

Endocrine response to intense interval exercise

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Summary. This investigation provides an insight into the physiological changes produced, and processes operating, during and after a typical interval exercise training regime. The role of interval exercise in the modulation of the plasma concentration of sex hormone binding globulin (SHBG) and the hormones β -oestradiol, testosterone, prolactin and growth hormone was assessed. Eight trained male athletes [mean maximal oxygen uptake ($\dot{V}O_{2\max}$) 64.3 (SD 3.8) ml·kg⁻¹·min⁻¹, mean age 31.5 (SD 4.5) years] undertook an intense interval exercise (treadmill running) protocol to exhaustion. Subjects completed an average of 15.6 × 1-min runs. This interval protocol produced significant increase in the plasma concentration of SHBG and all four hormones (all $P < 0.01$) in the immediate post-test period. The plasma concentration of the hormones increased as indicated: β -oestradiol (45%), testosterone (38%), prolactin (230%), growth hormone (2000%). These hormones have an established capacity to interact with components of many physiological systems and, as such, may provide a mechanism for the changes induced by intense exercise in many of these systems.

Key words: Anaerobic – Growth hormone – Oestradiol – Testosterone – Prolactin – Sex hormone binding globulin

Introduction

The hormone response to exercise has been of interest for many years and has been shown to depend on the intensity, duration and nature of the exercise undertaken (Virus 1985; Galbo 1983). However, despite the abundance of data concerning exercise-induced changes in peripheral hormone concentrations, little effort has been directed toward the study of exercise regimes that are directly relevant to the training of athletes. Particularly lacking have been studies investigating training regimes

requiring a significant anaerobic (“lactic”) energy contribution. One of the most commonly used training regimes involves interval exercise, that is, repeated intense periods of exercise interspersed with only partial recovery periods. Such regimes have resulted in a significant accumulation of lactic acid in the blood and tissues (Telford 1991).

This investigation employs an intense interval exercise protocol modelled on those which have been frequently used by athletes involved in swimming, middle/long distance running and football (Telford 1991). The effects of such a protocol on peripheral hormone concentrations are largely unknown. Many of the previous investigations into the effects of interval exercise on the endocrine system have unfortunately, employed protocols of little relevance to the training of athletes.

Previous research has indicated that moderate aerobic exercise, above a threshold intensity, is capable of elevating plasma concentrations of most hormones (Virus 1985). Given the repetitively stressful nature of intense interval exercise, the protocol employed in this study was expected to lead to both quantitative and qualitative differences in the hormonal milieu from that produced by other forms of exercise, e.g. aerobic or a single supra-maximal effort.

These hormones are derived from different endocrine organs. Growth hormone and prolactin are anterior pituitary hormones, whose release is regulated by specific hypothalamic derived releasing and release-inhibiting factors. In men, testosterone is produced principally by the testes, with minor amounts by the adrenal medulla. Minor amounts of β -oestradiol, in men, are produced by adreno-cortical and testicular pathways associated with testosterone synthesis (Ganong 1983). A proportion of both total β -oestradiol and total testosterone present in plasma are transported by sex hormone binding globulin (SHBG) (Anderson 1974). Knowledge of the plasma concentrations of these sex hormones and SHBG provides an index of the amount of unbound hormone readily available for target tissues.

The four hormones under investigation have a range of physiological effects including modulation of lipo-

protein profiles (β -oestradiol), protein anabolism (testosterone and growth hormone) and breast milk secretion in women (prolactin; Ganong 1983). In addition each has an ability to modulate elements of the immune system (Gala 1991; Grossman 1984; Jansson 1991; Kelley 1989).

However, while exercise-induced modulation of many physiological systems may be mediated by the action of these hormones, the aim of this study was to establish the effects of an interval exercise regime on the peripheral blood concentrations of the hormones β -oestradiol, testosterone, prolactin and growth hormone.

Methods

Subjects. Eight trained men, mean age 31.5 (SD 4.5) years were employed as exercise subjects. All were, and had been for at least 1 year, engaged in a regular training programme. All were free of symptoms of infection and none were taking medication or drugs of any kind. All fasted overnight and none had exercised within the previous 24 h.

Exercise tests. Subjects were familiarized with test procedures for both the determination of maximal oxygen uptake ($\dot{V}O_{2\max}$) and interval running tests and all participants signed a consent form that detailed the scope of the study and its attendant risks. Testing commenced between 0800 and 0900 hours. Procedures were approved by the Ethics Committee of the Australian Institute of Sport.

