



Timo Vuorimaa

Neuromuscular, Hormonal and Oxidative Stress Responses to Endurance Running Exercises in Well-Trained Runners





ABSTRACT

Vuorimaa, Timo

Neuromuscular, hormonal and oxidative stress responses to endurance running exercises in well-trained runners

Jyväskylä: University of Jyväskylä, 2007, 94 p.

(Studies in Physical Education and Health,

ISSN 0356-1070; 121)

ISBN 978-951-39-2759-2 (PDF), 978-951-39-2740-0 (nid.)

Diss.

The aim of this study was to investigate to which type of running exercises well trained endurance runners respond more favourably with regard to acute neuromuscular and endocrinological responses and exercise-induced oxidative stress. It was hypothesized that these responses depend on the type of running exercise and relate to the physical characteristics. Altogether nine different running exercises and a long walking exercise were performed by a total of 88 male athletes. An intermittent exercise consisting of progressive 20 s runs decreased vertical jump performance in middle distance runners (MID), but improved it in marathon runners (MAR). An 18 - 24 min exhaustive test run, a 40 min intermittent run (IR) and a 40 min tempo run (TR), led to significant acute improvements in the vertical jump and half squat performance, simultaneously with a decrease in activation of the knee extensor muscles in well-trained endurance runners. The TR - induced improvement in half squat power correlated positively with the velocity associated with maximal oxygen uptake (VO_{2max}) while the corresponding correlation in the case of IR was negative. IR resulted in a higher acute serum testosterone response and TR in a higher serum cortisol response in MID compared to MAR. The TR - induced increase in the molar ratio of serum testosterone to cortisol was the greater the higher the runners' VO_{2max} . Serum antioxidant capacity was shown to increase after three different endurance exercises of long duration in trained endurance athletes. An unexpected acute decrease in the moderately oxidized LDL was found after a low intensity 6h walking exercise but not after a 31-km intensive run or after a competitive marathon run. On the basis of the present results, it is likely that after intensive running the use of a different coordination strategy counteracts strength loss and even improves power in extension exercises performed vertically with both legs in well-trained endurance runners. It is also likely, that marathon runners respond more favourably to intensive continuous type of running with regard to acute leg extension power and hormonal changes while middle distance runners respond better to intermittent type of running. The present findings further suggest that, in well-trained endurance athletes, an acute exercise of long duration and low intensity may not increase oxidized LDL-cholesterol and thus not cause oxidative stress.

Key words: Endurance running exercises, well-trained runners, testosterone, cortisol, muscle power, muscle activation, oxidative stress, antioxidants

Author's address Timo Vuorimaa
Sport Institute of Finland
Kaskelantie 10
19120 Vierumäki, Finland

Supervisors Professor Keijo Häkkinen
Department of Biology of Physical Activity
University of Jyväskylä, Jyväskylä, Finland

Professor Tommi Vasankari
Department of Health and Exercise &
Paavo Nurmi Center
University of Turku, Turku, Finland

Reviewers Professor Véronique Billat
Department of Applied Sport and Exercise Science
Université d'Evry Val d'Essonne, Paris

Professor Timothy D. Noakes
Department of Human Biology
Sports Science Institute of Africa

Opponent Director Esteban Gorostiaga, MD, PhD
Studies, Research and Sports Medicine Center
Government of Navarra, Pamplona, Spain

ACKNOWLEDGEMENTS

The work described in this dissertation was carried out by the author in the Department of Biology of Physical Activity at the University of Jyväskylä, Finland, in co-operation with the Department of Health and Exercise & Paavo Nurmi Center at the University of Turku, Finland. I would like to acknowledge all the people I have had a pleasure to collaborate with and who in different ways made this work possible.

First, my deepest gratitude is directed to my supervisors Professor Keijo Häkkinen, Ph.D., and Professor Tommi Vasankari, M.D., for providing me opportunity for doctoral studies. During all stages of the study they gave me great advice, criticism and support. I also express my warm thanks to Professor Heikki Rusko, Ph.D., for supporting and supervising me, especially during the early stages of this study.

I am grateful to the referees of my thesis, Professors Veronique Billat and Timothy D. Noakes, for their thorough review and valuable comments on the manuscript of this thesis.

I wish to address my sincere thanks to co-authors Markku Ahotupa, Kerttu Irjala, Pasi Kurkilahti, Urho Kujala, Tuula Vasankari, Rami Virlander and Pekka Vähäsöyrinki, for their contribution during the different stages of this work.

I would like to express my gratitude to all the colleagues, as well as laboratory and technical staff who work at the Sport Institute of Finland, Department of Clinical Chemistry at the University of Turku and at the Sports Medical Research Unit of the Paavo Nurmi Center, Turku, for fruitful collaboration. I owe my special thanks to Mrs. Maritsa Ilkka and Mr. Harri Virtanen for their contributions in helping the data collection.

I owe my special thanks to all the subjects who volunteered to participate in this study. Without their "endurance" and activity this work could not have been possible.

Finally, I owe my warmest gratitude to the persons closest to me, my wife Hanna and our daughters Anna, Heta and Helmi, for their "endurance", understanding and love during my studies over the years. I owe also my deepest thanks to my parents Veikko and Siiri, who have supported me by all possible ways during the years of my studies and who really showed "endurance" when waiting this day.

This study received financial support from Finnish Central Sport Federation, Finnish Amateur Athletic Association, Finnish Olympic Committee, the Ministry of Education, Finland, the National Technology Agency of Finland and the Juho Vainio Foundation, Finland.

Heinola, 04.01.2007

Timo Vuorimaa

LIST OF ORIGINAL ARTICLES

The present study is based on the results presented in the following papers, which will be referred to by their Roman numerals:

I. Vuorimaa, T., Häkkinen, K., Vähäsöyrinki, P., Rusko, H. (1996) Comparison of three maximal anaerobic running test protocols in marathon runners, middle distance runners and sprinters. *Int. J. Sports Med.* 17 (suppl. 2):S109-S113.

II. Vuorimaa, T., Vasankari, T., and Rusko, H. (2000) Comparison of physiological strain and muscular performance of athletes during two intermittent running exercises at the velocity associated with VO_{2max} . *Int. J. Sports Med.* 21:96-101.

III. Vuorimaa T. Virlander R. Kurkilahti P. Vasankari T. Häkkinen K. (2005) Acute Changes in muscle activation and leg extension performance after different running exercises in elite long distance runners. *Eur. J. Appl. Physiol.* 96:282-291.

IV. Vuorimaa T., Ahotupa M., Häkkinen K., Vasankari T. Hormonal response to continuous and intermittent running. (Submitted for publication)

V. Vasankari TJ., Kujala UM., Vasankari TM., Vuorimaa T., Ahotupa M. (1997) Effects of acute prolonged exercise on serum and LDL oxidation and antioxidant defences. *Free Rad. Biol. Med.* 22:509-13.

VI. Vuorimaa, T., Ahotupa, M., Irjala, K., Vasankari, T. (2005) Acute Prolonged exercise reduces moderately oxidized LDL in healthy men. *Int. J. Sport Med.* 26:420-425.

ABBREVIATIONS AND DEFINITIONS

AOD	accumulated oxygen deficit
ATP	adenosine triphosphate
CMJ	counter movement jump
CMJ _{max}	maximal counter movement jump
EMG	electromyography
IR	an intermittent run
IR ₆₀	an intermittent exercise consisted of 14 bouts of 60-second runs at vVO_{2max} with 60-seconds rest between the runs.
IR ₁₂₀	an intermittent exercise consisted of 7 bouts of 120-second runs at vVO_{2max} with 120-seconds rest between the runs.
LDL	low-density lipoproteins
MAR	marathon runners
MART	maximal anaerobic running test
MART1	a modification of MART where n sets of 1 x 20 s runs with 100 s recovery were performed progressively until exhaustion
MART3	a modification of MART where n sets of 3 x 20 s runs with 100 s recovery were performed progressively until exhaustion
MART5	a modification of MART where n sets of 5 x 20 s runs with 100 s recovery were performed progressively until exhaustion
MID	middle distance runners
MR	VO_{2max} test run
P	mechanical power during a dynamic exercise and expressed in watts (W)
SD	standard deviation
TR	a tempo run = an intensive constant velocity run
TRAP	total peroxy radical trapping antioxidant potential
VO_{2max}	maximal oxygen uptake
vVO_{2max}	velocity associated with maximal oxygen uptake (the lowest velocity at which oxygen uptake reaches the highest individual level)

CONTENTS

ABSTRACT

ACKNOWLEDGEMENTS

LIST OF ORIGINAL ARTICLES

ABBREVIATIONS AND DEFINITIONS

CONTENTS

1	INTRODUCTION	13
2	REVIEW OF THE LITERATURE.....	16
2.1	Energy delivery in endurance running	16
2.1.1	Energy delivery in middle and long distances is based on oxygen-dependent metabolism.....	16
2.1.2	VO _{2max} and endurance running performance	17
2.1.3	Best runners are economical.....	18
2.2	Neuromuscular characteristics and muscle power in endurance running.....	19
2.2.1	Muscle activation and muscle power	19
2.2.2	Muscle structure.....	20
2.2.3	Running mechanics	21
2.2.4	Development of muscle fatigue.....	21
2.3	Definition and characteristics of endurance running exercises	22
2.3.1	Exercise models.....	22
2.3.2	Continuous versus intermittent exercises	23
2.3.3	Modifications of intermittent exercises	24
2.4	Muscle activation and muscle performance responses to endurance running exercises	25
2.4.1	Exercise-induced strength loss	25
2.4.2	Counteracting exercise-induced strength loss.....	25
2.5	Effects of physical exercise on adrenal and testicular functions.....	26
2.5.1	Acute adrenal and testicular responses to different type of endurance running exercises	26
2.5.2	Individual variation in adrenal and testicular responses	28
2.6	Exercise induced acute oxidative stress and antioxidant defence.....	28
2.6.1	Free radicals and oxidative damage	28
2.6.2	Methods of measuring exercise-induced oxidative stress and antioxidant defence	29
2.6.3	Oxidized LDL as an indicator of exercise-induced oxidative stress	29
3	THE PURPOSE OF THE STUDY	31
4	MATERIAL AND METHODS	33

4.1	Subjects.....	33
4.2	Study designs	34
4.3	Measurements and analyses	39
4.3.1	Maximal aerobic power test (I, II, III, IV)	39
4.3.2	Maximal anaerobic power test (I).....	39
4.3.3	Determining accumulated oxygen deficit (II).....	40
4.3.4	Maximal vertical jump tests (I, II, III)	40
4.3.5	Mechanical power and EMG in half squats (III)	40
4.3.6	Serum hormone analyses (IV).....	42
4.3.7	Maximal and submaximal 1000m running performance (VI).....	42
4.3.8	Lipid and lipoprotein analyses (V, VI)	42
4.4	Statistical methods.....	43
5	RESULTS	44
5.1	Blood lactate accumulation and oxygen uptake in endurance running exercises (I, II, III, IV, V)	44
5.1.1	Blood lactate responses to intermittent and continuous running (I, II, III, IV, V).....	44
5.1.2	VO ₂ responses to intermittent running at the velocity associated with VO _{2max} (II).....	46
5.2	Effects of maximal and submaximal running exercises on muscle power and muscle activation (I, II, III).....	47
5.2.1	Exercise-induced acute changes in maximal vertical jumping performance (I, II, III).....	47
5.2.2	Effects of intermittent and continuous runs on muscle activation and mechanical power in half squats (III).....	48
5.2.3	Maximal anaerobic running power in different intermittent running test modifications (I).....	51
5.2.4	Acute effects of 6 h low intensity exercising on submaximal and maximal 1000 m running (VI).....	52
5.3	Acute serum hormone responses to intermittent and continuous running exercises (IV)	52
5.3.1	Differences in cortisol and testosterone responses between middle distance and marathon runners (IV).....	52
5.3.2	Time course in exercise-induced serum cortisol and testosterone changes (IV)	53
5.4	Lipid and lipoprotein responses to prolonged exercise (V, VI).....	54
6	DISCUSSION	56
6.1	Energy release in intermittent running exercises.....	56
6.2	Muscle power and muscle activation responses to intermittent and continuous running	58
6.3	Acute effects of running exercises on maximal vertical jumping.....	60
6.4	Factors relating with running-induced acute changes in power performance.....	61

6.5	Factors underlying running power in maximal anaerobic interval run	62
6.6	Different adrenal and testicular responses to continuous and intermittent runs between marathon and middle distance runners	64
6.7	Acute lipid and lipoprotein responses to prolonged exercise	68
7	PRIMARY FINDINGS AND CONCLUSIONS.....	71
	YHTEENVETO	74
	REFERENCES	77
	ORIGINAL PAPERS	

1 INTRODUCTION

In endurance running technique does not limit performance to the same extent as in many other sports and high intensity can be maintained over long distances without obvious changes in technique. Therefore, physiological and biomechanical responses to endurance running exercises, to running induced fatigue, and to running training have inspired sport scientists, and have been studied extensively during the past decades. Most of the early studies concentrated on the behavior of the circulatory and respiratory systems (e.g. Hill and Lupton 1923) and on the limitations of energy production processes during different types of running exercises (e.g. Åstrand et al. 1960a,b and Christensen et al. 1960). Later, considerable attention has also been paid to changes in the neuromuscular system during and after running performances (e.g. Nicol et al. 1991a,b, Paavolainen et al. 1999b,c) and training (e.g. Paavolainen et al. 1999a). It has been shown, that especially in middle distance running, both central factors related to oxygen uptake, and muscle power factors related to various neuromuscular and anaerobic characteristics, limit the performance in well-trained athletes (Noakes et al. 1990, Rusko et al. 1993, Paavolainen et al. 1999c).

