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Adrenal and Testicular Androgens in Serum of Men after Physical Endurance Training

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격심한 운동후 남성 혈청 내 부신 및 정소 Androgen 변화

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ABSTRACT : Recently many studies have reported that total and bioavailable androgens reduced in male and female athletes and that physical exercise reduces the body weight and increases the reproductive abnormalities such as oligomenorrhea, anovulation, inadequate luteal phase, and delayed puberty in women by the inhibition of the hypothalamic-pituitary-gonadal (HPG) axis. In addition, high mileage endurance running, psychological stress, and military endurance training in men also reduce the secretion of reproductive hormones. To investigate the effects of physical endurance exercise on the secretion of reproductive hormones in men, androgenic hormones from adrenal glands and testis were measured in serum by the conventional radioimmunoassays after long-term (more than 3 months), short-term (1 week), and acute (1~2 hours) physical exercises. Androgenic hormones from adrenal glands and testis such as total testosterone (TT), free testosterone (fT), dehydroepiandrosterone (DHEA), and androstenedione (A) decreased after these strenuous endurance trainings, whereas ACTH, cortisol, and dehydroepiandrosterone sulfates (DHEAS) increased. Gonadotropins (LH and FSH) were not influenced by the physical exercises. Based upon the present results, we assume that the decrease in adrenal and testicular androgens by physical endurance exercises might be associated with the reproductive abnormalities in athletes by unknown factor(s) in addition to the HPG axis disturbance.

Key words : Androgens, Endurance training, Exercise, Acute exercise.

요 약 : 최근 격심한 운동은 시상하부-뇌하수체-생식소 (HPG) 축의 교란을 일으켜 여성과 남성의 생식기능에 장애를 유발하는 것으로 알려지고 있다. 격심한 운동을 하는 여성 운동선수의 경우는 과소월경, 무배란, 황체기 단축, 성성숙 지연 등에 따른 생식호르몬의 변화가 유발된다. 남성 운동선수의 경우는 과도한 운동, 정신적 장애와 긴장, 군 훈련 등 격심한 운동으로 생식호르몬의 분비에 이상이 생긴다고 보고되고 있으나, 임상적인 증상은 미약한 것으로 보고되고 있다. 본 연구는 격심한 운동이 생식이상을 초래하는 원인을 밝히는 연구의 일환으로, 정상 남자가 장기적 운동 (3개월 이상), 단기간의 격심한 운동 (1주일), 그리고 1~2시간의 집약된 심한 운동 후 혈청내 호르몬의 변화를 방사면역측정법을 이용하여 측정하였다. 세 가지 운동 형태의 경우 모두가 부신 및 고환에서 분비되는 총 testosterone (TT), 유리형 T (fT), DHEA, A의 분비 변화를 일으켰다. 특히 단기간과 1~2시간의 격한 운동 후 일시적으로 증가되었다가, 이어 정상치 이하로 감소하는 것이 발견되었다. Cortisol, DHEAS, ACTH 등은 운동 후 지속적인 증가를 초래하였다. 장기간의 운동 후에서는 생식소자극호르몬의 분비가 큰 변동이 없었으나, 단기간 운동 후에는 FSH의 감소가 발견되었다. 이러한 결과는 부신 및 고환의 androgen이 격한 운동에 의해 감소되며, 장기간의 지속적인 남성호르몬의 변화는 생식기능의 이상을 초래하는 원인이 된다고 가정된다. 또한 단기간의 격한 운동이 HPG축 호르몬의 변화를 유발하나, 장기간의 운동으로 일어나는 androgen의 감소는 HPG축 이외의 요인(들)에 의해 남성호르몬의 분비이상이 생긴다고 가정된다.