Maximal oxygen uptake test (test 1). All subjects underwent a progressive running test employing increases in speed and gradient to determine their $\dot{V}O_{2\max}$. The initial treadmill speed was 11 km·h⁻¹ with an increase of 1 km·h⁻¹ each minute for the first 7 min. At this stage the treadmill gradient was increased at a rate of 2% per minute. All subjects exercised until subjective estimation of exhaustion brought about the voluntary conclusion of the test. The respiratory analysis followed procedures described previously (Hahn et al. 1988). Criteria for attainment of $\dot{V}O_{2\max}$ were: a "plateau" in oxygen uptake (an increment of less than 0.15 l·min⁻¹, heart rate equal to predicted maximum or a respiratory exchange ratio (R) in excess of 1.10.

Interval running test (test 2). This test was performed at least 1 week following test 1. All subjects underwent a standard warm-up procedure involving 5 min running at both 7 km·h⁻¹ and 11 km·h⁻¹ and 3 min at 15 km·h⁻¹. All warm-up procedures were performed at 0% treadmill gradient. Subjects then performed the interval running test (IRT), i.e. alternating 1-min periods of treadmill running and active (walk) recovery. Treadmill speed and gradient for IRT were selected for the individual using the speed and gradient at which $\dot{V}O_{2\max}$ was achieved. Subjects were informed of the elapsed time every 15 s and all received encouragement during the course of each exercise period. Subjects were asked to perform as many exercise periods as possible and continued until they could no longer maintain the required speed.

Blood collection. Blood samples, obtained with subjects in a supine position, were collected by venepuncture of an antecubital vein. Six samples were collected; before exercise (after the subject had been sitting quietly for 20 min), post warm-up, post-test, 1, 6, 24 h post-test. Samples were placed immediately into ethylene diamine tetra-acetic acid (EDTA) vacutainers (Becton Dickinson, Mountain View, Calif., USA) prior to centrifugation and separation at 4°C. Samples were stored at -20°C before analysis.

Full blood counts. A Coulter S550 cell counter (Coulter Electronics, Hialeah, Fla., USA) was employed to obtain a full blood count, including packed cell volume, for each sample.

Whole blood lactate. All blood samples were analysed for whole blood L-lactate with a YSI Model 23L L-lactate analyser (Yellow Springs Instrument Company, Ohio, USA).

Hormone assays. All hormone assays (for total β -oestradiol, growth hormone, total testosterone, prolactin) were performed using commercially available radio-immunoassay (RIA) kits (Biodata/Serono, Australian Diagnostics, Sydney). The SHBG was also assayed by RIA (Farnos, Australian Laboratory Services, Sydney).

Statistics. Data were analysed by a repeated measures one-way ANOVA and Fisher's PLSD was used for posthoc comparisons. PLSD = Protected Least Significant Difference. Significance was indicated at the $P < 0.05$ level.

Results

Physical and physiological characteristics of subjects

Table 1 gives details of the subjects employed in this study. The intensity of the interval exercise protocol is illustrated by the high post-test whole blood lactate concentration (7.6 mmol·l⁻¹), such an accumulation of lactic acid indicating considerable involvement of anaerobic energy pathways.

Variation of hormone concentrations with exercise

The resting concentrations of all four hormones in all subjects were within the appropriate reference ranges (as specified by the manufacturers of the RIA kits employed).

Plasma β -oestradiol concentrations were elevated significantly postwarm-up ($P < 0.05$), immediately after (45% increase), and at 1 (38%), 6 (26%) and 24 (25%) h after the exercise test (all $P < 0.01$; Table 2). Evidence of a haemoconcentration occurred only at the time of the immediate post-test sample where, according to the estimation outlined by Van Beaumont (1972), the plasma volume decreased by approximately 10%. At post-test sampling times plasma volume was estimated to undergo either a minor reduction (-2.0% at 1 h post-test) or was increased slightly in relation to resting values (+3.0% and +0.5% at 6 and 24 h post-test, respectively; data not shown). The SHBG concentrations differed significantly ($P < 0.01$) from rest only immediately post-test when it increased by approximately 8%, which might well be explained by the estimated haemoconcentration (Table 2).

Testosterone concentration increased significantly immediately after completion of the exercise test (38% elevation; $P < 0.01$), but had decreased significantly by 6 h post-test (16% decrease; $P < 0.05$).

