Effects of continuous and interval running training on serum growth and cortisol hormones in junior male basketball players

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Effects of two different eight-week aerobic training programs consisting of continuous (CR) or extensive interval running (IR) on serum growth (GH) and cortisol hormones in 33 male basketball players aged 15–16 were assessed. The CR group ran 4.8 km and the IR group ran 4×1.2 km, using equal work-to-rest ratio, three times per week. Aerobic power scores of all subjects and anaerobic power marks of the training subjects increased (p<0.01). Upon exertion, though serum GH levels increased in both exercise groups (p<0.01) prior to and following training; cortisol levels increased only in the IR group prior to training, and in both exercise groups following training (p<0.05). Following the eight week period, resting cortisol levels rose in the training (p<0.05) and control (p<0.01) groups.

To conclude, an 8-week training program consisting of continuous or extensive interval running has been effective on acute GH and cortisol secretion in 15–16 year-old male athletes.

Keywords: aerobic training, basketball, body fat ratio, serum growth hormone, serum cortisol

Assessing the effects of endurance training and different types of activities on serum hormones in adolescents has become more interesting, especially with the fall in starting age to high level competitive sports in recent years (1, 2, 10, 13, 26). Most of the research conducted so far have focused on the acute hormonal responses to exercise, and the effects of prolonged training in adolescence on blood hormone levels have not been investigated extensively. Some researchers point out that intensive training performed in puberty is effective on the anthropometric and motor characteristics of children, while others suggest that these effects result from natural improvement upon growth and maturity (1, 2, 5).

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Beside improving aerobic capacity (2), endurance training involving either continuous or extensive interval running decreases body fat (1), resting heart rate (25), and blood pressure (7). Furthermore, a continuous form of loading is reported to be more effective when compared with interval loading (1).

Studies investigating hormone and exercise relationships have shown that an increase occurs in blood hormone concentrations during exercise (10, 12, 15) and that there is a strong relationship between exercise and hormone levels (10, 23). At the same exercise intensity, hormone release is greater in trained people than the untrained ones (4, 10, 19), or nearly the same during supra-maximal exercise (21, 22). Type, intensity, duration of the exercise, and the training level of the person effect hormone secretion (9, 10). In studies concerning GH, it has been stated that the release of this hormone increases (10, 26), decreases or remains unchanged (20) depending on training status. Unlike GH, cortisol does not increase with low intensity exercise, but it does so with strenuous exercise (10, 11). In different studies; cortisol, known as a stress hormone, has been found to increase (12), decrease (16), or stay stable (6).

This research was conducted to assess the effects of 8-week aerobic training programs consisting of either continuous or extensive interval running on serum growth and cortisol hormones, together with body fat ratio, resting heart rate, and blood pressure scores in 15–16 year-old male basketball players.

Materials and Methods

Subjects

A total of 36 healthy, 15–16 year-old male basketball players participated in this study voluntarily. Prior written consent was obtained from the subjects and their parents. Subjects were randomly grouped as controls (C, n = 12, 15.6±0.5 yr), continuous running (CR, n = 12, 15.7±0.5 yr), and extensive interval running (IR, n = 12, 15.8±0.4 yr), paying attention to homogeneous age distribution among groups (Pearson, p>0.05). Controls were warned not to perform any physical activity. Subjects underwent a detailed medical examination before the study. During the training period, two subjects – one from each group – who discontinued the study were not evaluated.

Test procedure

The training program consisted of running three times a week for eight weeks on a synthetic track between 8:00–9:00 AM. Free warm-up was allowed before each session. Laboratory tests were carried out in the nearby sports centre on site and in the university hospital endocrinology laboratory. Tests were rehearsed twice prior to the actual ones in order to familiarise the athletes. Average temperatures were 13 °C and 18 °C, and relative humidity rates were 73.7% and 76.0% during the first and last measurements, respectively. Body height and weight, resting heart rates (RHR), systolic and diastolic blood pressures were recorded, body fat ratios were calculated, and aerobic and anaerobic power tests were performed two days prior to and following the training program. Blood samplings for hormone measurements were done at the start and the end of both first and last training sessions. Subjects were requested to completely rest the day before the tests.