INTRODUCTION

Reproductive dysfunction is common among female athletes with very low body mass, such as ballet dancers, long distance runners, marathoners, gymnasts, figure skaters, and basket ball players, whereas reproductive abnormalities are substantially lower in swimmers and cyclers (Highet, 1989; Constantini and Warren, 1994). Strenuous endurance training, physical exercise,

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has been associated with inhibition of the hypothalamic-pituitary -ovarian axis (Cumming, 1990). Furthermore, acute severe exercise decreases further LH pulsatile release in normally menstruating runners (Cumming, 1990). In a few studies on females from various types of athletic discipline, profiles of reproductive hormones in amenorrheic athletes generally showed lower levels of gonadotropins, prolactin, and estrogens but elevated levels of testosterone (T) and dehydroepiandrosterone sulfates (DHEAS).

In men, reduced levels of total testosterone (TT) and free testosterone (fT) in serum have been associated with high mileage endurance running, resistance training, lowering body fat, weight lifting, and making weight by wrestlers (Wheeler et al., 1984, 1991; Ayers et al., 1985; Strauss et al., 1985; Hackney et al., 1988; Carli et al., 1992). LH pulsative release has been described as reduced in very highly trained runners (MacConnie et al., 1986; McColl et al., 1989) and as normal in runners with lower training intensity and fitness (Rogol et al., 1984). Supraphysiological high mileage running has been associated with elevated cortisol levels and impaired response to ACTH (Barron et al., 1985). Hypercortisolemia may also be associated with inhibition of the hypotalamic-pituitary-gonadal (HPG) axis (Cumming et al., 1983).

Most studies showed an increase of 10~30% in plasma testosterone during short- term exercise due to hemoconcentration, decreased degradation, and probably increased testicular secretion (Schmid et al., 1982; Opstad, 1992). Opstad and Aakvaag (1982, 1983) demonstrated that the plasma levels of androgens decreased under prolonged physical stress, such as bicycle competition, military operations, surgery, and anesthesia.

The present study is designed to investigate the endocrinological effects of long-term endurance exercise (running training for 3 months), and of short-term physiological stress (for one week), and of acute and prolonged exercise (for 2 hour) on the secretions of adrenal and testicular androgens in men.

MATERIALS AND METHODS

1. Subjects and training schedules

Subjects for the long-term exercise study were 10 healthy male students (22.3 ± 1.2 yr), who wished to undergo a program of long-term jogging. The subjects underwent a 3-month supervised running training, with mileage increasing to a mean of 48 km/week. Ten subjects (21.4 ± 0.6 yr) were recruited for the studies of short-term endurance training and 10 male (21.7 ±1.2 yr) students for acute endurance exercise in Medical School in Hanyang University and Chungbuk National University. The subjects were mild smokers, and alcohol intake was in the mild to moderate range. All trainees were well educated, in good mental and physical conditions, and understood the meaning of the study objects.

Subjects for the short-term exercise study took to two times heavy physical exercise (8km/h) running, starting on Monday afternoon (day 1) and finished on the next Monday (day 7). Water intake was unlimited in order to exclude the possibility of hormone increases due to the haemoconcentration. There was no organized sleep during the whole training course, but the students had about 4-hours sleep in total between activities. Ten subjects for the acute endurance exercise were recruited among 20 students of short-term training study and ran for 1 hour at full speed.

Weight (kilograms), percent body fat by underwater weighing, and lean body mass by calculation from these variables were measured before and after the 3-month physical endurance training.

2. Blood sampling

Blood was collected daily between 06:00-08:00 h in long-term study and short-term study, using the ice-chilled evacuated 10-mL tubes containing 0.12 mL 0.34 M EDTA K3 and aprotinin (5000 kallikrein inhibitor units). These were stored on 4°C refrigerator. After clotting, serum was separated and frozen immediately to −80°C on dry ice and kept in a deep -freezer until analyzed. Before and after 3-month training in the study of long-term endurance running, after 2, 4, 6 days of short-term training in the second study, and after 30 min, 1, 5 hrs of acute prolonged exercise study, single blood samples were obtained in all subjects. No subject had trained in the 24 h before sampling in short-term and long-term studies.