An elevation in prolactin occurred immediately post-test when the plasma concentration increased 230% ($P < 0.01$) but at no other time did values differ from those obtained at rest, prior to exercise.

Table 1. Physical and physiological characteristics of the subjects

Maximal oxygen uptake ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)		Age (years)		Mass (kg)		Intervals completed		[la^-] _b ($\text{mmol} \cdot \text{l}^{-1}$) post-test	
mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
64.3	3.8	31.5	4.5	74.0	8.8	15.6	6.6	7.6	1.6

[la^-]_b, blood lactate concentration

Table 2. Hormone concentrations

	Sampling time						
	Rest	Post-warmup	Post-test	1-h post	6-h post	24-h post	
β -oestradiol ($\text{pg} \cdot \text{ml}^{-1}$)							
mean	34.2	40.5*	49.8**	47.2**	43.2**	42.8**	
SD	7.2	6.7	7.1	6.3	5.3	10.0	
Testosterone ($\text{ng} \cdot \text{ml}^{-1}$)							
mean	7.5	8.6	10.4**	7.2	6.3*	7.4	
SD	2.5	2.8	3.9	2.6	2.3	3.1	
Prolactin ($\text{ng} \cdot \text{ml}^{-1}$)							
mean	9.7	10.1	32.6**	11.2	8.4	9.9	
SD	2.6	2.0	15.1	2.7	2.3	3.4	
Growth hormone ($\text{ng} \cdot \text{ml}^{-1}$)							
mean	1.3	6.7*	26.8**	8.7**	0.9	1.0	
SD	0.7	6.7	6.2	9.5	0.2	0.1	
Sex hormone binding globulin ($\text{nmol} \cdot \text{l}^{-1}$)							
mean	30.7	32.3	33.1**	32.0	30.9	31.5	
SD	9.2	8.2	8.8	8.2	8.2	9.3	

Significantly different from rest, * $P < 0.05$, ** $P < 0.01$

Growth hormone concentrations were elevated significantly both immediately after exercise (approximately 20 times resting value), and at 1 h later (six times; $P < 0.01$). At no other times were they significantly different from the resting values.

Discussion

This study has demonstrated that interval exercise, involving considerable anaerobic energy production, is associated with significant elevation of plasma SHBG, β -oestradiol, testosterone, prolactin and growth hormone concentrations. The limited exercise-induced modulation of SHBG concentration (apart from that due to haemoconcentration) indicates that the interval exercise-induced changes in total β -oestradiol and total testosterone concentrations are largely due to increases in the concentration of the specific unbound hormones.

Despite this protocol producing increases in the plasma concentration of all four hormones, the kinetics of these processes differed among hormones. The significant increase in β -oestradiol and growth hormone following warm-up indicates that plasma concentrations of these hormones are elevated at relatively low exercise intensities. The absence of significant increases in the plas-

ma concentration of prolactin and testosterone would suggest, given constant metabolic clearance rates (MCR), that the activation threshold concentrations for these hormones were not exceeded during the warm-up procedure.

Data are limited on the effect of exercise on male oestradiol concentrations. Nevertheless, Adlercreutz et al. (1976) have reported a 160% elevation of oestradiol in men following running 3×300 m, while Brown et al. (1980), in a study employing 2 h of exercise at 55% $\dot{V}O_{2\text{max}}$, have reported a postexercise elevation of plasma oestradiol. Thus, there appears to be both exercise duration and intensity dependent components to exercise induced elevation of plasma β -oestradiol concentrations.

The sustained elevation in β -oestradiol, apparent at 24 h postexercise with this interval training protocol, would indicate an alteration of the dynamics of β -oestradiol production and/or elimination. A postexercise decrease observed in the MCR for β -oestradiol (Keizer et al. 1980) implies that increased secretion may not be the sole cause of the sustained elevation of plasma β -oestradiol. The mechanism of this altered MCR has not been fully established but a decrease in hepatic blood flow (Galbo 1983) would appear to be only one of a number of components capable of contributing to this

phenomenon (Keizer et al. 1980). The sustained elevation of oestradiol may have beneficial effects in relation to the incidence of coronary heart disease, being associated with favourable changes in high density lipoproteins (Godsland et al. 1987).