In order to improve the quality and specificity of training methods, it is important to understand both the responses to acute exercises and to training in a particular sport. It is well known that training programs of elite endurance runners usually consist of an early "aerobic base" component, complemented by high-intensity interval training sessions nearer to the competitive season (Laursen and Jenkins 2002). "Aerobic base" and marathon type of running performance have traditionally been trained mostly using continuous type submaximal running exercises, while high-intensity interval exercises have mostly been used to sharpen the performance of runners specialized for middle distances (Hawley et al. 1997). Since the early studies of Åstrand et al. (1960a,b) and Christensen et al. (1960), it is known that by working intermittently the target distance can be run faster than when compared to working continuously. This is associated with the use of myoglobin oxygen and phosphocreatine stores and their rapid recharging during the recovery periods (Åstrand 1992). On the other hand, running the same distance continuously is more strenuous but, at

the same, may be a greater training stimulus than running it intermittently. It has been suggested that during fast continuous running greater physiological strain associates with the inability of the circulatory and respiratory systems to provide oxygen sufficiently fast to fuel energy production in the active muscles concomitantly with the increased rate of lactate production (Åstrand 1992). Recently, this “cardiovascular / anaerobic model” has, however, been questioned and changes in prolonged running performance has been explained by regulative changes at the central nervous system level (“central governor model”) or changes in the muscle contractile functions (“muscle power model”) (Noakes 2001). However, by using intermittent running it is possible to run longer at the target power output, e.g. at the velocity associated with VO_{2max} (Billat et al.2000).

A meta-analysis completed by Londeree (1997) has shown that continuous type of training may fail to elicit further performance improvements in already highly trained athletes and that some trained athletes tend to respond better to high-intensity interval type of training. Therefore, exercise scientists have recently paid special attention to examining the physiological mechanisms behind high-intensity interval training sessions (e.g. James and Doust 1998, 1999, Billat et al. 1999b). It is well known that a single endurance running session could lead to a favourable performance development, if it sufficiently loads the respiratory and oxygen transport system concomitantly with acute positive physiological and neuromuscular responses. Exercise-induced acute anabolic activity and / or a shift to a more anabolic hormone environment have been considered as an example of a positive physiological response (e.g. Schwarz and Kinderman 1990, Häkkinen and Pakarinen 1993). It has been shown that after high intensity running remarkable exercise induced increases in serum concentrations of both testosterone (e.g. Kuoppasalmi et al 1976, 1980, Slowinska-Lisowska and Majda 2002) and cortisol (Kuoppasalmi et al 1976, 1980, Kargotich et al. 1997) may take place and that they depend on the nature of the performed exercise (Galbo 1981).

Although high rates of oxygen delivery to fuel energy production in the active muscle mass may be considered necessary in developing endurance running performance it may, at least in some circumstances, also cause unfavourable physiological changes. There is strong evidence, that when large amounts of oxygen are inhaled into the human body, increased release of pro-oxidants may cause oxidative stress (Davies et al. 1982, Jenkins 1988) and harm various biological structures (Del Maestro 1980, Davies et al. 1982). The magnitude of this damage is suspected to relate to the intensity and duration of the exercise and the capacity of the antioxidant defence system of the athlete (Davies et al. 1982, Halliwell 1994, Haramäki and Packer 1994, Kujala et al. 1996).

Endurance running exercises also produce acute changes in muscular performance. The magnitude of these changes, usually a decrease in performance, related with muscle fatigue, has been shown to depend on the type of exercise performed (Viitasalo et al. 1982, Häkkinen 1993, Lepers et al.

2000a, Millet and Lepers 2004). A dramatic strength loss in the leg muscles has been demonstrated during running exercise of long duration (e.g. Nicol et al 1991a). However, under some circumstances, e.g. after warming up and after short intense repetitions, intra-and inter-muscular factors like muscle twitch potentiation (Rassier and McIntosh 2000) and coactivation of synergist muscles (Gandevia 2001) may effect muscle strength output in the opposite direction. There is also some evidence that the acute effects of a running exercise on the performance of leg muscles depends on the physical characteristics and training history of the athlete. Lower and higher calibre male distance runners have differences in their muscular responses to a 10 km time trial (Paavolainen et al. 1999b) and different muscular performance responses to intensive intermittent exercise have been documented between long distance and short distance runners (Paavolainen et al. 1994).

Although much specific scientific data are available about the running exercise-induced changes in oxygen dependent metabolism, endocrinology, neuromuscular functions and oxidative stress, there is a lack of studies where all these parameters have been taken into consideration. The aim of the present series of studies was to examine the benefits of different types of endurance running exercises from an endocrine, neuromuscular and oxidative stress response view point, in well-trained marathon and middle-distance runners. It was hypothesized that the acute exercise-induced changes in muscular and physiological functions are related to the type of running exercise and to the physical characteristics and / or training background of an athlete.

2 REVIEW OF THE LITERATURE

2.1 Energy delivery in endurance running

2.1.1 Energy delivery in middle and long distances is based on oxygen-dependent metabolism

According to the “Cardiovascular / Anaerobic model”, named and criticized by Noakes (2001), the performance capacity of human muscles is dependent on the continuous delivery of energy in the form of ATP. The utilization and production of ATP are delicately balanced, although in athletic performances the energy needs of the working muscles may increase dramatically, e.g. during middle distance running over 1000-fold in a couple of seconds (Newsholme 1978, Bangsbo et al. 1990).

The most important biochemical processes producing ATP in human cells are oxygen independent glycolysis of carbohydrates and the aerobic metabolic pathways for the metabolism of carbohydrates and free fatty acids (the citric acid cycle, beta-oxidation and the respiratory chain). The relative importance of these processes differs from tissue to tissue and depends on the functional and nutritional state of the body. The choice of fuel (carbohydrates vs. free fatty acids) and the amount needed depend on the type and duration of muscle work (Newsholme 1976, Bangsbo et al. 1990).

It is well known that in prolonged athletic performances such as middle and long distance running ATP is produced mostly by oxygen-dependent metabolism in the mitochondria and that the performance is related to a great extent on the oxygen supply to these organs (e.g. Bassett and Howley 1997). On the basis of the early findings of Hill and Lupton (1923), it was long accepted that oxygen consumption rises as an exponential function of running speed and that anaerobic metabolism begins after the athlete achieves either the absolute VO_{2max} or an individual VO_2 plateau at a certain running speed. Later, the role of oxygen-dependent metabolism in prolonged exercises has been more critically discussed (e.g. Noakes 2001). On one hand, it is well documented that hypoxia develops in active muscles during an intensive exercise and causes

fatigue. Based on that, and agreeing with Hill and Lupton (1923), many scientists have suggested that it is the VO_{2max} , which mainly limits the performance in prolonged muscle work as endurance running (Costill et al. 1973, Foster et al. 1978, Davies and Thomson 1979, Joyner 1991, Basset and Howley 1997). On the other hand, and especially in the case of endurance running, it is suggested that the maximum achieved work rate rather than the VO_{2max} is the best predictor of running potential (Scrimgeour et al. 1986, Lacour et al. 1990, Padilla et al. 1992, Yoshida et al. 1993, Grant et al. 1997). Recently, it has been shown that the lowest velocity by which VO_{2max} can be achieved (the velocity associated with VO_{2max}) and further the time to exhaustion at this velocity are good predictors of performance in elite long distance runners (Billat et al. 1994, 1996, 1999). Especially on longer distances and in athletes with similar VO_{2max} values, the fractional utilization of VO_{2max} ($\%VO_{2max}$) and/or the lactate or respiratory compensation (ventilatory) thresholds measured in the laboratory (Costill et al. 1973, Farrell et al. 1979, Aunola and Rusko 1986), are suggested to relate with prolonged running performance (Costill et al. 1973, Farrell et al. 1979, Kumagai et al. 1982, Tanaka and Matsuura 1984, Noakes et al. 1990). Further, factors related with running economy have been linked to endurance running performance (Conley and Krahenbuhl 1980, Morgan et al. 1989a) especially in high level runners and with similar VO_{2max} values.

2.1.2 VO_{2max} and endurance running performance

Based on the facts that oxygen-dependent metabolism delivers most of the needed energy in endurance running and that VO_{2max} , measured in the laboratory, is a good indicator of aerobic work capacity, many running studies have propagated the idea that VO_{2max} can predict how well any endurance runner will perform on the road or track (e.g. Costill 1967, Costill et al. 1973, Davies and Thompson 1979, Foster 1983, Bassett and Howley 1997, 2000). However, all these studies have looked at groups of athletes with quite different abilities, including the high level and the low level runners. Such an approach will, according to Noakes (2001), deliver only predictable results - slow runners achieve low VO_{2max} values and fast runners higher VO_{2max} values. In a homogeneous group of well-trained endurance runners, only low correlations between VO_{2max} and endurance running performance are documented (Daniels et al. 1978, Conley and Krahenbuhl 1980, Noakes 1988, Morgan et al. 1989, Noakes et al. 1990). Therefore, VO_{2max} seems not to be a sensitive predictor of endurance running performance when groups of athletes with similar endurance running results are studied (Costill and Winrow 1970a,b, Pollock 1977, Conley and Krahenbuhl 1980, Noakes et al. 1990, Abe et al. 1998).

Even the whole idea that intensive running exercises will cause hypoxia and that this hypoxia leads to fatigue and therefore limits endurance running performance has been questioned (Noakes 1988, 1998, 2001). The evidence for this criticism is based on the fact, that the plateau phenomenon and oxygen transport, as a limiting factor for VO_{2max} (Hill and Lupton 1923, Basset and Howley 1997), have never really been proved (Noakes 1988, 1998). Recently, it

has been more intensively suggested that neural activation have an important role as a determinant of endurance performance and $\text{VO}_{2\text{max}}$. (Paavolainen et al. 1999c, Noakes 2001). This hypothesis is based on the idea that an inability to properly activate the appropriate muscles or a failure in some excitation-contraction process within the recruited fibers could limit running performance and thus prevent a runner to reach a high VO_2 level (Noakes 2001). According to some earlier studies (e.g. Mainwood and Renaud 1985, Green and Patla 1992) an increased H^+ ion concentration could be the factor which may impair the contractile properties of the muscles during intensive exercising. Noakes (1988) presents that endurance running performance may not only be limited by central factors related to oxygen uptake, but also by a so called muscle power factor. Muscle power in this regard is linked to neuromuscular characteristics but not fully defined (Rusko et al. 1993, Rusko and Nummela 1996, Paavolainen et al. 1999c).

It is hypothesized that a combination of heart and skeletal muscle factors, other than only the central cardiovascular and respiratory factors related to oxygen transport and utilization and/or factors regulating oxygen use by the active muscles alone, determine athletic potential in endurance running (Green and Patla 1992, Liefeldt et al. 1992, Noakes 1998, 1997). This suggestion is based on the fact that skeletal muscle function during intensive endurance exercise appears to be regulated and limited by different preventive reasons (Noakes 1997). First, to prevent the development of muscle ATP depletion, which may cause irreversible skeletal muscle rigor (Lewis and Haller 1986, Spriet et al. 1987). Second, to prevent cerebral hypoxia during exercise at altitude (Green et al. 1989, Kayser et al. 1994). Third, to prevent a catastrophic fall in blood pressure (Rowell et al. 1986), which could reduce perfusion of the diseased coronary arteries in chronic heart failure.

2.1.3 Best runners are economical

It is well known that there may be differences in the amount of oxygen athletes actually require when running at the same speeds and that these differences in running economy could be an important factor explaining differences in running performance in athletes with similar $\text{VO}_{2\text{max}}$ values (Costill and Winrow 1970b, Daniels 1974, Noakes 1988). It has been reported that in trained athletes running economy (VO_2 at same submaximal running velocity) can differ by as much as 30% (Sjodin and Svedenhag 1985, Daniels and Daniels 1992), which usually is far more than the documented difference in their best running results (Costill and Windrow 1970a, Daniels and Daniels 1992).

It is documented that some of the very best athletes consume the least oxygen at the same submaximal running velocities near to the competition velocity, and thus seem to be the most economical endurance runners (Noakes 1988, 2001). This finding is supported e.g. by Conley and Krahenbuhl (1980). They studied endurance runners whose best 10-km times were between 30,5 and 33,5 min, and found that the runners who used the least oxygen at submaximal running speeds (14.5, 16.1 and 17.7 $\text{km} \cdot \text{h}^{-1}$), and were therefore

the most economical, had the fastest 10-km times. The high $\text{VO}_{2\text{max}}$ (above $67 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) only seemed to be a factor, which helped them to reach the high level performances not the factor controlling success in the 10-km race. This kind of finding may also be interpreted in a somewhat different way. To join the elite group of runners, the athlete needs a superior and efficient heart, which is able to achieve a high cardiac output at the maximum test to exhaustion (Noakes 2001). The high work rate demands a high rate of oxygen consumption that is interpreted as high $\text{VO}_{2\text{max}}$. But, the exact $\text{VO}_{2\text{max}}$ value, that each athlete achieves (whether in laboratory or during racing), will be determined by his or her running economy, and it is independent of the exact peak work rate that is achieved (Noakes 1988). At the same maximal work rate, uneconomical runners (e.g. when the runner gets older) will have higher $\text{VO}_{2\text{max}}$ values, and economical runners will have lower values. Therefore, it could be concluded that the real predictor of performance is actually the achieved maximal work rate (running speed), and not the $\text{VO}_{2\text{max}}$ measured at that work rate (Noakes 2001).

2.2 Neuromuscular characteristics and muscle power in endurance running

2.2.1 Muscle activation and muscle power

In middle distance running and during the final sprint in longer distances the running power exceeds maximal aerobic running velocity (Billat et al. 1995) and, in addition to loading aerobic pathways maximally, a large amount of energy is produced by anaerobic metabolism (e.g. Di Prampero et al. 1993). Especially in these situations, running performance may be limited by the neuromuscular characteristics and changes in neuromuscular functions (Noakes 1988, Green and Patla 1992). Inside the muscle fibers, disturbances in functions of sarcolemma and sarcoplasmic reticulum as well as regulatory and contractile proteins could theoretically regulate maximal endurance running performance (Green and Patla 1992). It has been shown that an exercise-induced inability to sustain calcium release from the sarcoplasmic reticulum would lead to lower activation levels and consequently to less force production. In addition, a prolongation of time required to remove the calcium from the cytosol would prolong the dissociation of the actin and myosin and result in a delay in the relaxation of the muscle during the recovery phase (Tate et al. 1991, Allen et al. 1992, Green and Patla 1992, Green 1997).