3. Hormone analyses

The hormones were analyzed by the conventional radioimmunoassay (RIA) kits or by the in house non-isotopic immunoassay (NIA) methods. Intra-assay (within-assay variation, WAV, first) and inter-assay (between-assay variation, BAV, second) coeffici-

ents of variation are given in parentheses. Since 20~25 samples from each study were analyzed in one assay batch and with the same standards, the BAV has relevance in the comparison between the assay batches. Cortisol (WAV, 4.7% ; BAV, 6.4%), dehydroepiandrosterone (DHEA, 5.9% and 11.3 %), testosterone (T, 4.3% and 8.8%), free testosterone (fT, 6.3% and 8.5%), androstenedione (A, 5.6% and 8.7%) and estradiol (E, 7.1% and 8.4%) for the samples from long-term exercise study were analyzed with Coat-a-Count kits, and LH (4.3% and 6.7%), FSH (4.2% and 6.9%), and prolactin (5.9% and 7.6%) were analyzed with the double antibody kits from Diagnostic Products Corp. (DPC; Los Angeles, CA). The samples for the long-term endurance training were analyzed for DHEA sulfate (DHEAS, 5.3% and 7.8%) and for steroid hormone binding globulin (SHBG, 6.3% and 10.7%) with kits from api bioMerieux kits, whereas the samples from short-term and acute studies for DHEAS and DHEA were analyzed with a kit from DPC (4.9% and 11.7%). Cortisol (3.5% and 6.3%), T (4.4% and 5.7%), fT (5.7% and 6.6%), A (5.4% and 7.2%), and E (6.1% and 7.7%) for the samples from short-term and acute exercise studies were analyzed in duplicates with in-house methods in order to confirm the results of the conventional kits (Yoon, 1981; Yoon and Kim 1987; Yoon et al., 2000).

4. Statistics

The results are presented as the mean ± SEM. Analysis of variance (ANOVA) for repeated measures was used, with days as the repeated factor. The students t test was used to identify overall significant alterations.

RESULTS

1. Hormonal changes after long-term endurance exercise

All subjects lost a small amount of body weight (2 – to 5 – kg), mostly fat, although there was a small but significant decrease in lean body mass.

The hormonal changes after endurance training over 3-month jogging are summarized in Table 1. Mean total (TT) and free testosterone (fT) levels were significantly lower, but within the normal range, in the post-training samples than in pre-training ones (Table 1). We found no significant changes in the levels of LH, FSH, and SHBG between in the pre-training and in the post-training samples, suggesting that the HPG axis and hypothalamus-pituitary-adrenal (HPA) axis were not influenced by the long term training. However, the mean serum PRL levels were significantly lower in the single sample after the strenuous endurance training for 3 months. The mean serum cortisol levels were increased over the 3-month training period, although they remained within the normal range.

Table 1. Characteristics of the 10 adult male students before and after long-term endurance training

Variables	Pre-training	Post-training
Age (year)	22.3 ± 1.2	22.3 ± 1.2
Height (cm)	173.4 ± 4.7	173.4 ± 4.7
Weight (kg)	66.5 ± 3.4	61.5 ± 2.1*
Lean body mass (kg)	58.4 ± 1.9	55.6 ± 0.8*
Total testosterone (nmol/L)	37.1 ± 1.4	28.8 ± 2.1*
Free testosterone (pmol/L)	146.5 ± 9.6	118.4 ± 16.3*
SHBG (nmol/L)	30.4 ± 2.7	28.8 ± 2.4
ACTH (pmol/L)	14.8 ± 6.3	15.3 ± 3.2
Cortisol (μmol/L)	497 ± 91	563 ± 61*
Prolactin (ng/ml)	11.9 ± 0.8	8.1 ± 0.4*
LH (mIU/mL)	3.1 ± 0.2	2.9 ± 0.4
FSH (mIU/mL)	8.3 ± 0.5	7.6 ± 0.6

Values are expressed as the mean ± SEM. Variations significant at $P<0.05$

2. Short-term exercise

The hormonal changes after short-term training course are summarized in Table 2. Testosterone decreased abruptly from the level of 33.1 ± 1.7 (pre-training sample) to 1/2 levels of control after two day training and then gradually decreased to 1/6 level on 6 day ($P< 0.001$). Free testosterone (fT) decreased in similar pattern of T from 64.3 on day 2 to 1/5 of pre-training concentration. Estradiol increased significantly and gradually from 115 to a maximum of 167 on day 4, followed by a decrease to 83 on day 6 ($P<0.001$). Androstenedione (9.8 in pre-training) showed a gradual decrease to 1/3 of control at the end of training.