The increase in plasma testosterone concentration observed immediately post-test does not agree with the findings of Kuoppasalmi et al. (1980), who have reported no increase in plasma testosterone following repeated 20–40 m runs at maximal speed. However, the 38% increase in testosterone concentration observed with this protocol immediately post-test does agree with those seen by Kindermann et al. (1982), who have reported an increase of 14% in plasma testosterone concentration following a maximal anaerobic running test to exhaustion. Despite the differences in subject characteristics and exercise conditions relating to these studies, this discrepancy would support a role for exercise duration in modulating plasma testosterone concentrations. These findings would also appear to indicate an interaction of exercise intensity and duration in elevation of plasma testosterone concentrations, and may be related to the anaerobic energy contribution. The protocol employed by Kuoppasalmi et al. (1980) would appear to have been predominantly "alactic" whilst that of Kindermann et al. (1982) and the current authors was largely "lactic" in nature.

Our findings conflict with those of both Wilkerson et al. (1980) and Galbo et al. (1977). Wilkerson et al. (1980) have reported no change in plasma testosterone concentrations (above that due to haemoconcentration) following 20 min running at various exercise intensities below that corresponding to $\dot{V}O_{2\max}$. Galbo et al. (1977) have proposed that increases in plasma testosterone concentrations following maximal exercise can be explained by changes in plasma volume. Our findings do not support this hypothesis and would indicate that both the intensity, and nature of the exercise undertaken (i.e. intermittent or continuous) are important factors affecting plasma testosterone concentrations.

The preceding point clearly illustrates the need to examine very carefully the effects of interval exercise protocols on physiological parameters and not to assume that such exercise will induce similar changes to those produced by other protocols, as evidenced by the work of Karagiorgos et al. (1979). The relatively high postexercise whole blood lactate concentration ($7.6 \text{ mmol} \cdot \text{l}^{-1}$), probably sustained throughout much of the exercise protocol due to the repeated high intensity efforts (Telford 1991), is indicative of the anaerobic energy contribution required during this form of exercise. Moreover Karagiorgos et al. (1979), in a comparison of intermittent and continuous physical activity, have noted that the former resulted in significantly higher postexercise blood lactate concentrations. These findings have served to illustrate the unique physiological demands associated with intense interval exercise.

The post-exercise elevation in plasma testosterone concentrations would appear to have been mediated by a reduction in MCR. Sutton et al. (1976a) have shown that exercise-induced increases of up to 50% (above

resting levels) in testosterone could be explained by a reduction in MCR. A role for luteinizing hormone (LH) in this increase would appear to be unlikely given the repeated finding of either no change or a reduction in plasma LH concentrations following a range of exercise protocols (Jurkowski et al. 1978; Sutton et al. 1973; Galbo 1983). In particular, Elias et al. (1991) have found a significant postexercise decrease in plasma LH concentration which reached its nadir 90 min post-test. Kuoppasalmi et al. (1980) have also reported a delayed decrease (30 mins post-test) in plasma LH concentrations following aerobic exercise. Such changes may play a role in the decreased testosterone concentration reported in this study at 6 h post-test.

The limited time-course of the postexercise elevation of testosterone concentration would suggest that the anabolic activities of testosterone appear to be of limited importance in relation to this interval exercise protocol. Greater importance could be attached to the significant decrease in testosterone concentration at 6 h postexercise should this change be sustained for an extended period.

The 20-fold increase observed in plasma growth hormone concentration (to approximately $30 \text{ ng} \cdot \text{ml}^{-1}$) corresponds closely to values reported by Kindermann et al. (1982) following aerobic exercise. Farrell et al. (1983) have reported increases in peripheral growth hormone concentrations following exercise at 65% (6-fold increase), 80% (20-fold) and 100% (11-fold) $\dot{V}O_{2\max}$. Bunt et al. (1986) have reported an increase to only $16 \text{ ng} \cdot \text{ml}^{-1}$ following 60 min running at 60% $\dot{V}O_{2\max}$. Viru (1985), in an extensive review of exercise induced modulation of peripheral hormone concentrations, has concluded that growth hormone concentration displays an activation threshold in excess of the 50% $\dot{V}O_{2\max}$ level. The growth hormone concentrations observed in this study, immediately postexercise, while being in excess of those produced by moderate aerobic exercise, at approximately 60% $\dot{V}O_{2\max}$ (Bunt et al. 1986), are in agreement with those obtained by relatively intense exercise, at 80%–100% $\dot{V}O_{2\max}$ (Farrell et al. 1983). These findings support an exercise intensity dependent modulation of plasma growth hormone concentration, although the work of Kindermann et al. (1982) may indicate a duration effect as well.