Training program

Subjects of the CR training program (n = 11) ran 4800 m continuously at 80% of their maximal heart rate (HR_{max}). Subjects of the IR program ran the same total distance as 4×1200 m runs also at 80% of their HR_{max}. They were allowed to 1:1 active resting between repetitions. Target heart rates (THR) to determine the training intensity were calculated with the maximal heart rate reserve method (9):

$$THR = (HR_{max} - RHR) \times 0.80 + RHR$$

where

$$HR_{max} = 220 - age$$
 (yrs), and $RHR = resting$ heart rate

During training, the last 10-sec heart rates served as basis for exercise intensity adjustment. In order to reach target heart rate, athletes had to increase their speed over the same distance in subsequent sessions. In the IR group, if resting period was not sufficient, subjects continued to rest until their HR lowered to 120 bpm (9).

Anthropometric measurements

Height was measured bare-footed with a Holtain measurer of 0.1 cm sensitivity. Weight was measured with an Angel electronic scale of 0.01 kg sensitivity. Body fat ratios of the subjects were determined via the Behnke and Wilmore equation (9) using skinfold measurements taken from the right-hand side abdomen and leg with a Holtain skinfold caliper:

Body fat ratio (%) =
$$(4.95/body \text{ density}-4.50) \times 100$$
,

with

Body density $(g/ml) = 1.08543-0.00086 \times (abdominal skinfold, mm) - 0.0004 \times (leg skinfold, mm).$

Lean body mass (kg) = Body weight \times (1-body fat ratio/100).

Physiological measurements

The average scores of two repeated measurements of resting heart rate, systolic and diastolic blood pressures obtained using a sphygmomanometer and stethoscope following a 10-min horizontal rest, were recorded.

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Aerobic power

The aerobic power of the subjects was determined using the 20 m Shuttle Run Test (18). Subjects ran shuttles of 20 m between two lines, adjusting their speed according to the sound signal emitted by a tape recorder. The test started at a speed of $8.5 \text{ km} \cdot \text{h}^{-1}$, and the pace increased by $0.5 \text{ km} \cdot \text{h}^{-1}$ every minute until the subjects failed to maintain the required rhythm twice in a row. VO_{2max} scores in ml·min⁻¹·kg⁻¹ were obtained from related tables.

Anaerobic power

The Sargent vertical jump test was used to evaluate this variable (9). Subjects had three attempts, of which the best was considered. The Lewis formula in metric units was used to calculate anaerobic power:

$$P = 4.9^{1/2} \times (W) \times D^{1/2}$$

where P is anaerobic power, $kg \cdot m \cdot s^{-1}$; W is body weight, kg; and D is vertical distance, m.

Hormone measurements

Subjects reported to the lab at 7:30 AM after a 12 h fasting. Following a 15 min rest, 10 ml of blood was drawn from an antecubital vein, in the supine position. A sample was also drawn in EDTA-vacutainer tubes for haematological analysis using a Cell-Dyn 400 counter (USA). After a 30 min coagulation period at 25 °C, samples were centrifuged. Serums were kept at -20 °C until being assayed. Subjects started the training session following a 20 min rest after blood sampling, without consuming any liquids. Blood samples drawn right after completing the session were again processed accordingly. Hormone level determination was done via the CLEIA (Chemiluminescent Enzyme Immunoassay, DPC, USA) method. The sensitivity of the method is 0.003 ng \cdot ml⁻¹ for GH and 0.2 µg \cdot dl⁻¹ for serum cortisol. Sampling was performed at the same hour of the day, prior to and following the training period, before and after the 1st and 24th training sessions.

Statistical analysis

SPSS for Windows v6.0 was used for paired or unpaired *t*-tests to evaluate group data obtained before and after the training program. Anova and Duncan multiple range tests were applied to assess inter-group differences. The results were evaluated at p<0.05 and p<0.01 significance levels.

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Results

Physical and physiological data of the subjects obtained before and after the training program are given in Table I. Aerobic power figures of all groups were similar at the start of the study (p>0.05), and increased (p<0.01) at the end of the eight-week period. The extent of the increase was 15.4%, 12.9%, and 5.4% in CR, IR and C groups, respectively. Anaerobic power means of the groups, which were again similar to those obtained at the start of the study, increased by 2.7%, 3.6% (p<0.01), and 2.0% (p>0.05) at the end of the eight week period in CR, IR, and C groups, respectively. No significant differences among groups for any of the remaining variables at both stages of the study were found by Anova, with the exception of CR group's RHR being lower than that for the IR group before training, and lower than those for both IR and C groups following training (p<0.05).