It is well known, that the force output of muscle contraction depends on muscular, neural and mechanical factors (Komi 1986, Enoka 1988a, Green 1997). The myofibril cross-section area is related to maximal muscle strength so that larger muscles, as a result of an increase in fiber size, are able to produce greater force output than muscles with a small cross-sectional area (e.g. Sale et al. 1987).

However, a poor correlation exists between training-induced increases in strength and muscle size (Enoka 1988a) because strength performance is determined not only by quantity of the involved muscles but also by the ability of the nervous system to appropriately activate the muscles (Enoka 1988b, Häkkinen et al. 2000). Increased muscle activity (that more motor units have been recruited and/or motor units are firing at higher rates) can be observed as an increase in the integrated electromyographic activity (IEMG) (Enoka 1988, Häkkinen et al. 2000). In addition to voluntary neural control, changes in reflex potentiation may affect to the muscle activation and muscle power during an athletic performance (Häkkinen and Komi 1983).

During an athletic performance the velocity of action, type of action, and movement pattern could affect motor unit recruitment within a muscle (Sale 1991). The power output and the efficiency of human performance are also related to elasticity and stretch reflexes in muscles and tendons (e.g. Komi 1984, 1991, Enoka 1988b). It has been suggested that especially in fast running the nervous system plays an important role in regulating muscle stiffness and utilization of muscle elasticity during stretch-shortening actions (Aura and Komi 1986, Kyröläinen et al. 2005).

2.2.2 Muscle structure

The capacity to sprint and long distance running performances is suggested to be, at least partly, of genetic origin and dependant on individual differences in muscle structure (Abe et al. 2000) and relative proportions of fast- (FT) and slow- (ST) twitch fibers in the runners' muscles (Komi et al. 1977, Komi and Karlsson 1979). Sprinters tend to have larger muscles with longer muscle fascicle lengths, which are a measure of the number of sarcomeres stacked end to end (in series) in their muscles (Abe et al. 2000). Especially African-American sprinters also seem to have a different muscle shape with greater muscle thickness in the upper parts of their muscles near where they originate from the skeleton (Abe et al. 1999, 2000). The pennation angle, which is a measure of the angle at which the muscle fibers insert into the tendons, is also less in sprinters than in distance runners (Abe et al. 2000).

It is well known that endurance athletes have a higher proportion of ST fibers in their active muscle groups than sprint athletes (e.g. Gollnick et al. 1972, Costill et al. 1976). Muscles composed of greater percentage of FT fibers have greater muscle strength, a shorter electromechanical delay, a higher contraction velocity and shorter relaxation times compared to ST type muscles (e.g. Viitasalo and Komi 1981, Komi 1984). ST fibers have a longer cross-bridge cycle time and may therefore be able to better utilize long and slow stretches (Bosco et al. 1982). On the other hand, it is documented that some black African distance runners who perform best at distances from 5 to 42 km have more similar muscle fiber composition as found in white middle distance runners racing at distances up to 3 km (Weston et al. 1999). However, the black African runners seem to have a good resistance to fatigue and they are documented to be more economical, at least at high running velocities, than white runners

(Weston et al. 2000). Therefore, one possible explanation is that the fast twitch fibers of the black runners are fundamentally different from those of the white middle-distance runners and are characterized by extreme fatigue resistance (Noakes 2001).

2.2.3 Running mechanics

Distance running performance and running economy at different running velocities may also be influenced by stride characteristics and running kinematics such as; stride length and rate, ground contact times, ground reaction forces, joint and segment angles and movements of the trunk and arms (Williams 1985, Williams and Cavanagh 1987, Andersson 1996). Although it is suggested that the performance in distance running is related to a weighted sum of the influences of many kinetic and kinematic variables, the most relevant biomechanical factors of the running performance in elite distance runners are still unknown (Williams and Cavanagh 1987, Andersson 1996). It has been shown (Cavanagh and Williams 1982) that runners appear to choose the individual stride length at which they are most economical – that is, at which their oxygen uptake is the least for that particular running speed. When forced to take either shorter or longer strides but to maintain the same running pace, they become less economical and require an increased oxygen uptake.

2.2.4 Development of muscle fatigue

An acute decrease in force generating capacity during the exercise-induced development of fatigue has been well demonstrated and it is linked to both metabolic and neural changes (e.g. Moritani et al. 1990, Nicol et al. 1991a, Paavolainen et al. 1999b). In exercises of long duration, such as long distance running, two types of fatigue have been suggested to impair the neuromuscular performance (Gibson and Edwards 1985, Asmussen 1979). It can be of peripheral origin and related to an impairment of the function of the peripheral nerves, neuromuscular junction transmission, electrical activity of muscle fibers or failure of sarcolemma and sarcoplasmic reticulum in the excitation and contraction processes (Edwards 1981, Gibson and Edwards 1985, Green 1997). Central causes of fatigue include motivation, impaired signal transmission down the spinal cord and impaired recruitment of motor neurons (Asmussen 1979, Gibson and Edwards 1985, Green 1997).

Repeated stretch-shortening cycles during prolonged fatiguing exercises have been shown to decrease force production by reducing neural input to the muscle and the efficiency of the contractile mechanism (Gollhofer et al. 1987a,b, Moritani et al. 1990, Nicol et al. 1991a,b, Nummela et al. 1994). Paavolainen et al. (1999b) found that a 10 km test run led to a significant reduction in force production and velocity in a maximal 20 m run immediately after the 10 km test both in high and low caliber runners. Similar findings have been shown after a marathon race, where maximal sprint velocity and ground reaction forces decreased and ground contact times increased, suggesting a reduced tolerance

to stretch load as well as a loss in the recoil characteristics of the muscles (Nicol et al. 1991a). Because the ability to store and use elastic energy is affected by the velocity of the pre-stretch action, the coupling-time, velocity of stretch and muscle stiffness, the fatigue related failure in stiffness characteristics and longer transition time between the braking and propulsion phases could reduce the storage of elastic energy during the braking phase and thus reduce running efficiency (Dietz et al. 1979, Bosco et al. 1981, Komi 1984, Enoka 1988a, Nicol et al. 1991a). Although some fatigue-induced changes in neuromuscular functions have been observed after strenuous prolonged runs submaximal running does not necessarily result in marked changes in running kinematics during or after the exercise (Williams et al. 1991).

2.3 Definition and characteristics of endurance running exercises

2.3.1 Exercise models

In training of endurance running performance several different exercise models at different intensities have been used successfully although they are not based on scientific findings and documented in scientific publications. The successful training usually is a mixture of slow continuous type of exercises and of faster intermittent type of exercises. Daniels (1998) has presented a model of the six training intensities that he considers most effective in enhancing running performance: 1) easy / long pace (continuous runs at an intensity of about 70% VO_{2max}), 2) marathon pace (continuous runs at a marathon pace), 3) threshold pace (continuous runs or long repetitions at an intensity of about 88-90% VO_{2max}), 4) interval pace (intermittent runs at the speed that can be maintained for 10 to 15 minutes in a race situation), 5) repetition pace (intermittent runs faster than VO_{2max} pace), 6) rest.

Pfizinger and Douglas (1999) have provided another approach to the training of serious distance runners and described five components of training: 1) Short, fast speed work to improve "leg turnover and running form". 2) Longer repetitions of 2 to 6 minutes at 3- to 5-km race pace to improve the VO_{2max} . 3) Tempo runs of 20 to 40 minutes at 10-mile (16km) race pace to increase the lactate threshold. 4) Long runs to build endurance. 5) Easy recovery runs to allow a maximum effort on the hard training days.

Burfoot and Billing (1985) present that optimum training is achieved by including regular runs at three different intensities. First, long runs at intensities between 56% and 75% VO_{2max} , which correspond to the pace below that at which a runner runs the marathon. The purpose of these runs is to improve running efficiency. Second, they prescribe a once-a-week run of 5 to 10 km at 85% VO_{2max} to shift the lactate turnpoint to a higher percentage VO_{2max} . This exercise intensity corresponds to the speed an athlete can maintain during races of 10 to 21 km. It is not necessary to run the 5 to 10km of this session

continuously. Rather the authors suggest that this type of running exercise should be run on the track or road as series of repeat runs of 2 to 3km. Finally, Burfoot and Billing (1985) suggest once-a-week a running exercise performed at an intensity eliciting $\text{VO}_{2\text{max}}$. This intensity corresponds to the speed at which an athlete can run a 3 km race. They suggest 3 to 6 x 800m or 8 to 12 x 400m for a competitive runner.

2.3.2 Continuous versus intermittent exercises

In scientific studies, running exercises are usually classified into continuous and intermittent exercise models (Laursen and Jenkins 2002). Continuous exercises are usually performed at submaximal velocities / intensities under the lactate threshold (anaerobic threshold) while intermittent runs are performed at higher velocities / intensities (Daniels and Scardina 1984, Billat et al. 2000, Laursen and Jenkins 2002).

The training programs undertaken by highly trained endurance runners usually consist of an early “aerobic base” component, complemented by high intensity intermittent training sessions nearer to the competitive season (Laursen and Jenkins 2002). The “aerobic base” training consists mainly submaximal (continuous) running exercises and only a minimal amount of high intensity intermittent runs (Laursen and Jenkins 2002).

For highly trained athletes it is problematic that most scientific advice on training principles is based on studies completed with previously untrained or recreationally active individuals (Laursen and Jenkins 2002). It has consistently been shown that submaximal endurance training in previously untrained individuals will increase plasma volume (Green et al. 1987,1990), oxygen delivery to exercising muscles (Green et al. 1990, 1991a), and utilization of oxygen by the working muscles (Green et al. 1991b, 1992). However, changes in these variables do not occur when already highly trained athletes increase the volume of their submaximal training (Costill et al. 1988, Henriksson 1992). It has even been suggested, that there is a critical $\text{VO}_{2\text{max}}$ value at a level of $60 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Once an individual has reached this level, endurance performance is not improved by a further increase in submaximal (continuous) training volume (Londeree 1997).

Changes in VO_2 during typical middle distance (intermittent) exercises have been shown to depend on different intermittent work and rest modifications, although the subjects have seldom been well-trained athletes (Åstrand et al. 1960a, Christensen et al. 1960, Lindsay et al. 1996, Tabata et al. 1997). With regard to muscle power factors, corresponding changes between different intermittent exercise modifications have not been well documented. However, it has been suggested that especially the performance of trained middle distance runners may benefit from fast intermittent exercises (Daniels et al. 1978, Gullstrand and Lawrence 1987, Noakes et al. 1990)

2.3.3 Modifications of intermittent exercises

Recently, the running velocity associated with VO_{2max} , which corresponds to the velocity of a 3000-5000 m race, has been described to be a suitable training intensity for middle distance running (Hill and Rowell 1997). It may be considered especially effective, provided that the skeletal muscle contractility, running efficiency and oxygen delivery could be maintained at high level (Hultman and Spriet 1986, Noakes 2001).

One of the oldest studies (Christensen et al. 1960) of intermittent muscle work has shown that at the intensities exceeding the maximal oxygen uptake, intermittent exercises can be performed without blood lactate accumulation and muscle fatigue if the duration of the exercise bouts are not longer than 30 seconds. This is possible through unloading of myoglobin oxygen and phosphocreatine stores and their rapid recharging during the recovery periods (Åstrand 1992). If the duration of high intensity exercise bouts (and rest periods) is lengthened the physiological strain will increase and the desired exercise can only be accomplished at a great effort (Åstrand et al. 1960a). When the intensity of intermittent exercises does not exceed maximal aerobic power (3000-5000m competitive pace) the physiological strain will be lower and obviously at least 60 second exercise bouts could be run with a moderate effort and without remarkable muscle fatigue and decrease in muscular performance (Billat 2001, Laursen and Jenkins 2002).

Despite the fact that coaches have long used high intensity intermittent exercises to improve the performance of their elite endurance athletes (Hawley et al. 1997), exercise scientists have only recently sought to understand the physiological mechanisms behind the practice (Laursen and Jenkins 2002). As stated earlier by Åstrand and Rodahl (1986) and later by Billat (2001) it is an important, but still unsolved question, which type of training is most effective: to maintain a level representing 80-90% of the VO_{2max} for 30-50 minutes, or, to exercise at the level of about 100% of the VO_2 capacity for 10-20 minutes? In addition to the longitudinal studies focusing on this question, it is important to determine the acute metabolic response solicited by the different interval training protocols used by trainers (Daniels et al. 1986).

In middle distance running, the role of cardiorespiratory fitness should be examined critically. Even if optimal improvement in cardiorespiratory fitness is thought to occur from training at an intensity corresponding to 90 to 100% of VO_{2max} , this central factor of performance (Åstrand and Rodahl 1986) is not the only one to induce improvement in running performance. Consequently, time spent at VO_{2max} is not the only parameter to be taken into account to judge the efficiency of a certain pattern of interval training, when trying to improve performance in middle distance running (Billat 2001).

2.4 Muscle activation and muscle performance responses to endurance running exercises

2.4.1 Exercise-induced strength loss

Different types of intensive or prolonged physical exercises produce acute changes in muscular performance. The magnitude of these changes, usually a decrease in performance, related to different types of fatigue, differs according to the types of contraction involved, the muscle groups tested and the exercise duration / intensity (Viitasalo et al. 1982, Kraemer et al. 1990, Häkkinen 1993, Lepers et al. 2000a, Millet and Lepers 2004, Izquierdo et al. 2006). After an exercise of long duration, isometric strength loss has been reported to increase in a non-linear way with the exercise duration (Nicol et al. 1990a,b, Lepers et al. 2000b, Millet et al. 2002, Millet et al. 2003, Millet and Lepers 2004, Place et al. 2004) and the strength loss seems to be lower in concentric than in isometric or eccentric contractions (Lepers 2000b, Millet and Lepers 2004).