The similar changes in plasma growth hormone concentrations produced by aerobic (Farrell et al. 1983; Kindermann et al. 1982) and interval exercise (this study) would imply that postexercise plasma growth hormone concentrations may be influenced by either the intensity or duration of exercise rather than by the nature of the exercise undertaken.

As indicated in Table 2, plasma growth hormone concentrations exhibited a sustained (1 h) post-exercise elevation. Given that the half-life of growth hormone has been reported as being approximately 16 min (Lassare et al. 1974) and between 17 and 45 min (Shephard and Sidney 1975), the significantly elevated concentration at the 1-h post-test is consistent with normal plasma clearance rates. However, other factors such as low blood glucose concentration (Ganong 1983), which was

not measured in our study, or elevated lactic acid concentration/reduced pH may be involved. However Sutton et al. (1976b) have reported an independence of plasma lactate and growth hormone concentrations. This may indicate that exercise-induced increases in growth hormone and prolactin concentrations, relate to alteration of either hypothalamic regulation or plasma clearance. Measurement of specific releasing or inhibitory factors during and after the exercise period may better define the mechanism of these exercise-induced changes.

The physiological role of prolactin in men is unclear. However, the effect of this protocol on the kinetics of plasma prolactin concentration would appear to differ from those displayed by growth hormone. Despite the sequence homology shared by growth hormone and prolactin (Ganong 1983) and similar immune system activities (Gala 1991), exercise-induced elevation of prolactin concentration would appear to have a threshold higher than that of the growth hormone, given that the former was not elevated by the warm-up procedure. The increase in growth hormone concentration is more sustained, still being significantly elevated at 1 h post-test.

Prolactin has frequently been reported to increase following acute exercise, a change apparently related to the intensity of exercise (Sowers et al. 1977; Brisson et al. 1981; Galbo 1983). Intense interval exercise clearly provides a potent stimulus for increase in peripheral prolactin concentration, this protocol producing an increase of 230%. Prolactin secretion from the anterior pituitary has been found to be inhibited by dopamine (DA) (Ben-Jonathan 1985). An exercise-induced decrease in peripheral plasma DA concentration reported by Gray et al. (unpublished observations) may reflect a role for DA in elevation of prolactin concentrations in the post-exercise period.

Brisson et al. (1986) have implicated thermal stress as an agent mediating exercise-induced elevations in plasma prolactin concentrations. The relative contributions of thermal, psychological and physical stresses provided by this form of exercise, to the elevation of plasma prolactin concentrations, remain to be determined. The physiological effects, and in particular those relating to the immune system, of the hormonal changes induced by this commonly used form of extended interval exercise await further investigation.

In conclusion, this study has demonstrated that interval training, involving considerable anaerobic energy production, is a stimulus to the elevation of peripheral concentrations of β -oestradiol, testosterone, prolactin and growth hormone.

