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Effects of human chorionic gonadotropin administration on testicular testosterone secretion during prolonged exercise

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A decrease in testosterone concentrations in men after prolonged exercise has been confirmed in many studies, although testosterone responses are somewhat variable and seem to depend on the duration and intensity of the exercise (1, 2). The decline in serum testosterone levels after prolonged exercise generally is ascribed to a decrease in the rate of secretion caused by a decrease in testicular blood flow, an increase in body temperature, and an increase in plasma concentrations of prolactin and cortisol. Data have shown that testicular secretion of testosterone may be influenced by prolonged exercise (3), but no direct evidence has yet been adduced.

The aim of this study was to determine whether the administration of hCG would prevent the decrease in plasma levels of testosterone that occurs in men after prolonged exercise.

MATERIALS AND METHODS

A group of 18 adult male long-distance runners (i.e., marathon runners) volunteered for the study. All the runners had at least 5 years of experience at the regional competitive level. None were taking any concurrent medications. The experiment consisted of two sessions of running performed at a 72-hour interval. Both sessions occurred in the afternoon at 3:00 P.M. Before each session, the runners consumed a standard light lunch (60% carbohydrates, 20% fats, and 20% proteins; approximately 2,933 kJ).

During each session, the athletes ran for 90 minutes at a predetermined speed. The speed was determined in a field test performed 1 week before the experiment by a modification of Mader's procedure (4), in which the relation between blood lactate concentrations and running speed was assessed for each individual. For blood lactate assays, capillary blood samples were obtained from the ear lobe before each run and 1, 3, 5, and 7 minutes afterward. Lactate concentrations were determined with a YSI model 23L lactate analyzer (YSI, Yellow Springs, OH).

The linear relation between the peak lactate concentrations and the corresponding running speeds in the trial was determined for each individual, and the running speed that corresponded to a lactate concentration of 2 mmol/L^{-1} (v_2) was calculated by extrapolation or interpolation. This speed is considered to be the upper limit of exclusively aerobic metabolism for energy supply. In both sessions, the athletes performed the 90-minute run on a 400-m track at their own v_2. The runners were apprised of their performance at every lap.

During the first session of running, four venous blood samples were obtained: before exercise (E0), at the end of the 90-minute run (E1.5), and 90 (R1.5) and 180 minutes (R3) after exercise. The athletes were divided randomly into groups A (n = 9) and B (n = 9). At the same time on the next day, the men in group A were given 5,000 IU of hCG i.m. The men in group B received a placebo (saline solution). In the second session, which began 48 hours after the injection of hCG, four blood samples (E0′, E1.5′, R1.5′, and R3′) were obtained at the same time as in the first session.

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TABLE 1

Hormone concentrations in exercise sessions 1 and 2.

	Session 1 concentration				Session 2 concentration			
Hormone	E0	E1.5	R1.5	R3	E0′	E1.5′	R1.5′	R3′
ACTH (pg/mL)	15.2 ± 6.6	46.9 ± 18.8*	19.7 ± 15.3†	16.0 ± 6.5†	17.6 ± 7.8	48.8 ± 9.7‡	21.2 ± 12.3	18.2 ± 9.5
Cortisol (μg/L)	121.2 ± 41.4	326.2 ± 59.5*	258.0 ± 90.4*†	221.9 ± 109.4†	112.9 ± 36.6	319.7 ± 55.9‡	244.0 ± 54.7‡§	210.9 ± 110.9§
Prolactin (ng/mL)	7.6 ± 5.0	58.9 ± 21.5*	18.7 ± 5.0*†	10.7 ± 4.6†‖	6.6 ± 3.5	57.5 ± 27.8‡	15.1 ± 5.2‡§	9.9 ± 2.8‡§¶
LH (mIU/mL)	2.8 ± 0.3	2.6 ± 0.4	2.1 ± 0.2*	3.0 ± 1.4	1.5 ± 0.5	1.0 ± 0.4‡	0.8 ± 0.3‡	1.1 ± 0.4
FSH (mIU/mL)	3.4 ± 1.3	3.2 ± 1.2	3.9 ± 1.1	3.8 ± 1.1	2.3 ± 0.8	2.2 ± 1.4	2.3 ± 0.7	2.3 ± 0.9
DHEAS (μg/mL)	2.0 ± 0.4	3.4 ± 0.8*	2.5 ± 0.8†	2.6 ± 0.9	2.3 ± 0.7	3.0 ± 0.6	2.4 ± 0.5§	2.7 ± 0.8
Testosterone (ng/mL)	2.8 ± 0.6	4.4 ± 1.3*	2.3 ± 1.1†	2.0 ± 0.9*†	6.3 ± 1.0	9.4 ± 1.4‡	7.4 ± 0.7‡§	7.9 ± 1.4‡§
Free testosterone (pg/mL)	12.2 ± 3.3	18.2 ± 5.3*	9.9 ± 4.2*†	8.2 ± 3.3*†‖	33.7 ± 5.9	56.4 ± 10.7‡	43.3 ± 6.9‡§	46.2 ± 8.8‡
E_2 (pg/mL)	11.0 ± 2.3	19.5 ± 5.3*	13.5 ± 4.0*†	12.5 ± 2.2*†	44.5 ± 14.6	86.2 ± 63.1‡	49.1 ± 17.4	56.3 ± 21.7‡
Androstenedione (pg/mL)	320.1 ± 77.4*	663.0 ± 107.6†	276.2 ± 148.2*†	203.4 ± 96.6	530.4 ± 182.4	925.5 ± 168.5‡	454.9 ± 144.6§	497.4 ± 189.7§
Sex hormone-binding globulin (nmol/L)	29.2 ± 11.9	32.1 ± 18.2	32.1 ± 13.4	31.1 ± 11.0	32.6 ± 9.6	31.5 ± 12.9	29.8 ± 11.4	29.0 ± 8.1