The role of central fatigue in neuromuscular perturbation has been studied using the twitch interpolation technique, the ratio of the EMG signal during maximal voluntary contraction normalized to the M-wave amplitude or the comparison of forces achieved with voluntary and electrically-evoked contraction (Millet and Lepers 2004). In addition, peripheral fatigue has been studied by measuring changes in muscle performances associated with changes in various physiological parameters. This has been done to find out to what extent the strength loss results from a decreased neural input to the muscles, and / or, to a failure in the contractile mechanism of the working muscles (Lepers et al. 2000a,b, Rassier and MacIntosh 2000, Gandevia 2001, McIntosh and Rassier 2002, Place et al. 2004).

2.4.2 Counteracting exercise-induced strength loss

While a dramatic strength loss of leg muscles has been demonstrated during exercises of long duration by many authors, it is also known that under some circumstances intra- and inter-muscle factors like muscle twitch potentiation (Rassier and MacIntosh 2000) and coactivation of synergist muscles (Gandevia 2001) may at least partly counteract strength loss during a prolonged exercise. A brief period of repetitive stimulation is known to result in enhanced contractile response of a muscle (Rassier and MacIntosh 2000), while coactivation synergism is assumed to occur predominantly under conditions of high load and increases with the duration of exercise (Sirin and Patala 1987). There is also some evidence that the acute effects of a running exercise on the performance of leg muscles depend on the physical characteristics and training history of the athlete. The muscular performance responses to intensive intermittent exercise are different in long distance runners compared to runners adjusted to shorter distances (Paavolainen et al. 1994). Lower and higher calibre

male distance runners also seem to have differences in their muscular responses to a 10 km time trial (Paavolainen et al. 1999b).

It is also well known, that fatigue is delayed in an intermittent compared to a continuous type of exercise. Long distance runners have been demonstrated to double the total distance covered at the velocity associated with maximal oxygen consumption in intermittent running (2 min running bouts / 2 min rest) compared with continuous running at the same velocity (Billat 2001). This is possible through the unloading of myoglobin oxygen and phosphocreatine stores and their rapid recharging during the recovery periods (Åstrand 1992). Besides the metabolic differences, the repeated running bouts of short duration obviously also effects muscular performance differently compared to a prolonged continuous running exercise. During the initial phases of a progressive and intermittent anaerobic running tests (Paavolainen et al. 1994) counter movement jumping performance has been shown to remain unchanged or even to improve although after prolonged running of long duration it has been reported to decrease by about 10 % (Lepers et al. 2000b).

2.5 Effects of physical exercise on adrenal and testicular functions

2.5.1 Acute adrenal and testicular responses to different type of endurance running exercises

It is well known that hard physical training sessions induce remarkable acute hormonal responses on the pituitary-testicular axis (testosterone, LH, FSH) and adrenal cortex (cortisol) (Sutton et al. 1973, Galbo et al. 1977, Galbo 1981, Schmid et al. 1982). The hormonal secretion involved in these endocrine organs has been studied during and immediately after single bouts of exercise in numerous strength and endurance training protocols (e.g. Schwarz and Kinderman 1990, Jensen et al. 1991, Häkkinen and Pakarinen 1993, Vasankari et al. 1993a,b, Kenefick et al. 1998). An exercise-induced shift in the hormonal environment towards an anabolic or catabolic direction, has been evaluated by comparing the changes in serum testosterone and other anabolic hormones, e.g. dehydroepiandrosterone (DHEAS), to the changes in serum cortisol (Kenefick et al. 1998, Gorostiaga et al. 2004, Daly et al. 2005, Tremblay et al. 2005).

In the case of low intensity and continuous type of running, the ratios of anabolic hormones to cortisol has been reported to depend on the exercise time: after 40-min of running the ratio is greater compared to the pre-exercise level while beyond 80 min of running there is a shift to a catabolic hormonal environment (Tremblay et al. 2005). After high intensity running remarkable exercise-induced increases in the serum concentrations of both cortisol (e.g. Kargotich et al. 1997) and testosterone (e.g. Slowinska-Lisowska and Majda 2002) have been documented and been shown to depend on the exercise intensity (Galbo 1981).

The concentration of serum cortisol has been shown to increase at the work loads exceeding 50-60% of VO_{2max} and the increase is documented to be due to increased secretion of the hormone (Galbo 1981). At work intensities below 50% VO_{2max} , the cortisol concentration in serum usually decreases because the rate of removal of cortisol from plasma is higher during exercise than at rest, while the secretion rate at low work loads tends to be lower than at rest (Galbo 1981). Serum cortisol concentration especially seems to respond differently to anaerobic compared to aerobic exercises (Kindermann et al. 1982, Hackney et al. 1995), but also depends on various other types of stress factors (Berson and Yallow 1968).

The concentration of serum testosterone has been shown to increase during high intensity exercises, defined as an exercise intensity exceeding 80% of VO_{2max} (Sutton et al. 1973, Schmid et al. 1982, Slowinska-Lisowska and Majda 2002) while short term exercises performed at intensities lower than 35% VO_{2max} seem not to affect serum testosterone (Sutton et al. 1973, Galbo et al. 1977, Tremblay et al. 2005). The change in the serum testosterone concentration during exercise can be caused by a change in the production rate, altered binding or changed clearance (Cumming et al. 1989). During short term exercise, the increase in serum testosterone concentration is not usually associated with a preceding LH spike (Cumming et al. 1986). Therefore, it has been suggested that several nonendocrine mechanisms, including increased activation of sympathetic nervous system (Sutton et al. 1974), could lead to an exercise-induced increase in serum testosterone concentration (Cumming et al. 1989). A decreased plasma volume is one possible explanation to the increased testosterone concentration during exercise (Galbo et al. 1977, Kindermann et al. 1982). Reduced metabolic clearance may also increase the testosterone levels during exercise (Jensen et al. 1991). However, other steroid hormones, e.g. androstenedione and dehydroepiandrosterone have been shown to increase 25 to 30 min after the start of the testosterone rise during exercise (Cumming et al. 1986) and this difference in the time responses suggests that also other mechanisms are involved as far as testosterone is concerned.

During intensive exercises of long duration (more than 2h) serum testosterone has usually been shown to decrease (e.g. Morville et al. 1979, Schmid et al. 1982, Tremblay et al. 2005). At the same, the concentration of serum gonadotropins has either been reported to be unchanged (e.g. Terjung 1979, Galbo 1981) or decreased, provided that the duration of intense exercise is long and sufficient analysis methods are used (Vasankari et al. 1993a,b). It has been concluded that in the case of prolonged exercises of long duration and high intensity, the exercise-induced decrease in circulating testosterone may, at least partly, be caused by regulative changes within the hypothalamic-pituitary axis (Vasankari et al. 1993a,b)

2.5.2 Individual variation in adrenal and testicular responses

It is suggested that the activity of the pituitary-adrenocortical system during short-term intensive exercise may be affected differently in subjects of high and low level of physical fitness (Kuoppasalmi et al. 1976) and depending on the training background of athletes, hard endurance type exercises may have different effects on circulating anabolic and catabolic hormones. A prolonged exercise has been shown to increase both serum testosterone and cortisol, in subjects of high and low work capacities (Sutton 1978), although large inter-individual variability may exist in the exercise-induced responses of both hormones (Virtanen et al. 1992). After prolonged exercises serum cortisol concentration has been shown to increase more in non-athletes compared to conditioned male athletes (Mathur et al. 1986, Vasankari et al. 1993a). Some evidence also suggests that a hard endurance type exercise may result in serum testosterone concentration to behave differently in the less-fit subjects than in the trained subjects (Remes et al. 1985, Vasankari et al. 1993a). On the basis of this, it is suggested that the activity of the pituitary-adrenocortical system may be a good indicator of the effort expended during intensive prolonged exercise (Kuoppasalmi et al. 1980).

It is well known that both elite middle distance runners and marathon runners run high mileages in their daily training, but have adjusted differently to fast intermittent or slow continuous types of running exercises (Noakes 2001). Due to different training background and / or genetic differences middle distance runners are faster and have more developed anaerobic capacity compared to marathon runners who have more developed aerobic capacity (Noakes 2001, Arrese et al. 2005). It has also been shown that the capability of the neuromuscular system to produce force rapidly during prolonged running is related to the physical characteristics / performance level of the runners (Paavolainen et al. 1999b). It is obvious that, besides the differences in muscular responses, these types of running exercises also stimulate the endocrine system and the hormonal secretion involved in the pituitary-testicular axis (testosterone, LH, FSH) and adrenal cortex (cortisol) differently in middle distance runners when compared to marathon runners.

2.6 Exercise induced acute oxidative stress and antioxidant defence

2.6.1 Free radicals and oxidative damage

During acute physical exercise large amounts of oxygen are inhaled into the human body. Oxygen consumption in the working peripheral skeletal muscle tissue may increase even 20-fold during intensive endurance exercising (Brooks and Fahey 1984). It is well known that prolonged aerobic training has many positive effects on human cardiorespiratory function and lipid metabolism, e.g.

a reduction in the level of plasma triglyceride and an increase in the level of HDL cholesterol (Durstine and Haskell 1994). However, there is also strong evidence that the pro-oxidants released during a single bout of heavy and exhausting exercise may cause oxidative stress (Davies et al. 1982, Jenkins 1988) and harm various biological structures (Dillard et al. 1978, Del Maestro 1980, Davies et al. 1982, Brooks and Fahey 1984).

The magnitude of oxidative damage may be related to the power of the pro-oxidant attack (intensity and duration of physical exercise) and the capacity of the individual exerciser's antioxidant defence system (Davies et al. 1982, Halliwell 1994, Haramäki and Packer 1994, Kujala et al. 1995). It has been suggested that oxidative stress associated with exercise is better tolerated by trained subjects working at moderate intensity and not to exhaustion (Alessio 1993). Direct measurement of free radical signals can be made by electron spin resonance and indirect measures include e.g. mitochondrial membrane damage, conjugated dienes, hydroperoxides, thiobarbituric acid reactive substances, short chain hydrocarbons, and oxidized nucleosides (Alessio 1993).

2.6.2 Methods of measuring exercise-induced oxidative stress and antioxidant defence

The oxidative damage sustained by lipoproteins has been measured by a number of methods (Slater 1985). Serum diene conjugation has been found to be a useful method to estimate the level of lipid peroxidation (Vasankari et al. 1995). It measures the early events of lipid peroxidation and might, therefore, be less sensitive to the numerous compensatory antioxidative mechanisms that occur in the organism and are a problem in some other measurements (e.g. thiobarbituric acid reactive material and fluorescent chromolipids); these methods measure end products of lipid peroxidation rather than the early phases of the process. However, all these commonly used analytical methods have some limitations and the main analytical question is unresolved. In addition to the influences of methodology, exercise intensity, training, nutritional status, and delayed-onset of lipid peroxidation after exercise might be responsible for inconsistency of results (Davies et al. 1982, Haramäki and Packer 1994, Ji 1995).

A prolonged exercise program also seems to enhance the antioxidant defences of the human body (Halliwell 1994, Haramäki and Packer 1994), which have been reflected by changes in serum and LDL antioxidant potential (Vasankari et al. 1998). Contradictory findings have been reported in top soccer and basketball players by elevated titers of autoantibodies against oxidized LDL (Pincemail et al. 2000).

2.6.3 Oxidized LDL as an indicator of exercise-induced oxidative stress

Oxidative modification of LDL plays a special role in oxidative stress studies, because oxidized LDL is strongly associated with atherosclerosis, including coronary artery diseases (Steinberg 1994, Witztum 1994). It has been found that

several years of intensive physical training (Kujala et al. 1996) as well as a 10-month exercise program (Vasankari et al. 1998) is associated with reduced oxidized LDL, reflected by low levels of baseline LDL diene conjugation (LDL-BDC).

There is only limited information on the effects of a single bout of prolonged exercise on LDL oxidation. It is not known whether the low LDL-BDC in veteran endurance athletes that has been demonstrated in the study of Kujala et al. (1996) is caused by a single endurance exercise. It is known that a single bout of ultra-endurance exercise reduces the susceptibility of lipids to peroxidation (Ginsburg et al. 1996). It is also known that a short period of submaximal exercise (i.e. < 70% $\text{VO}_{2\text{max}}$), but not maximal exercise of the same duration, reduce free radical generation and lipid peroxidation as evaluated by plasma malonaldehyde formation (Lovlin et al. 1987). This fragmentary information suggests that there are positive lipid and lipoprotein changes associated with acute bouts of exercise and that these changes seem, at least partly, to be connected to the duration and intensity of the exercise. During prolonged exercises carbohydrate ingestion is commonly used to prevent depletion of muscle glycogen and possible muscle fatigue associated to it. However, contradictory findings have been presented about the role of carbohydrate ingestion in preventing the possible exercise induced oxidative stress (Mooradian et al. 1994, Vasankari et al. 1998).

3 THE PURPOSE OF THE STUDY

The present series of studies investigated acute neuromuscular, hormonal and oxidative stress responses to different type of endurance running exercises in well-trained athletes. In order to find out whether the endurance runners have specific responses to different exercises with regard to intensity, duration and model, ten different endurance running exercises were studied. It was hypothesized that the acute exercise-induced changes in muscular and physiological functions are related to the type of running exercise and to the physical characteristics and/or training background of an athlete.