The stature of all subjects increased (p<0.01). Mean body weight for the IR group increased at the p<0.01 level and that for the CR and C groups at the p<0.05 level. Decreases in body fat ratio for the training groups and increases in lean body mass for all groups (p<0.01) were observed. Following the training program; mean RHR, systolic (SBP) and diastolic (DBP) blood pressures for training groups (p<0.01), and only mean RHR of the C group decreased (p<0.05).

Using the Duncan multiple range test (at p<0.05), height increase was higher in the IR group than in the controls. The decrease in body fat ratio for the CR group and the increases in lean body masses for both training groups were higher than that for the C group. When compared with the controls, the extent of the decreases in RHR (f=13.9) and SBP (f=7.0) were significantly higher (p<0.05) in the CR and IR groups. For the CR group, the decreases in RHR and DBP were more pronounced than those observed for the IR and control groups, respectively.

To assess the possible effects of plasma volume change (ΔPV) on serum hormone levels, the formula of Dill and Costill (8) was used:

$\Delta PV (\%) = 100 \text{ Hb}^{0}(100 - \text{Hct}^{1})/\text{Hb}^{1}(100 - \text{Hct}^{0}) - 100$

where Hb is blood haemoglobin, Hct is haematocrit ratio, 0 and 1 stand for resting and post-exercise levels, respectively. Mean Hb and Hct levels increased in both CR and IR groups during both the first and the 24th sessions. The Δ PV figures were calculated as $-6.0\pm4.2\%$ and $-2.2\pm4.6\%$ in the first training session, and $-1.4\pm9.9\%$ and $-8.7\pm5.0\%$ in the 24th training session, for the CR and IR groups, respectively. Correcting serum hormone levels for plasma volume changes made no relevant differences in the intraand inter-group comparisons of the data. Thus, uncorrected data were used in the assessment.

According to Anova, there were no significant differences for resting GH, cortisol, Hb and Hct levels among groups at the start of the study (Table II). Before the 24th training session, only the mean resting cortisol levels of the control group were higher (p<0.05) than those of the IR group, using the Duncan test. For GH levels, significant increases (p<0.01) were found after the first training session in both CR and

IR groups; mean cortisol hormone levels of the IR group were also increased at the p<0.05 level. No group differences were observed, though (p>0.05). Mean Hb levels for the CR group increased (p<0.01) as well.

There was no significant change in the mean resting GH level in any group at the end of the training period (Table II). Resting cortisol hormone levels increased in IR, C (p<0.01) and CR (p<0.05) groups. The extent of the increases was not significantly different among groups. Anova test results showed no differences between the GH, cortisol, Hb and Hct values in any of the group, with the exception of the resting cortisol levels in C group at the end of the study being higher than those of the IR group. At the end of the 24th training session, mean GH (p<0.01) and cortisol (p<0.05) levels of both CR and IR groups increased. GH levels for the CR group reached significantly higher values (p<0.05) compared with the IR group. Post-exercise cortisol levels displayed higher figures (p<0.01) at the 24th training session comparing with the 1st session. The Duncan test revealed that the increase was higher in the CR group (p<0.05). Hb and Hct levels in the IR group increased (p<0.01) as well (Table II).

Discussion

The main principles to improve endurance capacity recommended by the ACSM were taken into consideration in the choice of the training program used in this study. It is known that training programs involving either continuous or interval running of aerobic nature both improve endurance (2, 3, 9, 16). In this respect, aerobic power scores of the subjects in both training groups improved, and the increases (p<0.01) have been higher than those observed by Adeniran and Toriola (2) in girls. Predominantly aerobic training positively influences anaerobic power as well (2, 3, 9). In fact, similar increases in anaerobic power in both training groups were obtained in the present study.

At the end of the training program, significant decreases (p<0.01) in resting heart rate, systolic and diastolic blood pressures in both groups were found. These decreases indicate the positive effects of endurance training methods on the cardiovascular system (25). The decrease in RHR in the control group may be explained by a possible activity of the particitants throughout the study.