Note: Values are means ± SD. E = exercise; R = recovery. Values are for group A.

* $P<.05$ vs. E0.

† $P<.05$ vs. E1.5.

‡ $P<.05$ vs. E0′.

§ $P<.05$ vs. E1.5′

‖ $P<.05$ vs. R1.5.

¶ $P<.05$ vs. R1.5′.

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The samples were placed in tubes containing ethylenediaminetetraacetic acid and centrifuged at 4°C. The plasma was kept at −20°C until analysis. At the end of both running sessions, capillary blood samples were obtained for determination of lactate concentrations by the procedure used in the field test.

The study was approved by the institutional review board of the University of Siena. Written informed consent was obtained from each subject.

RESULTS

The results are expressed as means ± SD. Variables that did not have a gaussian distribution were logarithmically transformed before analysis. For clarity, the data that were not log-transformed are presented in Table 1. Standard statistical methods, including analysis of variance for repeated samples and Student's *t*-tests for paired samples (Snedecor and Cochran) were applied as appropriate. Statistical significance was set at $P<.05$.

There were no differences in anthropometric parameters, ν_2 (1.87 ± 0.48 vs. 1.99 ± 0.74 mmol/L), or running speeds (15.1 ± 0.45 km/h vs. 15.15 ± 0.47 km/h) during the two sessions. The hematocrit increased after exercise (both sessions pooled: 46.9% ± 3.7% before exercise vs. 48.2% ± 4.3% after exercise; $P=.064$). There was a corresponding decrease in plasma volume of approximately 5% after recovery. The mean (±SD) hematocrit was 46.3% ± 4.1% after 90 minutes and 46.1% ± 3.9% after 180 minutes.

Plasma hormone concentrations during both sessions are shown in Table 1. Plasma concentrations of testosterone and free testosterone were higher than basal levels at the end of exercise (sample E1.5) during both sessions in both groups, and the percentage increments in testosterone and free testosterone were similar. However, in the first session, prolonged exercise elicited significant decrements in plasma testosterone and free testosterone levels during recovery (samples R1.5 and R3), with free testosterone showing more significant changes than testosterone. The injection of hCG, but not a placebo, changed plasma testosterone and free testosterone responses to prolonged exercise, although they remained elevated above baseline values.

DISCUSSION

The results of this study show a reduction in the recovery of testosterone levels after an exercise-induced increase. This reduction did not occur when hCG was administered before exercise. Free and total testosterone increased immediately after exercise; ACTH, cortisol, and DHEAS also increased, reflecting increased release of corticotropin-releasing hormone by the hypothalamus. At the same time, a significant increase in prolactin and a lesser increase in E_2 were observed. In the subsequent recovery phase, ACTH, cortisol, DHEAS, and prolactin returned to their preexertion levels, whereas free and total testosterone dropped significantly below basal levels. The same results have been reported previously in the literature.

The hormone patterns during exercise were similar during the second session, which was performed 48 hours after the administration of hCG. The main difference during the second session was that testosterone recovered to its initial levels or rose even higher. The administration of a placebo did not prevent the reduction in testosterone and free testosterone levels after exercise (group B).

This permits a clear distinction between the effects of hCG and training. Prolonged exercise caused a stress-stimulated release of large quantities of corticotropin-releasing hormone by the paraventricular nucleus of the hypothalamus, which may both activate the pituitary-adrenal axis and block GnRH release and gonad function. Direct administration of hCG stimulates the Leydig cells to produce testosterone. This demonstrates that the testicles are able to respond to LH and that the reduction in plasma levels of testosterone that occurs after exercise is due primarily to a reduction in pituitary LH secretion.

In conclusion, the present results show that heavy exertion causes endocrine hypofunction in the testicles secondary to an increase in corticotropin-releasing hormone secretion induced by physical exertion and by stress-induced central and peripheral endocrine, metabolic, and behavioral changes. The administration of hCG blocks the effects of physical stress by directly stimulating endocrine testicular function.

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