Special attention was given to providing answers to the following questions:

- 1) Does physiological strain and the aerobic energy released vary when the duration of running and recovery bouts is changed in an intermittent run of the same duration and intensity (II)?
- 2) What is the effect of a single maximal or submaximal endurance running exercise on muscle power and muscle activation in extension exercises performed vertically with both legs?
 - a) Are acute muscle activation (III) and muscle performance (I, II, III) responses specific to the type of intermittent (I, II, III) and continuous (III) running in well-trained endurance runners?
 - b) Do an intensive continuous run (III) and three different variations of intensive intermittent runs (I, II, III) have different acute effects on leg extension performances and muscle activation in athletes with different training backgrounds?
- 3) Do acute serum testosterone and cortisol responses differ between intermittent and continuous type of hard running exercises and between well-trained middle distance and marathon runners (IV)?

- 4) Does a single endurance running exercise of long duration cause acute oxidative stress in well-trained athletes?
 - a) What are the acute effects of an endurance exercise of a long duration on oxidation of lipids and antioxidant functions in LDL and serum (V, VI)?
 - b) Is the exercise-induced oxidative stress and changes in antioxidant defense different after an intensive 31-42-km run compared to a low intensity walking exercise of long duration (V, VI)?

4 MATERIAL AND METHODS

4.1 Subjects

A total of 88 male competitive and keep-fit runners volunteered as subjects for this study. The training background of the subjects were as follows: 5 were competitive sprinters engaged in regular sprint training (I), 26 were competitive middle distance runners engaged in middle distance type of training (I, II, III, IV), 25 were competitive marathon runners engaged in marathon type of training (I, III, IV, V), and 32 physically active keep-fit runners (V, VI). In studies III and IV, 20 of the subjects (10 middle distance runners and 10 marathon runners) were the same. Some characteristics of the subjects are summarized in table 1.

TABLE 1 Mean (+ SD) age, height, body weight and VO_{2max} of the subject groups. Ranges of the personal best results in different events are also shown.

Study / subjects (n)	Age (years)	Height (cm)	Body weight (kg)	VO_{2max} ($ml\ kg^{-1}\ min^{-1}$)	Personal best (h:min:sec)
STUDY I					
Marathon runners (6)	27.8 ± 3.4	179 ± 5	64.9 ± 3.3	73.2 ± 3.4	42.2km: 2:17.00 - 2:24.00
Middle distance runners (5)	24.1 ± 2.5	184 ± 4	72.0 ± 2.6	63.0 ± 2.7	800m: 1.47,00 - 1.52,00
Sprinters (5)	24.9 ± 2.6	183 ± 5	78.8 ± 2.0	60.4 ± 2.2	100m: 10,9 - 11,6
STUDY II					
Middle distance runners (10)	22.0 ± 3.2	178 ± 5	66.7 ± 7.6	69.4 ± 5.1	5000m: 14.16,2 - 15.18,2
STUDY III					
Middle distance and marathon runners (22)	24.3 ± 3.7	179 ± 3.6	65.4 ± 4.2	74.2 ± 4.4	5000m: 13.28,2 - 15.32,0
STUDY IV					
Marathon runners (10)	28.0 ± 4.4	176 ± 7	61.1 ± 4.6	75.7 ± 3.6	42,2km: 2:15.02 - 2:30.25
Middle distance runners (10)	21.2 ± 2.0	181 ± 3	68.8 ± 4.6	70.4 ± 5.3	800m: 1.48,5 - 1.56,5
STUDY V					
Marathon runners (8)	31.3 ± 3.2	173 ± 5	60.4 ± 3.4	-	42,2km: 2:12:00 - 3:37:00
Keep-fit marathon runners (22)	39.0 ± 8.8	178 ± 6	73.1 ± 11.5	-	42,2km: 2:23.00 - 4:50:00
STUDY VI					
Endurance athletes (10)	23.3 ± 3.4	177 ± 6	75.4 ± 4.1	-	12min run: 3150 - 3650m

No medication was taken by the subjects, which would have been expected to affect physical performance. All procedures were performed in accordance with the Declaration of Helsinki on the use of human subjects. After provision of written and oral information regarding the possible risks and discomforts of the study all subjects gave their written consent before participation.

4.2 Study designs

The experimental designs of the present studies comprised ten different type of maximal and submaximal endurance running exercises to study acute neuromuscular, hormonal and oxidative stress responses in well trained

endurance runners. The experimental protocols and primary measurements performed in studies I-VI are summarized in table 2.

(I)

Three groups of competitive runners (6 marathon runners, 5 middle distance runners, and 5 sprinters) performed three modifications of the maximal anaerobic running test (MART) in a random order with 1 week between the tests. One week before the first MART modification the following performance determinants were measured: Maximal 20-m running speed with a flying start ($V_{20m\ max}$) on an indoor track, maximal vertical jumping height (CMJmax) on a contact mat and maximal oxygen uptake (VO_{2max}) on a treadmill.

The MART tests consisted of n sets of 1 · 20-s (= MART1), 3 · 20-s (=MART3) and 5 · 20-s (=MART5) runs on a treadmill with a 40-s recovery between the repetitions and 100-s recovery between the sets.

(II)

A group of 10 well-trained male middle distance runners first performed a pretest consisting of a velocity-incremented treadmill test to exhaustion to determine the maximal oxygen uptake (VO_{2max}) and the velocity associated with VO_{2max} (vVO_{2max}). After that, in order to compare aerobic and anaerobic energy release and muscular performance in different interval exercises, they performed two intermittent running exercises (IR₆₀ and IR₁₂₀) on a treadmill, at intervals of one week, in random order. The pretest and intermittent exercises were done at the same time of the day for each subject and were preceded by three days without strenuous exercise and a 5-km warm-up run. The IR₆₀ exercise consisted of 14 bouts of 60-second runs at vVO_{2max} with 60-seconds rest between the runs. Correspondingly, the IR₁₂₀ test consisted of 7 bouts of 120-second run at vVO_{2max} with 120-second rest between the runs.

TABLE 2 Summary of the exercise protocols and measurements during and after the experimental endurance running exercises

Exercise type	Original paper	Exercise description	Total duration	Intensity (% $v\text{VO}_{2\text{max}}$)	Primary measurements during and after the exercises
Maximal intermittent running test consisted of 20 s repetitions	I	a) $n \times 20 \text{ s}/100 \text{ s}$ recovery, progressive intensity until exhaustion	18-30 min	From 60% up to 190%	Maximal vertical jumps Blood lactate Maximal running power
		b) $n \times 3 \times 20 \text{ s}/100 \text{ s}$ recovery, progressive intensity until exhaustion	42-54 min	From 60% up to 155%	
Intermittent running exercise consisted of 1 min repetitions	II	14 x 1 min/1 min recovery	28 min	100%	Maximal vertical jumps Blood lactate Oxygen deficit
Intermittent running exercise consisted of 2 min repetitions	II	7 x 2 min/2 min recovery	28 min	100%	Maximal vertical jumps Blood lactate Oxygen deficit Maximal vertical jumps Mechanical power in half squats EMG Serum testosterone, cortisol, LH and FSH concentrations
	III, IV	10 x 2 min / 2 min recovery	40 min	100%	
Maximal $\text{VO}_{2\text{max}}$ test run	I, II, III, IV	$n \times 2 \text{ min}$, progressive intensity until exhaustion	18-24 min	From 50% up to 100%	$\text{VO}_{2\text{max}}$ Velocity associated with $\text{VO}_{2\text{max}}$ Mechanical power in half squats EMG
Continuous running exercise	III, IV	40 min constant velocity training run	40 min	80%	Maximal vertical jumps Mechanical power in half squats EMG Serum testosterone, cortisol, LH and FSH concentrations
Continuous running exercise	V	a) 31 km training run	1h 50min	70-85%	LDL oxidation Serum antioxidants Serum antioxidant capacity
		b) 42,2 km marathon race	2h 42min-5h 10min	80%	
Continuous walking exercise	VI	fast walking	6 h	35-45%	LDL oxidation Serum antioxidant concentrations Serum antioxidant capacity

Before the intermittent runs and after 4th, 8th, 12th and 14th running bout during IR₆₀ and after 2nd, 4th, 6th and 7th bout during IR₁₂₀ as well as 3, 5 and 10 min after both exercises fingertip blood samples were taken to determine blood lactate concentration and three maximal counter movement jumps within 20 seconds were performed on a contact mat (Newtest Co., Oulu, Finland) beside the treadmill to determine maximal vertical jump height. During the IRs oxygen uptake and heart rate was recorded. The fingertip blood samples were taken immediately after stopping the treadmill and the vertical jumps were performed 15-35 s after the runs.

(III, IV)

Eleven middle distance runners (MID) and eleven marathon runners (MAR) performed three different running exercises on a treadmill on three different days separated by 7 days and performed at the same time of day (in the morning at 08 00 – 10 00h after a light breakfast at 06 00h). First, the maximal oxygen uptake (VO_{2max}) and the velocity associated with VO_{2max} (vVO_{2max}) were determined in a maximal test run (MR). One week after this, two non – exhaustive exercise runs were performed in a random order at an interval of one week. These exercises were selected as representative of continuous and intermittent types of intensive running used in training: a 40-min tempo run at a velocity corresponding to 80% vVO_{2max} (TR) and a 40-min intermittent run (2 min run and 2 min rest, during which slow walking beside the treadmill was allowed) at the velocity corresponding to 100% vVO_{2max} (IR). The total running distance completed in TR was 10666 ± 600 m and 10133 ± 733 m and in IR 6666 ± 300 m and 6333 ± 367 m in MAR and MID, respectively.

On the MR day, after measuring body height and weight and after a standard 40-min pre-competitive warm-up, maximal vertical jumping height (CMJ), mechanical half squat power (P), and maximal 20-m sprinting speed ($v20m_{max}$) were determined before the MR. Thereafter, jumping tests and mechanical power tests together with recording of electromyographic (EMG) activity of leg muscles were performed before and at least 1 min after each of the three exercises. In addition, finger tip and venous blood samples for lactate and serum hormone analyses were drawn before, (at rest), after 20 min of running, immediately after (between jumping and mechanical power tests) and 10 min and 90 min after TR and IR (figure 1).

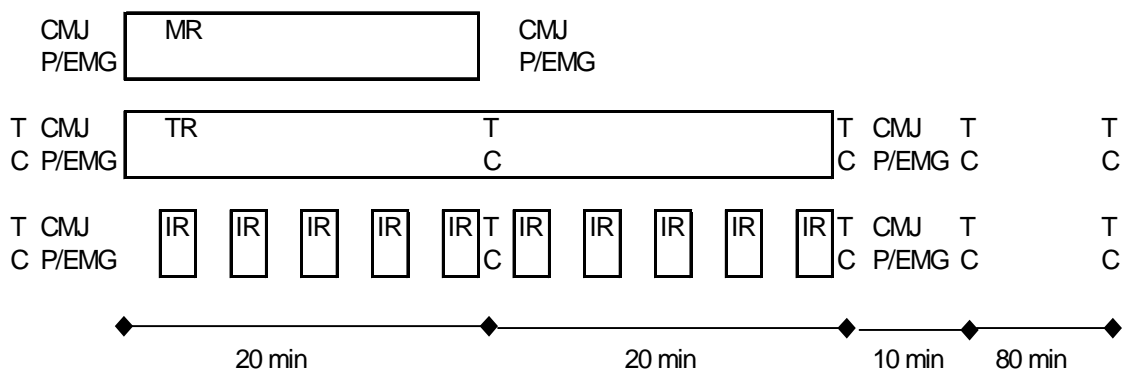


FIGURE 1 Chronological presentation of drawing the blood samples for serum testosterone (T) and cortisol (C) analyses, performing the vertical jumps for determining the vertical jumping height (CMJ), and performing the half squats for determining the mechanical power (P) and recording the electromyographic activity of leg muscles (EMG) before and after the maximal VO_{2max} test run (MR), tempo run (TR) and intermittent run (IR)

(V)

In trial I, a group of eight healthy male competitive endurance runners ran a total of 31-km test run. The test run consisted of 3 km warming up (HR 125 beats /min, blood lactate 1.6 mM), 4 km at a speed of 18 km/h (HR 162 beats /min, blood lactate 3.0 mM), 20 km at a speed of 15 km/h (HR 144 beats /min, blood lactate 2.2 mM), 4 km at a speed of 18 km/h (HR 167 beats /min, blood lactate 4.0 mM). The days preceding the test, the subjects consumed a normal Finnish diet without special vitamin supplementation or carbohydrate loading. On the day of the study, the subjects had a light mixed breakfast without caffeine drinks. During the run, the subjects were only allowed to drink water.

In trial II, a group of 22 male keep-fit marathon runners ran an international marathon race, Paavo Nurmi marathon. No special dietary restriction preceded the marathon. Eating and drinking was allowed during the run.

In both trials, antecubital venous blood samples were taken before warming up and immediately after the run. Serum was separated by centrifugation, after which it was stored at -70°C until analysed.

(VI)

Ten endurance athletes, experienced at long distance running, orienteering, and cross country skiing exercises (3 to 7 years experience), performed twice, in a randomised order, a 2-day walk exercise protocol separated by an interval of 14 days. The 2-day exercise consisted of a 6h fast walk on both consecutive days.

During the 6h walk the subjects drank $6 \text{ cl} \cdot \text{kg}^{-1}$ of water which contained either saccharose (CHO trial) or sweetened placebo (PLA trial). During the study the physical training was standardized and the subjects consumed a normal Finnish diet without any vitamin supplementation or carbohydrate loading. Laboratory and performance tests were carried out before (beginning at 07.00h) and after the 6h walk on the first and second exercise day (PRE, POST

1, POST 2 and POST 3). On both consecutive exercise days the walk exercise began at 09.30h.