DHEA also decreased abruptly and then gradually from 27.7 to 1/5 of control at the end of the training. DHEAS increased from 8.3 (pre-training) to a plateau of 13.6~14.3 on day 2~4 ($P < 0.001$), but increased to 23.7 levels by the end of training. Cortisol increased from 548 to 772 on day 4 and then gradually decreased to pre-training levels by the end of the training.

Table 2. Hormonal changes in serum of adult male students before and during short-term endurance training

Hormones	Pre-training	2day	4day	6day
ACTH (pmol/L)	6.3±0.4	6.8±0.7	4.8±0.6*	4.1±0.3*
Cortisol (μmol/L)	548±56	659±44*	772±86*	603±48*
Prolactin (ng/L)	10.5±2.7	46.4±7.6*	54.3±8.3*	55.7±6.9*
LH (mIU/mL)	3.3±0.2	2.6±0.1	2.8±0.1	4.5±0.2*
FSH (mIU/mL)	8.9±1.4	6.9±1.4	4.8±0.7*	4.0±0.9*
DHEAS (μmol/L)	8.3±1.4	13.6±1.2*	14.3±1.1*	23.7±0.4*
DHEA (nmol/L)	27.7±4.3	13.2±1.1*	6.4±0.4*	4.5±0.5*
Testosterone (nmol/L)	33.1±1.7	18.8±1.4*	10.7±0.3*	5.1±0.6*
Free testosterone (pmol/L)	141.5±11.4	64.3±10.8*	21.1±2.3*	28.7±1.9*
Estradiol (pmol/L)	115±8	138±9*	167±12*	83±6*
Androstenedione (nmol/L)	9.8±0.4	8.5±0.4	6.3±0.3*	3.3±0.2*

Hormones in 10 male students were determined by the conventional RIA kits or in house methods during short-term training with heavy runnings for 1 week. The control samples were obtained 1 week before the training. Blood samples were collected between 06:00~08:00 h. The results are presented as the mean ± SEM. Variations significant at $P<0.05$ comparing to the values of control levels are indicated by *.

ACTH also gradually but significantly decreased from 6.8 on day 2 to 4.1 on day 6 ($P<0.001$). Prolactin level increased 5 times on two day training and then gradually increased to 56 level at the end of training. LH (mIU/mL, 2nd IRP, human menopausal gonadotropin: hMG) decreased from 3.3 to 2.0 on day 2 and then slightly increased to subnormal levels (4.5) at the end of training. FSH (mIU/mL, 2nd IRP, hMG) decreased gradually from 8.9 in the control to 4.0 on day 6 during the training course.

Based upon these results, we assumed that the HPG and HPA axes are significantly influenced by the short-term endurance training.

3. Acute exercise

All hormones measured in this study increased during 1 h training except T and A, and then decreased to the normal levels. ACTH level at 30 min after exercise increased to three times higher than the level of pre-training (6.3) but recovered to normal range at 5 h (6.4) after acute 1 h prolonged exercise. The three times increased levels of cortisol at 30 min decreased gradually but maintained at supraphysiological level until 5 h after exercise. The gonadotropin levels were not changed during the acute exercise and recovery period. The prolactin level at 30 min after exercise increased to approximately 8 times higher than that of control level (10.5) and then to 2 times after 5 h.