Fat loss has been higher in the CR group compared with the IR group. This result supports the fact that prolonged aerobic training leads to greater fat loss when compared with short-term aerobic training. Lean body masses of subjects increased in all groups (p<0.01), implying that the primary energy source were fats. The increase in body weights thus points to an increase in muscle mass as well. The results obtained for the training groups in this study have similarities with the related literature (1, 14).

Following exercise, significant increments were observed in Hb levels for the CR group in the first training session and in both Hb and Hct levels for the IR group in the last training session. Changes recorded in this study are similar to those observed by Mathur (19). Since correction for plasma volume changes caused no relevant difference, uncorrected data were used.

BTATtBTATtBTATt 1754 ± 54 1769 ± 53 11.2^{**} 1784 ± 62 180.1 ± 61 174.1 ± 67 1755 ± 67 4.32^{**} 660 ± 85 667 ± 85 -3.05^{**} 682 ± 81 69.7 ± 84 -4.00^{***} 64.1 ± 7.1 64.9 ± 7.2 -2.44^{*} 14.9 ± 3.8 12.1 ± 3.0 9.34^{***} 14.6 ± 1.7 12.7 ± 2.1 4.55^{***} 12.6 ± 1.9 11.8 ± 1.8 1.75 56.0 ± 5.8 66.7 ± 8.5 -3.05^{**} 68.2 ± 8.1 60.7 ± 6.7 -4.98^{***} 55.9 ± 5.5 57.2 ± 6.1 -3.77^{**} 78.9 ± 2.3 $72.4 \pm 3.3^{**}$ $8.0.9 \pm 2.3$ 70.9 ± 2.3 71.6 ± 3.2 8.05^{***} 11.75 59.2 ± 5.5 57.2 ± 6.1 -3.77^{**} 78.9 ± 2.5 $72.4 \pm 3.3^{**}$ $8.0.9 \pm 2.3$ 76.6 ± 3.9 55.2 11.9 -3.77^{**} 78.9 ± 2.5 $72.4 \pm 3.3^{**}$ 80.9 ± 2.3 77.6 ± 3.9 57.2 ± 5.1 -3.06^{**} 78.9 ± 2.5 $72.4 \pm 3.3^{**}$ 80.9 ± 2.3 77.6 ± 2.3 77.6 ± 5.2 11.96 ± 5.2 11.96 ± 5.2 100.3 ± 12.0 103.1 ± 11.19 -5.60^{**} 99.3 ± 10.6 10.29^{**} 96.7 ± 12.2 98.3 ± 12.5 -1.96 41.0 ± 3.1 100.3 ± 12.0 103.1 ± 11.19 -5.09^{**} 96.7 ± 12.2 98.3 ± 12.5 -1.96 40.0 ± 3.1 100.3 ± 12.0 100.1 ± 11.19 -5.06^{**} 99.3 ± 10.7 102.9 ± 11.4 -5.99^{**} 96.7			CR group			IR group			Control group	
$ \begin{array}{llllllllllllllllllllllllllllllllllll$		BT	AT	t	BT	AT	t	BT	AT	t
$ \begin{array}{llllllllllllllllllllllllllllllllllll$		175.4 ± 5.4	176.9 ± 5.3	-11.2**	178.4 ± 6.2	180.1 ± 6.1	9.46**	174.1 ± 6.7	175.5 ± 6.7	4.32**
		66.0 ± 8.5	66.7 ± 8.5	-3.05*	68.2 ± 8.1	69.7 ± 8.4	-4.00^{**}	64.1 ± 7.1	64.9 ± 7.2	-2.44*
		14.9 ± 3.8	12.1 ± 3.0	9.34**	14.6 ± 1.7	12.7 ± 2.1	4.55**	12.6 ± 1.9	11.8 ± 1.8	1.75
$\begin{array}{cccccccccccccccccccccccccccccccccccc$		56.0 ± 5.8	58.4 ± 6.2	-8.87**	58.1 ± 6.4	60.7 ± 6.7	-4.98**	55.9 ± 5.5	57.2 ± 6.1	-3.77**
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$		78.9 ± 2.3	$72.4\pm3.3^{\$}$	8.53**	$80.9 \pm 2.3^{\#}$	76.9 ± 3.0	6.06**	79.1 ± 1.9	77.6 ± 2.5	2.39*
$\begin{array}{cccccccccccccccccccccccccccccccccccc$		122.3 ± 6.5	115.5 ± 7.9	5.59**	122.6 ± 5.9	116.8 ± 7.2	8.65**	121.4 ± 4.5	119.6 ± 5.2	1.79
$ \begin{tabular}{ l l l l l l l l l l l l l l l l l l l$		78.2 ± 2.5	73.6 ± 3.2	4.30^{**}	78.6 ± 2.3	75.6 ± 3.9	3.32**	77.3 ± 4.1	76.4 ± 5.1	0.80
$ \begin{tabular}{ c c c c c c c c c c c c c c c c c c c$	1.kg ⁻¹	41.0 ± 3.4	47.4 ± 3.5	-19.8^{**}	42.1 ± 3.3	47.6 ± 3.2	-8.81 **	39.6 ± 3.0	41.8 ± 3.1	-3.76**
an ± SD. as running interval running intig, t: paired t-test scores, dy mass, heart rate, blood pressure, c blood pressure, over (VO_2max),	_	100.3 ± 12.0	103.1 ± 11.9	-5.60^{**}	99.3 ± 10.7	102.9 ± 11.4	-5.99**	96.7 ± 12.2	98.3 ± 12.5	-1.96
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ining ing. 1: paired t-test scores, dy mass, heart rate, blood pressure, c blood pressure, over (VO_2max),	interva	ıl running								
ing, t: paired t-test scores, dy mass, heart rate, blood pressure, c blood pressure, over (VO ₂ max),	ining									
dy mas, heart rate, blood pressure, c blood pressure, over (VO ₂ max),	ning, t:	paired t-test score	ŝ							
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Table I