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References

- Aldercreutz H, Harkonen M, Kuoppasalmi K, Kosunen K, Naveri H, Rehnunen S (1976) Physical activity and hormones. *Adv Cardiol* 18:144-157
- Anderson DC (1974) Sex hormone binding globulin. *Clin Endocrinol* 3:69-96
- Ben-Jonathan N (1985) Dopamine: a PRL inhibiting hormone. *Endocr Rev* 6:564-589
- Brisson GR, Ledoux M, Peronnet F, Dulac S, Decarufel D, Volle MA, Rainville J, Audet A (1981) Prolactinemia in exercising male athletes. *Horm Res* 15:218-223
- Brisson GR, Peronnet F, Ledoux M, Pellerin-Massicotte J, Matton P, Garceau F, Boisvert P (1986) Temperature-induced hyperprolactinemia during exercise. *Horm Metab Res* 18:283-284
- Brown WJ, McGinley R, Vining RF, White S (1980) The effect of endurance exercise on the serum concentrations of estradiol and testosterone in trained and untrained men and women. *Proc Aust Physiol Pharmacol Soc* 11:131
- Bunt JC, Boileau RA, Bahr JM, Nelson RA (1986) Sex and training differences in human growth hormone levels during prolonged exercise. *J Appl Physiol* 61:1796-1801
- Elias AN, Wilson AF, Pandian MR, Chune G, Utsumi A, Kayaleh R, Stone SC (1991) Corticotropin releasing hormone and gonadotropin secretion in physically active males after acute exercise. *Eur J Appl Physiol* 62:171-174
- Farrell PA, Garthwaite TL, Gustafson AB (1983) Plasma adrenocortical and cortisol responses to submaximal and exhaustive exercise. *J Appl Physiol* 55:1441-1444
- Gala RD (1991) Prolactin and growth hormone in the regulation of the immune system. *Proc Soc Exp Biol Med* 198:513-527
- Galbo H (1983) Hormonal and metabolic adaptations to exercise. Thieme, New York
- Galbo H, Hummer L, Petersen IB, Christensen NJ, Bie N (1977) Thyroid and testicular hormone responses to graded and prolonged exercise in man. *Eur J Appl Physiol* 36:101-106
- Ganong WF (1983) Review of medical physiology (11th edn.). Lange, Los Altos, pp 330, 336-369
- Godsland IF, Wynn V, Crook D, Miller NE (1987) Sex, plasma lipoproteins and atherosclerosis: prevailing assumptions and outstanding questions. *Am Heart J* 114:1467-1503
- Grossmann CJ (1984) Regulation of the immune system by sex steroids. *Endocr Rev* 5:435-455
- Hahn AG, Pang PM, Tumilty DMcA, Telford RD (1988) General and specific aerobic power of elite marathon kayakers and canoeists. *Excel* 5:14-19
- Jansson G (1991) Oestrogen-induced enhancement of myeloperoxidase activity in human polymorphonuclear leukocytes - a possible cause of oxidative stress in inflammatory cells. *Free Radic Res Comm* 14:195-208
- Jurkowski JE, Jones NL, Walker WC, Younglai EV, Sutton JR (1978) Ovarian hormone responses to exercise. *J Appl Physiol* 44:109-114
- Karagiorgos A, Garcia J, Brooks GA (1979) Growth hormone response to continuous and intermittent exercise. *Med Sci Sports* 11:302-307
- Kelley KW (1989) Growth hormone, lymphocytes and macrophages. *Biochem Pharmacol* 38:705-713
- Keizer HA, Poortmans JR, Bunnik GS (1980) Influence of physical exercise on sex-hormone metabolism. *J Appl Physiol* 48:109-114
- Kindermann W, Schnabel A, Schmidt WM, Biro G, Cassens J, Weber F (1982) Catecholamine, growth hormone cortisol, insulin and sex hormones in anaerobic and aerobic exercise. *Eur J Appl Physiol* 49:389-399
- Kuoppasalmi K, Naveri H, Harkonen M, Aldercreutz A (1980) Plasma cortisol, androstenedione, testosterone, and luteinizing hormone in running exercise of different intensities. *Scand J Clin Invest* 40:403-409
- Lassare C, Girard F, Durand J, Raynaud J (1974) Kinetics of hu-

- man growth hormone during submaximal exercise. *J Appl Physiol* 37:826-830
- Shephard RJ, Sidney KH (1975) Effects of physical exercise on plasma growth hormone and cortisol levels in human subjects. In: Willmore JH, Keogh JF (eds) *Exercise and sport science reviews*. Academic Press, New York, pp 1-30
- Sowers JR, Raj RR, Hershman JM, Carlson HE, McCalleum RW (1977) The effect of stressful diagnostic studies and surgery on anterior pituitary hormone release in man. *Acta Endocrinol* 86:25-32
- Sutton JR, Coleman MJ, Casey J, Lazarus L (1973) Androgen responses during physical exercise. *BMJ*:520-522
- Sutton JR, Coleman MJ, Casey JH (1976a) Testosterone production rate during exercise. *International Congress of Physical Activity Science* (abstract) Quebec, p: 72. Cited in: Jurkowski JE, Jones NL, Walker WC, Younglai EV, Sutton JR (1978) Ovarian hormonal responses to exercise. *J Appl Physiol* 44:109-114
- Sutton JR, Jones NL, Toews CJ (1976b) Growth hormone secretion in acid-base alterations at rest and during exercise. *Clin Sci Mol Med* 50:241-247
- Telford RD (1991) Endurance training. In: Pyke FS (ed) *Better coaching: advanced coaches manual*. Australian Coaching Council, Canberra, pp 125-134
- Van Beaumont W (1972) Evaluation of haemoconcentration from haematocrit measurements. *J Appl Physiol* 32:712-713
- Viru A (1985) *Hormones in muscular activity*. CRC Press, Boca Raton
- Wilkerson JE, Horvath SM, Gutin B (1980) Plasma testosterone during treadmill exercise. *J Appl Physiol* 49:249-253