Table 3. Hormonal changes in serum of adult man before and after acute strenuous endurance training

Hormones	Pretraning	Hours after Exercise		
		30min	2hrs	5hrs
ACTH (pmol/L)	6.3±0.4	19.4±0.7*	10.1±0.8*	6.4±0.5
Cortisol (μmol/L)	548±56	1474±119*	1066±134*	876±48*
Prolactin (ng/L)	10.5±2.7	81.4±6.4*	46.1±3.9*	18.3±0.4*
LH (mIU/mL)	2.8 ± 0.3	3.0 ± 0.2	2.7 ± 0.1	2.6 ± 0.2
FSH (mIU/mL)	8.4 ± 1.3	8.1 ± 1.1	7.8 ± 0.6	7.9 ± 1.1
DHEA-S (μmol/L)	8.3±1.4	14.1±1.7*	16.2±1.4*	10.8±0.8*
DHEA (nmol/L)	27.7±1.7	34.1±3.8	29.8±3.4	30.5±4.4
Testosterone (nmol/L)	22.8±1.9	35.9±4.1*	20.4±3.5	18.4±1.4*
Free testosterone (pmol/L)	141.5±11.4	216.3±23*	114.6±9.8*	104.5±7.3*
Estradiol (pmol/L)	115±8	209±16*	148±9*	130.5±6.8*
Androstenedione (nmol/L)	9.8±0.4	9.9±1.2	10.3±1.7	6.4±1.2*

Hormones in 10 male students were determined by the conventional RIAs. The single blood sample was obtained 1 week before the training and at 30 min, 2h, and 5h after training. The results are presented as the mean ± SEM. *Variations significant at $P < 0.05$.

The concentrations of TT and fT were higher than basal levels after exercise but decreased promptly to subnormal levels during recovery. DHEA, DHEAS, and androstenedione increased 1 h after exercise but returned to their normal levels, whereas androstenedione decreased to significantly below basal levels. The present results suggest that the acute strenuous heavy training might cause the stimulation of HPA axis but decreases the productions of androgens.

DISCUSSION

The present study confirmed the previous cross-sectional findings displaying that circulating TT levels, measurement of bioavailable androgen, and serum PRL were reduced with endurance training without change in SHBG or single sample gonadotropin levels (Wheeler et al., 1984, 1991) in Table 1. The mechanism(s) underlying the suppression of the temporal hypodysfunction and their teleological significance were remained to be studied. The present study and the results of Wheeler et al. (1991) could not relate the decline in circulating testosterone to changes in serum cortisol levels as Strauss et al (1985) showed. These results suggest that the decreasing mechanism(s) of circulating testosterone in strenuous long-term endurance training and in runners with low body fat decreases may be different from that of the wrestlers trying to make weight (Ayers et al., 1985; Straus et al., 1985). We found that the individual hormone values generally remained within the normal range (Table 1), and that the degree of change was similar to the previously reported difference between runners training in excess of 64 km/week and non-exercising controls (Wheeler et al., 1984, 1991). The present data showed that HPG and HPA axes were not disturbed by the long-term exercise, suggesting that the reduction of androgen production is not influenced by these axes, but by the temporary hypofunctions of gonads and hyperfunction of adrenal glands.

Although many studies have reported changes in basal pulsatile LH release (MacConnie et al., 1986; McColl et al., 1989), the present study showed the decline in serum testosterone levels without change in measures of LH release. The temporal changes in LH pulsatile release have been described in response to acute exercise in women runners (Cumming et al., 1985), but in highly trained male runners, any significant effect of acute (60-120 min) strenuous running activity on LH pulsatile release could not be found (MacConnie et al., 1986; McColl et al., 1989). Transient suppression of serum testosterone levels by prolonged physical activity has been described in several studies (Cumming et al., 1989), but values returned to baseline within 24 h. The present studies showed that the productions of adrenal and gonadal steroid hormones increased but decreased gradually during 5h after acute training and also during the short-term trainging. This suppression mechanism of circulating androgen levels after abrupt stimulation remains to be cleared.