Resting PEx t-score Resting PEx t-score Resting GH ₁ , ng·ml ⁻¹ 2.0 ± 2.9 2.30 ± 9.4 -6.93** 0.7 ± 1.4 16.7 ± 10.0 -5.20** 1.2 ± 1.8 GH ₃₄ , ng·ml ⁻¹ 1.0 ± 1.4 2.80 ± 10.1 ⁵ -8.47** 1.0 ± 2.4 17.1 ± 11.1 -5.18** 0.8 ± 1.2 t-score 0.90 -1.75 -0.32 -0.19 -5.20** 12.1 t-score 0.90 -1.75 -0.37 9.6 ± 3.3 15.5 ± 5.9 -2.62* 10.1 ± 2.8' t-score 0.90 -1.75 -0.37 9.6 ± 3.3 15.5 ± 5.9 -2.62* 10.1 ± 2.8' t-score 0.90 -1.75 -2.25* 14.5 ± 4.3 192.2 ± 4.7 -2.29* 19.3 ± 2.8' t-score -2.80* -3.88** -3.58** -3.88** -0.10.83** -10.83** t-score 0.16 15.4 ± 1.1 15.6 ± 0.8 -2.29* 19.2 ± 4.7 -2.29* 19.2 ± 4.7 -2.29** 10.83** t-score -1.5.4			CR group			IR group		Control group
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$		Resting	PEx	t-score	Resting	PEx	t-score	Resting
t.score 0.90 -1.75 -0.32 -0.19 1.21 cortiol ₁ , µg·d ¹⁻¹ 11.7 ± 3.2 12.3 ± 4.4 -0.37 9.6 ± 3.3 15.5 ± 5.9 -2.62^* 10.8 ± 2.3 Cortisol ₂ , µg·d ¹⁻¹ 16.7 ± 5.7 20.1 ± 5.6 -2.25^* 14.5 ± 4.3 19.2 ± 4.7 -2.29^* 19.1 ± 2.8' t-score -2.80^* -3.88^{**} -4.52^{**} 19.2 ± 4.7 -2.29^* 19.1 ± 2.8' Hb ₁ , g ⁻¹ 15.4 ± 1.2 16.2 ± 1.3 -3.63^{**} 15.8 ± 1.1 16.0 ± 1.2 $-1.0.83^{**}$ -0.03^{**} -2.29^* 19.1 ± 2.8' -0.03^{**} -2.23^{**} $-1.0.83^{***}$ -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -10.83^{***} -1.15^{*} 10^{*} 0.70^{*} 0.70^{*} 0.70^{*} 0.40^{*} Hb ₁ ₂ , g [*] , g	$\begin{array}{c} \mathrm{GH}_1,\mathrm{ng}\mathrm{cm}^{l-1}&2\\ \mathrm{GH}_{24},\mathrm{ng}\mathrm{cm}^{l-1}&1 \end{array}$	2.0 ± 2.9 1.0 ± 1.4	$\begin{array}{rrrr} 23.0 \ \pm \ 9.4 \\ 28.0 \ \pm \ 10.1^{\$} \end{array}$	-6.93** -8.47**	$\begin{array}{cccc} 0.7 \pm 1.4 \ 1.0 \pm 2.4 \end{array}$	16.7 ± 10.0 17.1 ± 11.1	-5.20 ** -5.18 **	$1.2 \pm 1.8 \\ 0.8 \pm 1.2$
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	t-score	0.90	-1.75		-0.32	-0.19		1.21
trace -2.80^{*} -3.88^{**} -4.52^{**} -3.88^{**} -10.83^{**} Hb ₁ , g ⁺¹ 15.4 ± 1.2 16.2 ± 1.3 -3.63^{**} 15.8 ± 1.1 16.0 ± 1.2 -1.15 15.7 ± 0.7 Hb ₂₄ , g ⁺¹ 15.4 ± 1.1 15.6 ± 0.8 -0.34 15.0 ± 1.2 15.0 ± 1.2 -3.96^{**} 15.9 ± 0.9 Hb ₂₄ , g ⁺¹ 15.4 ± 1.1 15.6 ± 0.8 -0.34 15.0 ± 1.2 16.0 ± 1.2 -3.96^{**} 15.9 ± 0.9 t-score 