports achievements assessed by the time (speed) needed to run a specific distance even for trained athletes with similar values for VO2max (4,7). Our results for VO2submax presented as %VO2max showed that submaximal training induced a better adaptation of the trained rats and lowered the degree of utilization of their oxygen capacity in comparison with the untrained rats subjected to exercise at the same absolute intensity. There is a clear tendency towards increasing the differences in %VO2max between the untrained and trained rats with the increase of exercise intensity. The absence of differences in %VO2max between the two trained groups in this experiment suggests that although AAS improve the SRE of trained rats, this might not improve performance assessed by the time (speed) needed to run a definite distance in a greater degree than the training alone.

A 6-wk treatment with AAS did not increase the blood oxygen carrying capacity of the untrained rats, nor did it increase that of the trained rats, which shows that the effect AAS have on SRE cannot be due to the increase of blood oxygen capacity. Our findings agree with the results of studies conducted with humans (2) and trained horses (16) in which at least 3 months of AAS treatment are required to increase hemoglobin and/or red cell volume. As oxygen carrying capacity is one of the factors determining VO2max (4) these data are consistent with the absence of differences in the values of VO2max we found between the TP and TND groups at the end of experiment.

Our results show that AAS treatment can increase SRE only in combination with endurance training. Although nandrolone decanoate failed to have any effects on SRE in untrained rats, the interaction with training can be interpreted as AAS speeding up the adaptation processes caused by the training itself. The combined effect of ND and training on SRE is not related to changes in variables determining O2 transport to the working muscles. Therefore, our findings suggest that the improvement of SRE is due to the impact of AAS on exercise induced peripheral alterations in the skeletal muscles. It does seem that the im-

### Table 1: Red blood cells parameters of the studied groups at the end of experiment; values are expressed as mean ± SEM.

<table>
<thead>
<tr>
<th>Variable</th>
<th>SP (N = 10)</th>
<th>SNP (N = 10)</th>
<th>TP (N = 8)</th>
<th>TND (N = 10)</th>
<th>T vs S</th>
<th>ND vs Pl</th>
<th>T-ND</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythrocytes (10¹² L⁻¹)</td>
<td>7.86 ± 0.18</td>
<td>8.67 ± 0.22</td>
<td>8.73 ± 0.21</td>
<td>8.53 ± 0.24</td>
<td>NS</td>
<td>NS</td>
<td>*</td>
</tr>
<tr>
<td>Hemoglobin (g L⁻¹)</td>
<td>156.00 ± 1.31</td>
<td>163.13 ± 1.58</td>
<td>160.00 ± 2.49</td>
<td>160.00 ± 2.49</td>
<td>*</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Hematocrit (L L⁻¹)</td>
<td>0.47 ± 0.01</td>
<td>0.47 ± 0.01</td>
<td>0.47 ± 0.01</td>
<td>0.49 ± 0.01</td>
<td>*</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>MCV (FL)</td>
<td>59.70 ± 0.98</td>
<td>55.04 ± 0.72</td>
<td>59.19 ± 1.14</td>
<td>58.61 ± 0.64</td>
<td>NS</td>
<td>NS</td>
<td>*</td>
</tr>
</tbody>
</table>

* P < 0.05; ** P < 0.01; * P < 0.05 (in comparison with SP); *P < 0.01 (in comparison with TP); *P < 0.05 (in comparison with TP and ND).
improvement of SRE is not due to the higher skeletal muscle oxidative capacity because data from other studies suggest that AAS treatment on endurance-trained male rats does not increase the mitochondrial enzyme activity (24,28) and the utilization of fats (13) in a greater degree than training alone. Although the physiological mechanisms for the AAS induced increase of the submaximal running endurance cannot be determined from the data presented here, some potential mechanisms can be suggested. Data from recent studies show that nandrolone decanoate treatment in endurance treadmill-trained male rats improves contractile responses of skeletal muscles (30) and may improve the tolerance to exercise training by increasing the levels of stress protein HSP72 in fast-twitch fibers (12) more than training alone. Further studies are necessary to investigate whether these effects of nandrolone decanoate on skeletal muscles of trained male rats are related to the improvement of SRE. Although the present investigation demonstrates the improvement of SRE of endurance-trained subjects after treatment with supraphysiological doses of nandrolone decanoate, care should be taken when one extrapolates data from animal models to humans. The abuse of high doses of AAS by athletes is associated with many somatic and psychosocial adverse effects on health (20,29).

In conclusion, the present findings show that treatment with AAS administered in combination with submaximal exercise on a treadmill enhances the SRE of male rats to a greater degree than does the training alone. AAS does not increase VO2max and the oxygen carrying capacity and does not improve running economy more than training alone; therefore, these parameters cannot explain the improvement of SRE. These results suggest AAS treatment effects other factors not measured in this study to induce adaptations leading to the increase of submaximal running endurance.

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