Our data confirmed the previous results of physiological reduction in serum TT and fT, and prolactin levels and that reduced levels of androgen have been associated with high mileage endurance runner. Previous studies reported that supraphysiological high mileage running has been associated with elevated cortisol levels and impaired response to ACTH, suggesting that the elevated cortisol cause the temporal hypofunction of testis (Barron et al., 1985; Wheeler et al., 1991). Our data also showed that the levels of ACTH significantly increased during the running and then returned to normal range after 5 h after acute training but were decreased during repeated exercise during short-term training, while cortisol levels were increased (Table 2 and 3). However, it is not clear whether this level of cortisol could inhibit the HPG axis or not.

The previous studies reported that plasma testosterone has a biphasic response to exercise, with an increase during the first minutes (Lamb, 1975; Kuoppasalmi, 1980; Schmid et al., 1982), followed by a decrease if the duration of exercise extended to hours or days (Opstad and Aakvaag, 1982, 1983). The present study also showed that there was a decrease in all androgen hormones from testis and adrenal gland during the days of continuous physical exercise (Table 2).

It has been reported that only 10~25% of plasma DHEA, 25~30% of androstenedione, and 10% of 17-hydroxyprogesterone derived from direct testicular secretion and that 80~90% of DHEA and 90~98% of DHEAS are thought to be derived from direct adrenal secretion (Parker, 1989). The present study showed that DHEA decreased during the physical endurance exercise (Table 2). Therefore, we assume that there have to be decreases in both testicular and adrenal androgen secretion during the endurance exercises.

ACTH, LH, and FSH are known to regulate more or less all steps in steroidogenesis (Lieberman et al., 1984; Simpson et al., 1988). The decrease in plasma FSH may explain the decrease in testicular androgens during the short-term severe exercise (Table 2). The decrease in ACTH could explain the decrease in adrenal androgens, but is inconsistent with the increase in cortisol (Table2).

We confirmed the results of de Leo's group, who showed a reduction in the recovery of testosterone levels after an exercise-induced increase. Our results demonstrated that all androgens increased immediately after acute training and then decreased significantly from 1 h to 5 h recovery period (Table 3) but recovered to normal values after 1 day (data not shown). The levels of ACTH, cortisol, prolactin DHEAS, and DHEA also increased after prolonged exercise and returned to their pre-training levels in a subsequent recovery phase. The same results have been reported previously (de Leo et al., 2000). However, the level of ACTH increased immediately after 30 min but reduced to normal range within 1 h.

Based upon the present study and the previous studies, we assume the hypothesis that strenuous acute training causes the temporal hypofunction of testis not by the impaired functions of gonadotropin secretion but by other factor(s). However, the chronic effects of this short-term hypofunction should be elucidated.

REFERENCES

Ayers JWT, Komesu Y, Romain T, Ansbacher RA (1985) Anthropometric hormonal and psychologic correlates of semen quality in endurance trained male athletes. Fertil Steril 43: 917-921.

Barron JL. Noakes TD, Levy W, Smith C, Millar RP (1985) Hypothalamic dysfunction in overtrained athletes. J Clin Endocrinol Metab 60: 803-806.

Carli G, Bonifazi M, Lodi L, Lupo C, Martelli G, Viti A (1992) Chnges in the exercise-induced hromone response to branched chain amino acid administration. Eur J Appl Physiol 64: 272-277.

Constantini NW, Warren MP (1994) Special problems of the female athletes. In: Panush RS, Lane NE, eds. Baillier's Clinical Rheumatology. London, Philadelphia, Sydney, Tokyo, Toronto: Balliere Tindall; 199-219.

Cumming DC (1990) Physical activity and control of the hypothalamic-pituitary-gonadal axis. Semin Repord Endocrinol 8: 15-24.

Cumming DC, Quigley ME, Yen SSC (1983) Acute suppression of circulating testosterone levels by cortisol in man. J Clin Endocrinol Metab 57: 671-673.

Cumming DC, Vickovic MM, Wall SR, Fluker MR, Belcasero AN (1985) The effect of acute exercise on LH pulsatile release in women runners. Am J Obstet Gynecol 153: 482-485.