0.06 1.53 -0.34 15.0 ± 1.2 16.0 ± 1.2 -3.96^{**} 15.9 ± 0.9 Hct ₁ , % 44.6 ± 1.1 45.4 ± 1.4 -2.03 44.4 ± 2.4 45.2 ± 2.1 -1.94 43.6 ± 2.0 Hct ₂₄ , % 45.3 ± 1.9 45.7 ± 1.8 -0.70 43.9 ± 2.6 45.7 ± 2.1 -6.14^{**} 44.1 ± 1.8 t-score -1.30 0.94 0.33 -1.60 -1.37	Cortisol ₁ , µg·dl ⁻¹ 11 Cortisol ₂₄ , µg·dl ⁻¹ 16	$.7 \pm 3.2$ 5.7 ± 5.7	12.3 ± 4.4 20.1 ± 5.6	-0.37 -2.25*	9.6 ± 3.3 14.5 ± 4.3	15.5 ± 5.9 19.2 ± 4.7	-2.62* -2.29*	$\begin{array}{rrr} 10.8 \ \pm \ 2.3 \\ 19.1 \ \pm \ 2.8^{\$} \end{array}$
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	t-score	-2.80*	-3.88**		-4.52**	-3.88**		-10.83^{**}
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	$\begin{array}{ccc} Hb_{1},g\cdot l^{-1} & 15 \\ Hb_{24},g\cdot l^{-1} & 15 \end{array}$	1.4 ± 1.2 1.4 ± 1.1	16.2 ± 1.3 15.6 ± 0.8	-3.63** -0.34	$\begin{array}{c} 15.8 \ \pm \ 1.1 \\ 15.0 \ \pm \ 1.2 \end{array}$	16.0 ± 1.2 16.0 ± 1.2	-1.15 -3.96**	$\begin{array}{c} 15.7 \ \pm \ 0.7 \\ 15.9 \ \pm \ 0.9 \end{array}$
Hct ₁ , % 44.6 ± 1.1 45.4 ± 1.4 -2.03 44.4 ± 2.4 45.2 ± 2.1 -1.94 43.6 ± 2.0 Hct ₂₄ , % 45.3 ± 1.9 45.7 ± 1.8 -0.70 43.9 ± 2.6 45.7 ± 2.1 -6.14** 44.1 ± 1.8 t-score -1.30 0.94 0.33 -1.60 -1.37 -1.37	t-score	0.06	1.53		1.49	-0.07		-0.40
Hct24, % 45.3 \pm 1.9 45.7 \pm 1.8 -0.70 43.9 \pm 2.6 45.7 \pm 2.1 -6.14** 44.1 \pm 1.8 t-score -1.30 0.94 0.33 -1.60 -1.37	Hct ₁ , % 44	i.6 ± 1.1	45.4 ± 1.4	-2.03	44.4 ± 2.4	45.2 ± 2.1	-1.94	43.6 ± 2.0
t-score –1.30 0.94 0.33 –1.60 –1.37	Hct ₂₄ , % 45	3 ± 1.9	$45.7~\pm~1.8$	-0.70	43.9 ± 2.6	$45.7~\pm~2.1$	-6.14^{**}	$44.1~\pm~1.8$
	t-score	-1.30	0.94		0.33	-1.60		-1.37
	R: continuous running,							
R: continuous running,	c: interval running,							
R: continuous running, R: interval running,	Ex: post-exercise,							
R: continuous running, A: interval running, Ex: post-exercise,	H: serum growth hormone,							
Recontinuous running, Re interval running, Exr: post-exercise, Eff: serum growth hormone,	b: blood haemoglobin,							
R: continuous running, R: interval running, H:s: post-exercise, H: serum growth hormone, tb: blood haemoglobin,	ct: haematocrit, 1: 1st training	g, ₂₄ : 24th traini	ng.					