4.3 Measurements and analyses

4.3.1 Maximal aerobic power test (I, II, III, IV)

For determining $\text{VO}_{2\text{max}}$ (I, II, III and IV) and $v\text{VO}_{2\text{max}}$ (II, III and IV) the subjects performed a maximal run, which consisted of a velocity-incremented continuous treadmill run at 1° slope until exhaustion. The duration of each stage of the test was two minutes; the initial 2-min stage was run at a velocity of $10 \text{ km} \cdot \text{h}^{-1}$ and thereafter the velocity was increased by $1 \text{ km} \cdot \text{h}^{-1}$ for each consecutive stage until exhaustion. Oxygen uptake during the test was measured by an automated Oxygon Sigma gas analyzer (Mijnhard, the Netherlands) and $\text{VO}_{2\text{max}}$ was defined as the highest 60-s VO_2 during the test. V_{max} was the treadmill velocity at which the subject first attained $\text{VO}_{2\text{max}}$ (Billat et al. 1995). This was either the velocity of the last 2-min stage or the previous 2-min velocity, if VO_2 was higher at this stage. If the value of VO_2 at the two last stages was the same, the mean of these two velocities was accepted as V_{max} (Lindsay et al. 1996). The reliability of this protocol has been confirmed by Billat et al. (1994a,b, 1996).

Fingertip blood samples were taken immediately after each stage and 5 minutes after exhaustion. The blood lactate concentrations (bLa) in these samples were analyzed by a standard enzymatic method (Boehringer, Mannheim, Germany). The highest bLa value was recorded as the bLa_{max} .

4.3.2 Maximal anaerobic power test (I)

Maximal and submaximal running power was determined in three different maximal anaerobic running test (MART) modifications. In each MART modification, the first set of the 20-s runs was performed at $3,0 \text{ m} \cdot \text{s}^{-1}$ with a 4° slope. Thereafter, the speed was increased gradually by $0,38 \text{ m} \cdot \text{s}^{-1}$ for each set of the runs until exhaustion and the inclination was kept constant at 4° . The submaximal and maximal running power in each MART test was expressed as the oxygen demand of the runs using the formula of American College of Sports Medicine (1): $\text{VO}_2 (\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = 12 \cdot v (\text{m} \cdot \text{s}^{-1}) + 54 \cdot g(\text{frac}) \cdot v(\text{m} \cdot \text{s}^{-1}) + 3,5$ where VO_2 is oxygen demand, v is the speed of the treadmill and g is the slope of the treadmill expressed as the tangent of the angle with the horizontal. A 5-s acceleration phase was not included in the running time.

To determine blood lactate concentration, fingertip blood samples were taken at rest, 40 s after each set of runs and 5 and 10 min after exhaustion.

The maximal running power (P_{\max}) was calculated from the last completed set of 20-s runs and from the exhaustion time of the following faster set of the runs (Rusko et al. 1993).

4.3.3 Determining accumulated oxygen deficit (II)

For determining the accumulated oxygen deficit (AOD), oxygen uptake and the related variables were measured by the same method as in the maximal aerobic power test. Breath-by-breath VO_2 data were averaged in 15-s intervals and the highest 15-s values of each exercise bout ($VO_{2\text{peak}}$) was recorded for further calculations.

Also, the heart rate (HR) of the subjects was monitored during the runs by the same method as in the maximal aerobic power test and the highest 5-s values of each exercise bout were recorded as peak exercise heart rates (HR_{peak}).

The relative aerobic and anaerobic energy release of the IR-exercises was calculated by the accumulated oxygen deficit method (Medbo et al. 1988, Medbo and Tabata 1989, Tabata et al. 1997). The individual oxygen demand during the IRs at V_{\max} was extrapolated from the linear regression function of the oxygen demand versus running velocity relationships established during the pretest (Gastin et al. 1995, Tabata et al. 1997). The oxygen deficit of individual single running bouts was the difference between the oxygen demand and the oxygen uptake during each run. Finally, the accumulated oxygen deficit (AOD) was calculated as the average of the individual oxygen deficits.

4.3.4 Maximal vertical jump tests (I, II, III)

For determination of maximal vertical jumping height (CMJ) three maximal counter movement jumps on a contact mat (Newtest Co., Oulu, Finland) were performed within 20 seconds. The jump height was calculated in meters according to the formula: $H = g \cdot t^2 \cdot 8^{-1}$, where t is the recorded flight time in seconds and g is the acceleration due to gravity ($9.81 \text{ m} \cdot \text{s}^{-2}$). To avoid immeasurable work, horizontal and lateral displacements were minimized, and the hands were kept on the hips throughout the test. During the CMJ the angular displacement of the knee was standardized so that the subjects were required to bend their knees to approximately 90° . The average of the two highest jumps was taken as the CMJ.

4.3.5 Mechanical power and EMG in half squats (III)

In study III, approximately 30 s after the CMJ the subjects started maximal vertical half squat exercises on a Smith machine (guided horizontal barbell) with additional loads of 35% of the subject's one-repetition maximum additional load (1RM) with both legs. Ten repetitions were made at intervals of 3 s between the repetitions. Before the $VO_{2\max}$ test a set of ten repetitions was performed twice; the recovery time allowed between the sets was 2 min. Since the results of the first half squat performance in each set usually are rather

variable, the averages of the last nine repetitions were used for statistical analysis, as has been recommended by Bosco et al. (1995, 2000). During the test, vertical displacement of the load was monitored with a sensor (encoder) device (Muscle Lab - Bosco System, Ergotest Technology, Langensund, Norway) connected to a PC. When the loads were moved by the subjects, a signal was transmitted by the sensor at every 3 mm of displacement and it was possible to calculate the average mechanical power (P) of each repetition from the start of the half squat movement (the moment when the velocity started to increase) until the end of the half squat movement (the moment when the velocity reached zero again). Since it has been shown, that P is the most sensitive parameter among all the mechanical variables that can be studied with this system, the average mechanical power was used for further calculations and statistical analysis (Bosco et al. 1995).

To determine the power and EMG changes during the set of the ten half squats, a measure of the relative fatigue / change ($\Delta\%$) was calculated using the formula: $\Delta\% = (R7+R8+R9 - R2+R3+R4) / (R7+R8+R9) * 100$, where R is the result of a single repetition.

EMG activity from the vastus medialis, vastus lateralis, lateral part of gastrocnemius and biceps femoris muscles of the right lower extremity were recorded during the half squat performances; bipolar surface electrodes (inter-electrode distance 1.2 cm) fixed longitudinally over the marked muscle belly were used. An amplifier (gain \times 600, input impedance 2 G Ω , common-mode rejection ratio 100 dB, band-pass filter 6-1500 Hz; Biochip Grenoble, France) was used. The MuscleLab encoder converted the amplified EMG raw signal to an average (root mean-square) (rms) signal via its built-in hardware circuit network (frequency response 450 kHz, averaging constant 100 ms, total error \pm 0.5%). The EMGrms was expressed as a function of time (μ V). Since the EMGrms signals were used in association with the biomechanical parameters measured with MuscleLab, they were sampled simultaneously at 100 Hz. The subjects wore a skin suit to prevent the cables from swinging and causing movement artifacts. A personal computer was used to collect and store the data. The EMGrms values from the same nine repetitions as in the case of mechanical P recordings and from each of the four muscles, the summed EMGrms of the leg extensors (vastus medialis and vastus lateralis) and the summed EMGrms of all the four muscles were used for statistical analysis (Bosco and Viitasalo 1982, Viitasalo and Bosco 1992).

The means (and SD) of the mechanical P in the two successive half squat trials performed before MR were 825 (143) W in trial 1 and 820 (126) W in trial 2. The corresponding EMGs (sum of the four muscles) were 786 (159) μ V and 775 (151) μ V. The reproducibility of both variables was high, $r = 0.96$ ($P < 0.001$).

4.3.6 Serum hormone analyses (IV)

Serum testosterone (S-testosterone) and cortisol (S-cortisol) were measured by RIA kits purchased from Farnos Diagnostica (Turku and Oulunsalo, Finland). Serum luteinizing hormone (S-LH) and follicle-stimulating hormone (S-FSH) were measured by time-resolved immunofluorometry (Delfia hLH for LH and Delfia hFSH for FSH) provided by Wallac Oy (Turku, Finland). The intra- and interassay coefficients of variation of the individual RIA and immunofluorometric assay methods were below 5% and 9% within the reference ranges, respectively. All samples from the same subjects were analyzed in duplicate in the same assay run. The reference ranges reported for men were as follows: testosterone, 9-38 nmol/L; LH, 1.0-8.4 IU/L; FSH, 1.0-10.5 IU/L; and cortisol, 150-650 nmol/L at 0800-0900 h.

4.3.7 Maximal and submaximal 1000m running performance (VI)

The performance measurements in study VI consisted of three 1000 m runs on a 200 m indoor track with 5 min of recovery between the repetitions. The velocities in the first and second 1000 m runs were 12 and 15 km · h⁻¹, respectively, and the velocity was controlled by an electric light - system (Smedic Corp., Heinola, Finland). The third 1000m run was to be run as fast as possible and the mean velocity ($V_{\max 1000m}$) was used for further calculations. Fingertip blood samples were taken two minutes after each run and the blood lactate concentrations (bLa) were analyzed by a standard enzymatic method (Boehringer, Mannheim, Germany).

4.3.8 Lipid and lipoprotein analyses (V, VI)

Serum total and HDL cholesterol and triglyceride concentrations were determined at the laboratory of the Turku University Central Hospital on an automatic enzymatic analyzer (Hitachi 917 automatic analyser, Hitachi Ltd, Tokyo, Japan). The following reagents were used: CHOD-PAP for Cholesterol, HDL-C plus for HDL and GPO-PAP for triglycerides (Boehringer Mannheim, Germany).

Baseline LDL conjugated dienes (LDL-BDC) were determined by a validated method (Ahotupa et al. 1996, 1998, 1999), after serum low density lipoproteins had been isolated by heparin precipitation (Weiland and Seidel 1983). Lipids were extracted from the LDL samples (100 µl) with chloroform-methanol (2:1), dried under nitrogen, then redissolved in cyclohexane and analyzed spectrophotometrically at 234 nm. For LDL baseline conjugated dienes, the CV for the within-assay precision (20 determinations from the same sample) was 4.4%; the CV for the between-assay precision was 4.5% over a 3-month period (Ahotupa et al. 1996).

The antioxidant potential of serum (S-TRAP) and LDL samples (LDL-TRAP) was assessed in vitro by their potency to resist ABAP-induced peroxidation (total peroxy radical trapping antioxidant potential) (Ahotupa

1996). The peroxy radical trapping capacity was defined by the half-peak time point. Trolox served as a standard radical scavenger. For antioxidant potential, the CV for the within-assay precision (20 determinations of the same sample) was 8.1%; the CV for the between-assay precision was 8.7% over a 3-month period (Ahotupa et al. 1996).

Serum concentrations of α -tocopherol, γ -tocopherol, β -carotene, and ubiquinol-10 were analyzed by standard HPLC procedures with UV-detection (Milne et al. 1986, Takada et al. 1985). In the case of α -tocopherol and γ -tocopherol, the ratio to serum lipids (cholesterol + triglyceride) was also calculated (Thurnam et al. 1986).

4.4 Statistical methods

Statistical analyses were done by the SPSS for windows Statistical Software, version 9.0. Means, standard deviations, and coefficients of correlation were calculated by standard methods. In study I, comparisons between the different groups and different test modifications were made by using a one-way analysis of variance and Scheffe method. In study II, the statistical differences between the means of the two exercise protocols were tested by multiple analysis of variance (MANOVA) and in case of significant difference by univariate F-tests.

For all performance, neuromuscular and hormone variables in studies III and IV, a two-way analysis of variance (trial \times time) with repeated measurements was used to detect significant differences across the different exercises. In the cases of significant exercise \times time and group \times time (study IV) response interactions, differences between the exercises and between the groups (study IV) were analysed using paired and unpaired t-tests. In study III, for analysing the differences between average values of repetitions 2-4 and 7-9 during the sets of ten half squat repetitions, matched-paired t-tests were performed.

In study V, the statistical differences between the samples taken before and after exercises, in both trials, were analysed by the paired Student's t-test.

In study VI, a one-way ANOVA with four repeated measures over the exercise time was used to examine the time responses within the both trials. In case of a significant time effect within the trial, the post-exercise samples were compared with the pre-exercise sample by using a paired t-test. A two-way ANOVA with four repeated measures over the exercise time and two repeated measures over the treatment was used to examine the differences between the treatments (time \times treatment interactions). Statistical significance for all analyses and comparisons was accepted at $P < 0.05$.

5 RESULTS

5.1 Blood lactate accumulation and oxygen uptake in endurance running exercises (I, II, III, IV, V)

5.1.1 Blood lactate responses to intermittent and continuous running (I, II, III, IV, V)

After MART₁ (n x 1 x 20-s / 100s recovery) middle distance runners and sprinters had more than two times higher peak blood lactate than marathon runners (12.9 ± 0.6 and 14.8 ± 1.8 vs. 6.3 ± 1.8 mM, $P < 0.001$). In marathon runners, but not in middle distance runners or sprinters, the peak blood lactate was higher the more runs each MART set contained ($P < 0.001$). (Table 3) (I)

The peak blood lactate concentration after the intermittent run of 7 x 120-s / 120-s recovery (IR₁₂₀) was higher than after the intermittent run of 14 x 60-s / 60-s recovery (IR₆₀) (8.8 ± 3.6 mM vs. 4.8 ± 1.1 mM, $P < 0.01$) and almost the same as after the VO_{2max} test run (8.0 ± 2.4) in the well-trained middle distance runners (table 3). (II)

In study III, the VO_{2max} test run raised blood lactate concentration to 10.5 ± 2.1 mM in the well-trained middle distance runners. After TR and IR it was significantly ($P < 0.001$ in both) lower (3.8 ± 1.6 and 6.6 ± 1.7 mM, respectively), and lower in TR compared to IR ($P < 0.001$) (table 3).