Cumming DC, Wheeler GW, McColl EM (1989) The effects of exercise on reproductive function in men. Sports Med 7: 1-17.

de Leo V, Marca A, Pasqui L, Zhu B, Morgante G (2000) Effects of human chorionic gonadotropin administration on testicular testosterone secretion during prolonged exercise. Fertil Steril 73: 864-876.

Hackney AC, Sinning WE, Bruot BC (1988) Reproductive hormonal profiles of endurance-trained and untrained males. Med Sci Sports Exerc 20: 60-65.

Highet R (1989) Athletic amenorrhea: An update on aethiology, complications and management. Sports Med 7: 82-108.

Kuoppasalmi K (1980) Plasma testosterone and sex-hormone -binding globulin capacity in physical exercise. Scand J Clin Lab Invest 40: 411-418.

Lamb DR (1975) Androgens and exercise. Med Sci Sports 7: 1-5.

Lieberman S, Greenfield NJ, Wolfson A (1984) A heuristic proposal for understanding steroidogenic processes. Endocr Rev 5: 128-148.

MacConnie SE, Barkan A, Lampman RM, Schork MA, Beitins IZ (1986) Decreased hypothalamic gonadotropin releasing hormone secretion in male marathon runners. N Engl J Med 315: 411-417.

McColl EM, Wheeler GD, Gomes P, Bhambhani Y, Cumming DC (1989) The effects of acute exercise on LH pulsatile release in high mileage male runners. Clin Endocrinol (Oxf.) 31; 617-629.

Opstad PK (1992) Androgenic hormones during prolonged physical stress, sleep, and energy deficiency. J Clin Endocrinol Metab 74: 1176-1183.

Opstad PK, Aakvaag A (1983) The effect of sleep deprivation on the plasma levels of hormones during prolonged physical strain and calorie deficiency. Eur J Appl Physiol 51: 97-107.

Opstad PK, Aakvaag A (1982) Decreased serum levels of oestradiol, testosterone and prolactin during prolonged physical strain and sleep deprivation, and the influence of a high calory diet. Eur J Appl Physiol 49: 343-348.

Parker LN (1989) Adrenal androgens in clinical medicine. New York: Academic Press 615.

Rogol AD, Veldhuis JD, Williams FA, Johnson ML (1984) Pulsatile secretion of gonadotopins and prolactin in male marathon runners. Relation to the endogenous opiate system. J Androl 5: 21-27.

Schmid P, Pusch HH, Wolf W, et al. (1982) Serum FSH, LH, and testosterone in humans after physical exercise. Int J Sports Med 3: 84-89.

Simpson ER, Waterman MR (1988) Regulation of the synthesis of steroidogenic enzymes in adrenal cortical cells by ACTH. Annu Rev Physiol 50: 427-440.

Strauss RH, Lanese RR, Malarkey WB (1985) Weight loss in amateur wrestlers and its effect on testosterone levels. JAMA 254: 3337-3338.

Wheeler GD, Mohan S, Pierce WD, Frank Eoling W, Cumming DC (1991) Endurance training decreases serum testosterone levels in men without change in luteinizing hormone pulsatile release. J Clin Endocrinol Metab 72: 422-425.

Wheeler GD, Wall SR, Belcastro AN, Cummimg DC. (1984) Reduced serum testosterone and prolactin levels in male distance runners. JAMA 252:514-516.

Yoon YD (1981) The hormonal levels of the short luteal phase in Korean women (I): LH, FSH, Estradiol, and Progesterone. J Basic Sci (Hanyang Univ) 1: 154-166.

Yoon YD, Kim SR (1987) Luminescence immunoassays and their applications for dihydrotestosterone and testosterone (I): Establishment of LIA. Kor J Fertil Steril 14: 138-148.

Yoon YD, Chun EH, Lee CJ, Do BR, Lee JY (2000) Ultrasensitive enzymeimmunoassay for testosterone in human saliva. Dev Reprod 4: 115-123.