Table II remone and blood narameter levels are- and not-everise before and following the training aroord

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Resting levels of serum GH and cortisol and the acute increases observed following both exercise types are in accordance with the literature (3, 9, 10, 12, 15, 17, 21, 23). At the same exercise intensity, less pronounced increases in GH (4) and cortisol (4, 10, 19) levels are generally found in the trained individuals, compared to the untrained ones. In the present study, post-exercise hormone levels at the 24th session were similar to the ones found at the first session for the IR group. In the CR group during the last session, however, the highly emotional state of four athletes who feared they might not complete the run, may have caused their cortisol $(22-30 \,\mu\text{g} \cdot \text{dl}^{-1})$ and GH $(37-40 \,\text{ng} \cdot \text{ml}^{-1})$ levels to be too high, affecting respective group means. In fact, there is evidence that excitement and panic may increase GH and cortisol levels (10, 11). There are also studies reporting higher post-exercise GH (21, 22) and cortisol (10, 21) levels in the trained state, especially if the exercise in question is supramaximal. Differences in type and intensity of the exercise in question might thus account for these different endocrine responses.

Various views are discussed in the literature on the effect of prolonged training programs on resting hormone levels. Whereas Zakas et al. (26) determined that resting GH levels of 13–16 year-old children increased with three months of continuous type exercise (p<0.05), Pyka et al. (20) observed no changes in general at the end of a 52 week weight training program. Data from the present study reveal non-significant GH changes in all groups. For cortisol, Carli et al. (6) determined that serum levels decrease depending on exercise duration in a 24-week swimming training progressively increasing with intensity. Kraemer et al. (16) observed an increase in an IR group and a decrease in a CR group. Hakkinen et al. (13) recorded an increase following a year of swimming training (p<0.05). In the present study, final resting cortisol levels increased. The smaller increases observed in the training groups might result from better stress management. Present work and similar ones (10, 12, 13, 16) indicate different endocrine adaptations to training programs of differing type, duration and intensity.

In fact, there are different views about how various types of training programs affect acute and chronic hormone responses (16, 17, 24). In the present study, endurance training programs of different type but of similar intensity affected GH levels differently. This result is differing from that described by Vanhelder et al. (24). Acute effects are similar to those reported by Kraemer et al. (17) and Frenkl et al. (10). For cortisol, the results of this study are again discordant with those of Vanhelder et al. (24), Kraemer et al. (17) and the CR group of Kraemer et al. (16), but are similar to those of Frenkl et al. (10) and the IR group of Kraemer et al. (16).

To conclude, an 8-week training program consisting of continuous or extensive interval running has been effective on acute GH and cortisol secretion in 15–16 year-old male athletes. Together with changes in some circulatory and anthropometric parameters, resting cortisol levels increased, while no significant changes took place in resting GH levels. Consequently, the effects of many factors affecting hormone secretion in exercising adolescents have yet to be to clarified. A longer training period that would also allow frequent measurements might be a promising approach.

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