In study IV, blood lactate concentrations were lower after the 40-min TR (3.6 ± 0.6 mM and 4.4 ± 2.3 mM) than and after the 40-min IR (7.2 ± 0.3 mM and 6.7 ± 1.5 mM) in marathon runners ($P < 0.001$) and middle distance runners ($P < 0.05$), respectively, but no significant differences between the groups were found. Both training sessions were rated as hard or very hard on the Borg scale. MAR rated IR harder than TR (17.0 ± 0.5 vs. 16.1 ± 0.7 , $P < 0.05$) whereas MID rated TR harder than IR (17.7 ± 0.7 vs. 16.4 ± 1.0 , $P < 0.01$).

During the 31-km test run (3 km warming up, 4 km at a speed of 18 km /h, 20 km at a speed of 15 km /h, 4 km at a speed of 18 km /h) the mean heart rate and blood lactate concentration in well-trained long distance runners were as follows: after 3 km, 125 beats /min and 1.6 mM; after 7 km, 162 beats /min

and 3.0 mM; after 27 km, 144 beats /min and 2.2 mM; after 31 km, 167 beats /min and 4.0 mM, respectively. (V)

TABLE 3 Post-exercise blood lactate levels and counter movement jump (CMJ) responses to different types of running exercises

Exercise (article)	Subjects (n)	Running intensity (% vVO _{2max})	Duration of running (total time)	Blood lactate post (mM)	CMJ (cm) pre (timing)	CMJ response
VO _{2max} test (III)	MDR and LDR (20)	from 50 to 100%	18-24 min (18-24 min)	10.5 ± 2.1	33.2 ± 5.0 (b.e.)	5% increase **
Tempo run (III)	MDR and LDR (20)	80 %	40 min (40 min)	3.8 ± 1.6	30.9 ± 5.7 (b.e.)	14% increase **
Intermittent run 10 x 2 min (III)	MDR and LDR (20)	100 %	20 min (40 min)	6.6 ± 1.7	31.3 ± 5.5 (b.e.)	9% increase **
MART ₁ n x 1 x 20-s (I)	MAR (6)	from 60 to 130%	3-4 min (18-22 min)	6.3 ± 1.8	28.0 ± 0.9 (after 1 st rep)	no significant changes
MART ₁ n x 1 x 20-s (I)	MDR (5)	from 65 to 170%	4-5 min (26-30 min)	12.9 ± 0.6	41.9 ± 2.5 (after 1 st rep)	no significant changes
MART ₁ n x 1 x 20-s (I)	SPR (5)	from 70 to 190%	4-5 min (24-30 min)	14.8 ± 1.8	50.2 ± 3.2 (after 1 st rep)	10% decrease **
MART ₃ n x 3 x 20-s (I)	MAR (6)	from 60 to 130%	9-10 min (42-46 min)	8.5 ± 1.6	28.2 ± 0.8 (after 1 st set)	6% increase **
MART ₃ n x 3 x 20-s (I)	MDR (5)	from 65 to 145%	10-12 min (46-54 min)	12.7 ± 1.0	42.1 ± 2.0 (after 1 st set)	8% decrease **
MART ₃ n x 3 x 20-s (I)	SPR (5)	from 70 to 155%	9-11 min (40-52 min)	13.0 ± 2.8	51.5 ± 3.0 (after 1 st set)	no significant changes
Intermittent run 14 x 1 min (II)	MDR (10)	100 %	14 min (28 min)	4.8 ± 1.1	38.1 ± 3.4 (after 7min)	no significant changes
Intermittent run 7 x 2min (II)	MDR (10)	100 %	14 min (28 min)	8.8 ± 3.6	37.7 ± 4.1 (after 7min)	no significant changes

** P < 0.01

SPR = sprint runners, MDR = middle distance runners, LDR = long distance runners, MAR = marathon runners.

b.e. = before exercise, rep = repetition

5.1.2 VO_2 responses to intermittent running at the velocity associated with $\text{VO}_{2\text{max}}$ (II)

Figure 2 shows the oxygen demand and the means of oxygen uptake during IR_{60} and IR_{120} exercises. The peak oxygen uptake in IR_{120} increased to the level of $\text{VO}_{2\text{max}}$ from the second repetition while the corresponding oxygen uptakes in IR_{60} remained at a 12% lower level ($P < 0.001$).

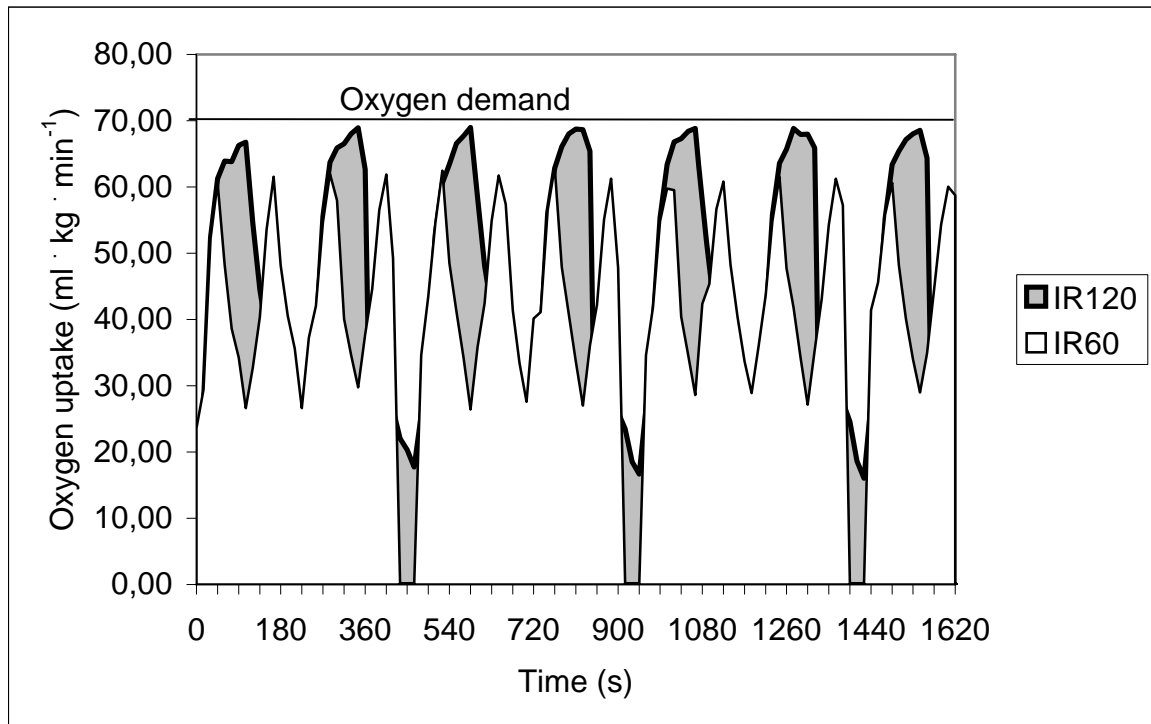


FIGURE 2 The oxygen demand and the mean values of oxygen uptake during IR_{60} and IR_{120} exercises ($n=10$).

Although the oxygen demand in both exercises was similar, the relative aerobic energy release in IR_{120} was significantly higher than in IR_{60} ($P < 0.001$). On the other hand, during the first 60 seconds in IR_{120} , the relative aerobic energy release was lower than in IR_{60} ($P < 0.001$). (Figure 3)

The total 28-minute VO_2 in both intermittent exercises was similar while the sum VO_2 during the runs (14-min) was lower in IR_{60} than in IR_{120} (678 ± 53.0 vs. 787 ± 58.0 $\text{ml} \cdot \text{kg}^{-1}$, $P < 0.001$) and the sum VO_2 during the recovery (14-min) was correspondingly higher (538 ± 31.0 vs. 432 ± 30.5 $\text{ml} \cdot \text{kg}^{-1}$, $P < 0.001$).

The training intensity indicators bLa , $\text{VO}_{2\text{peak}}$ and HR_{peak} did not correlate with each other either with regard to absolute or relative values within the two intermittent exercises IR_{60} and IR_{120} .

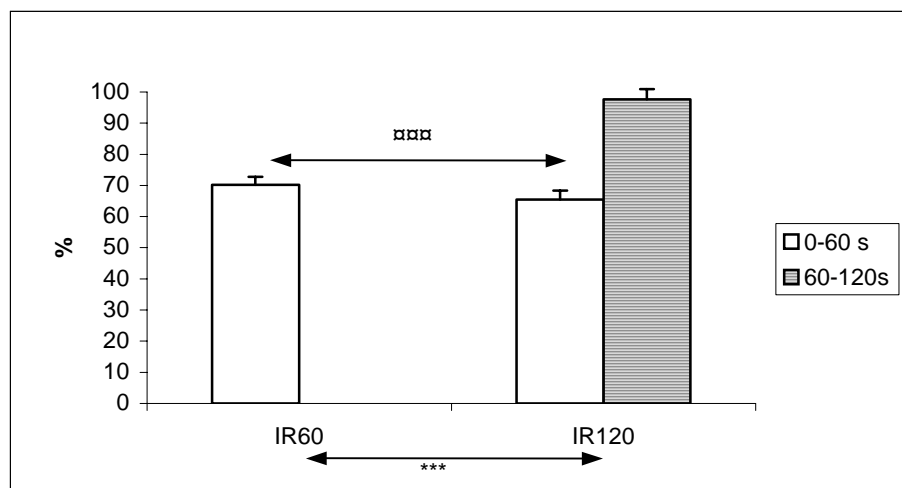


FIGURE 3 The relative aerobic energy release (mean \pm SD) measured by accumulated oxygen deficit method in IR₆₀ and IR₁₂₀ exercises, n=10.
 ***P < 0.001; difference between the relative aerobic energy in IR₆₀ and IR₁₂₀.
 □□□P < 0.001; difference between the relative aerobic energy release during the first 60 seconds in IR₆₀ and IR₁₂₀.

5.2 Effects of maximal and submaximal running exercises on muscle power and muscle activation (I, II, III)

5.2.1 Exercise-induced acute changes in maximal vertical jumping performance (I, II, III)

The resting level of maximal vertical jump height (CMJ) varied greatly between the runners specialized to different running events. In study I, it was less than 30 cm in marathon runners (MAR) and more than 10 and 20 cm higher in middle distance runners (MDR) and sprinters (SPR), respectively (ANOVA group effect P=0.001). The CMJ responses to repeated 20-s runs at increasing intensity until exhaustion (MART) were different in MDR and SPR compared to MAR. A significant increase in CMJ during MART₁ (n x 1 x 20-s / 100-s recovery) and MART₃ (n x 3 x 20-s / 40-s recovery between the repetitions and 100-s recovery between the sets) was observed in MAR (P<0.01, in both) while a significant decrease was found during MART₁ in SPR (P<0.01) and during MART₃ in MDR (P<0.01). (Table 3).

In study III, three different running exercises caused CMJ to increase in well-trained middle- and long distance runners. After an exhaustive VO_{2max} test run (MR), a 40-min tempo run at an intensity of 80% vVO_{2max} (TR) and an intermittent run of 10 x 2 min with 2 min recovery at an intensity of 100% vVO_{2max} (IR) CMJ increased by 5%, 14% and 9% (P<0.001, in all, respectively (table 3). In each of these exercises, the exercise-induced improvement in CMJ correlated negatively with the resting level of CMJ (r = -.63, r = -.51 and r = -.54, P<0.01, n = 20, respectively) and with the maximal 20-m sprinting speed (r = -

.57, $r = -.58$ and $r = -.56$, $P < 0.01$, $n = 20$, respectively). After MR the increase in CMJ correlated negatively with the increase in blood lactate level ($r = -0.59$, $n = 22$, $P < 0.01$), while after IR the correlation between CMJ and blood lactate increase was positive ($r = 0.62$, $n = 20$, $P < 0.01$) and after TR no significant correlation was found. In addition to this, in MR the improvement in CMJ correlated positively with the training volume ($P < 0.001$) and vVO_{2max} ($P < 0.05$), and negatively with the half squat_{1RM} ($P < 0.01$).

In the study II, during the 28-min intermittent runs of 14 x 1 min with 1 min recovery and 7 x 2 min with 2 min recovery at an intensity of 100% vVO_{2max} , CMJ was unchanged in well-trained middle distance runners. However, in these cases, the resting level of CMJ was not measured. The first CMJ measurement was done not before 7 minutes of exercising (table 3).

5.2.2 Effects of intermittent and continuous runs on muscle activation and mechanical power in half squats (III)

In study III, each of the three exercises, MR, TR and IR, led to a significant acute decrease in the EMGrms of the knee extensor muscles (ANOVA time effect without trial * time interaction, $P = 0.001$ for vastus medialis and vastus lateralis) and the sum of the four leg muscles' EMGrms (ANOVA trial * time interaction, $P = 0.021$) registered in a set of ten half squats with the 35 % 1 RM additional load. At the same time mechanical P was improved (ANOVA time effect without trial * time interaction, $P = 0.001$) and the ratio between EMGrms and P decreased (ANOVA trial * time interaction, $P = 0.009$). The reduction after TR (20.1 ± 9.5 %) was significantly greater ($P < 0.05$) than the relative decrease after MR (9.0 ± 8.9 %). The corresponding decrease after IR (15.0 ± 8.3 %) did not differ from the decrease after MR. (Table 4).

The decrease in EMGrms was not related to the change in P after TR. However, in the case of MR and IR, the greater was the increase in P the lesser the decrease in EMGrms ($r = 0.50$ and $r = 0.47$, $n = 16$, $P < 0.05$, respectively).

TABLE 4 Responses of EMGrms and mechanical power in half squats (P) to the maximal $\text{VO}_{2\text{max}}$ test run (MR), 40 min tempo run at an intensity of 80% $\text{vVO}_{2\text{max}}$ (TR) and intermittent run 10 x 2 min / 2 min recovery at an intensity of 100% $\text{vVO}_{2\text{max}}$ (IR) in middle and long distance runners.

Exercise	Response EMGrms ga (n=16)	Response EMGrms vm (n=16)	Response EMGrms vl (n=16)	Response EMGrms bf (n=16)	Response EMGrms S (n=16)	Response P (n=20)	Response EMGrms:P (n=16)
MR	no significant changes	11% decrease ***	11% decrease ***	no significant changes	8 % decrease **	2% increase *	9 % decrease **
TR	no significant changes	21% decrease ***	23% decrease ***	no significant changes	17 % decrease ***	4 % increase **	20 % decrease ***
IR	no significant changes	11% decrease **	17% decrease ***	no significant changes	12 % decrease ***	3 % increase **	15 % decrease ***

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

ga = gastrocnemius

vm = vastus medialis

vl = vastus lateralis

bf = biceps femoris

EMGrms S = EMGrms ga + EMGrms vm + EMGrms vl + EMGrms bf

In addition to the changes in mean mechanical power and EMGrms in the set of the ten half squat repetitions, some intra-set changes were also found in study III. In the pre-exercise measurements, the half squat power in the single repetitions remained at the same level during the whole set of the ten repetitions (a non-significant change in $\Delta P\%$) while $\Delta \text{EMGrms}\%$ and the $\Delta \text{EMGrms:P}\%$ increased by 4-6 %-units ($P < 0.05$). After the exhaustive MR $\Delta P\%$ and $\Delta \text{EMGrms}\%$ decreased during the set of the ten repetitions by 3 and 4.5 %-units ($P < 0.05$ for both). Corresponding changes were not found after the non-exhaustive TR and IR exercise. (Figures 4 a-c).

The exercise-induced acute improvement in the half squat power performance was related differently to the maximal running performance of the subjects in TR compared to IR. In TR the change in mechanical P correlated positively with $\text{vVO}_{2\text{max}}$ ($r = .43$, $P < 0.05$, $n = 20$), while in IR the correlation was negative ($r = -.43$, $P < 0.05$, $n = 20$).

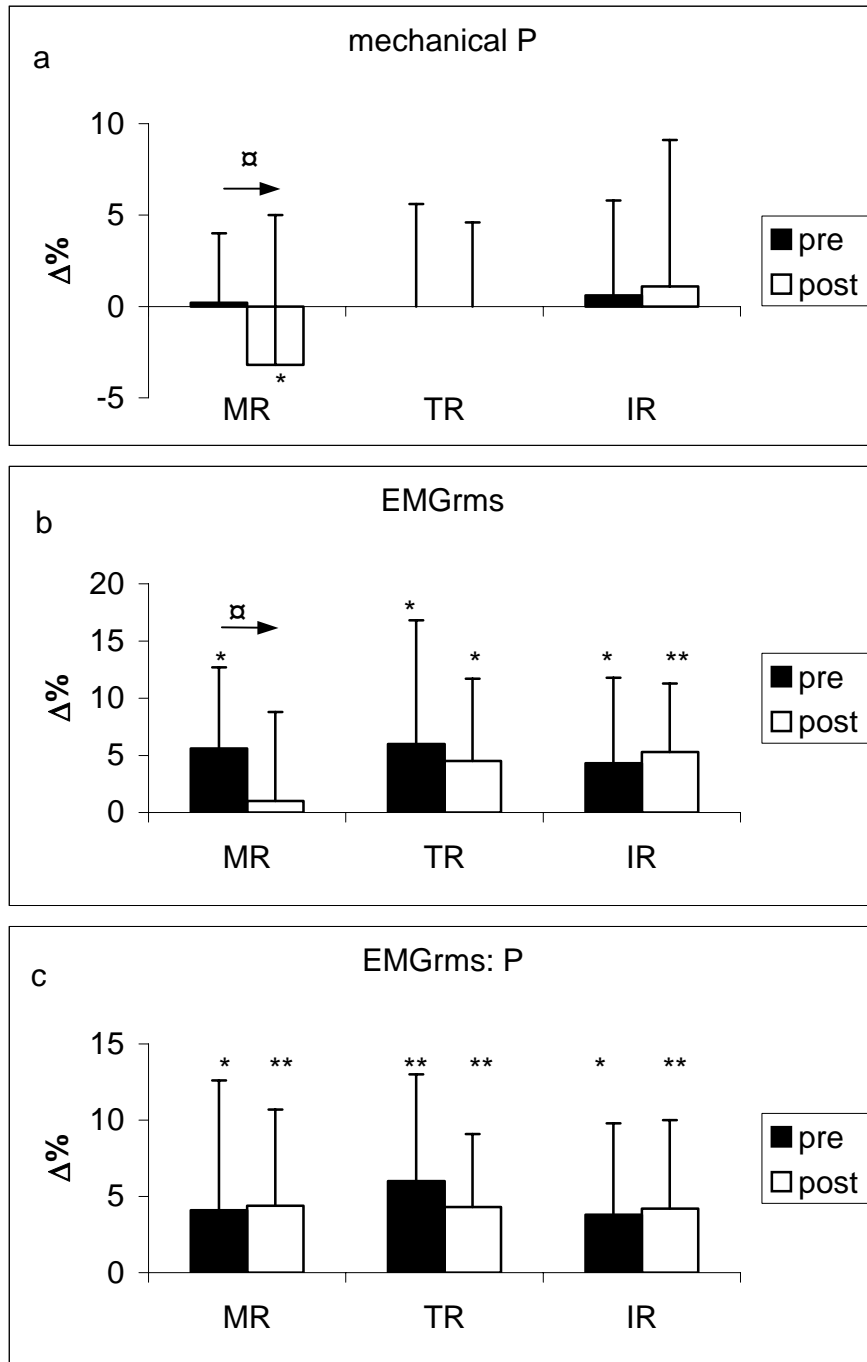


FIGURE 4 Intra-set changes ($\Delta\%$) of the mechanical P (a), EMGrms (b), and EMGrms: P (c). $\Delta\% = (R7+R8+R9 - R2+R3+R4) / (R7+R8+R9) * 100$, where R is the result of a single repetition.

*P < 0.05 and **P < 0.01; difference between R7+R8+R9 and R2+R3+R4

α P < 0.05; difference between pre-exercise and post-exercise measurements

5.2.3 Maximal anaerobic running power in different intermittent running test modifications (I)

In study I, the anaerobic running power (P_{\max}) in the maximal anaerobic running power test (MART) changed differently between MAR, MID and SPR when the number of repetitions in a set was increased. In MART₁ (n x 1 x 20-s / 100-s recovery between the sets) marathon runners had a lower maximal anaerobic running power than middle distance runners or sprinters (97.8 ± 5.4 vs. 121.0 ± 4.4 vs. 119.2 ± 5.4 ml · kg⁻¹ · min⁻¹, $P < 0.001$) while in MART₃ (n x 3 x 20-s / 40-s recovery between the repetitions and 100-s recovery between the sets) no significant differences were found between the groups. In marathon runners, no difference was found in the anaerobic running power between MART₁ and MART₃, while in middle distance runners and sprinters it was higher in MART₁ than in MART₃ ($P < 0.001$) (figure 5).

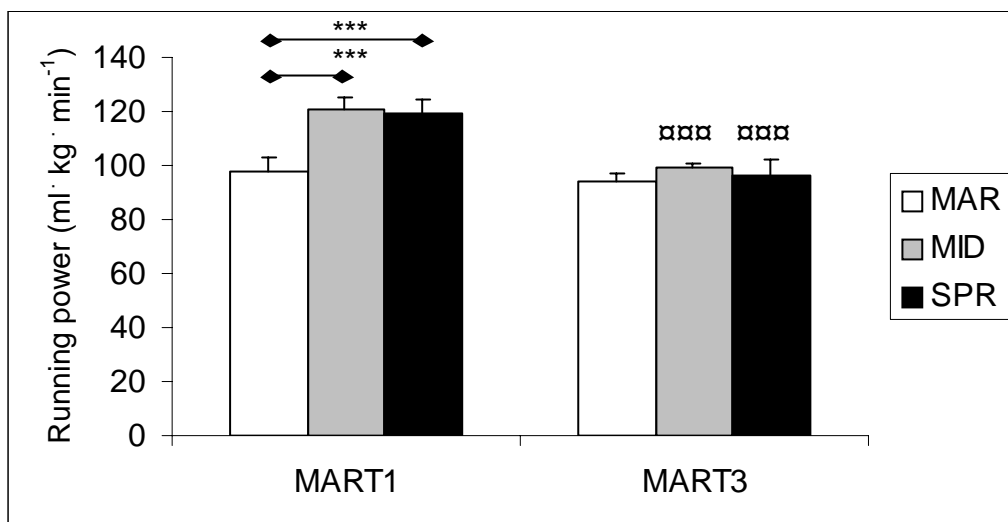


FIGURE 5 Anaerobic running power in MART₁ (n x 1 x 20-s / 100-s recovery between the sets) and MART₃ (n x 3 x 20-s / 40-s recovery between the repetitions and 100-s recovery between the sets) in marathon runners (MAR), middle distance runners (MID), and sprinters (SPR).
 *** $P < 0.001$; Difference between the groups
 □□□ $P < 0.001$; Difference between MART₁ and MART₃

Middle distance runners and sprinters were able to do significantly more work above their maximal aerobic running power ($VO_{2\max}$) than marathon runners in both MART₁ and MART₃. In MART₁, the power difference between the maximal anaerobic power and $VO_{2\max}$ was over two times greater in middle distance runners and sprinters compared to marathon runners (50.9 ± 3.2 and 57.0 ± 6.0 vs. 23.8 ± 6.1 ml · kg⁻¹ · min⁻¹, $P < 0.001$ in both, respectively). In each group, the power difference was higher the smaller the number of runs in each set ($P < 0.01$).

In MART₁, maximal anaerobic running power correlated positively with the maximal 20-m running speed ($r = .87$, $P < 0.001$, $n = 16$) and maximal vertical jump height ($r = .72$, $P < 0.01$, $n = 16$) and negatively with $VO_{2\max}$ ($r = -.82$, $P < 0.01$, $n = 16$). In MART₃, no significant correlations were found. (I)

5.2.4 Acute effects of 6 h low intensity exercising on submaximal and maximal 1000 m running (VI)

The 6h low intensity walking exercise caused maximal 1000-m running velocity ($V_{\max 1000m}$) to decrease from 5.08 ± 0.35 to 4.88 ± 0.37 $m \cdot s^{-1}$ ($P < 0.01$). Correspondingly, $bLa_{\max 1000m}$ decreased from 11.83 ± 1.91 to 9.36 ± 2.82 mM ($P < 0.05$). Thereafter, in the second exercise day both $V_{\max 1000m}$ and $bLa_{\max 1000m}$ remained unchanged. The blood lactate concentration measured after the submaximal 1000-m runs was not affected by the 6-h walking either during the first or second exercise day.

5.3 Acute serum hormone responses to intermittent and continuous running exercises (IV)

5.3.1 Differences in cortisol and testosterone responses between middle distance and marathon runners (IV)

In study IV, both a 40-min “tempo run” (TR) and a 40-min intermittent run (IR) caused serum testosterone and cortisol to increase significantly in the well-trained middle distance (MID) and marathon runners (MAR). No changes were found in serum LH or FSH concentrations. S-testosterone response to IR and s-cortisol response to TR was different between MID and MAR (ANOVA time x group interaction $P = 0.046$ and $P = 0.016$, respectively).

After IR s-testosterone increased from 23.8 ± 9.2 nmol/l up to 29.5 ± 10.5 nmol/l in MAR ($P < 0.001$) and from 23.7 ± 5.5 nmol/l up to 35.6 ± 10.5 nmol/l in MID ($P < 0.001$) (table 5). The IR-induced s-testosterone increase was the higher the higher blood lactate concentration the runner was able to achieve after the maximal running test ($r = 0.46$, $P < 0.05$, $n = 20$). 90 min after the end of IR, s-testosterone decreased back to the pre-exercise level in MID (20.0 ± 4.6 nmol/l) and below the pre-exercise level in MAR (17.7 ± 6.2 nmol/l, $P < 0.01$) (table 5). After TR, s-testosterone increased similarly in MAR and MID (from 20.7 ± 8.2 nmol/l up to 30.1 ± 10.9 nmol/l, $P < 0.001$ and from 25.1 ± 7.5 nmol/l up to 34.9 ± 10.0 nmol/l, $P < 0.001$, respectively) and decreased back to the pre-exercise level in 90 min (table 5).

S-cortisol increased in TR from the pre-exercise level 366 ± 103 nmol/l up to 487 ± 119 nmol/l in MAR ($P < 0.001$) and from 371 ± 98 nmol/l up to 628 ± 110 nmol/l in MID ($P < 0.001$) (table 5). The increase was higher the lower the runner's $VO_{2\max}$ ($r = -0.62$, $P < 0.01$, $n = 20$) and the faster the runner in 20-m

running test ($r = 0.45$, $P < 0.05$, $n = 20$) and the higher his maximal vertical jump ($r = 0.47$, $P < 0.05$, $n = 20$). Further, it increased more as the running training the runner had performed during the last year before the study decreased ($r = -0.46$, $P < 0.05$, $n = 20$). S-cortisol decreased back to the pre-exercise level 90 min after the end of TR in MAR (378 ± 85 nmol/l), but was still elevated in MID (468 ± 91 nmol/l, $P < 0.01$) (table 5). After IR, s-cortisol increased similarly in MAR and MID (from 397 ± 59 nmol/l up to 561 ± 85 nmol/l, $P < 0.001$ and from 366 ± 82 nmol/l up to 498 ± 151 nmol/l, $P < 0.001$, respectively). It decreased back to the pre-exercise level in 90 min in both groups. (Table 5).

The molar ratio of testosterone / cortisol behaved differently between the two exercises and the two groups (ANOVA time x exercise x group interaction, $P = 0.047$). After 20 min of exercising in TR it was elevated in marathon runners ($P < 0.01$) but not in middle distance runners. The increase after TR was higher the higher runner's VO_{2max} ($r = 0.57$, $P < 0.01$, $n = 20$).

5.3.2 Time course in exercise-induced serum cortisol and testosterone changes (IV)

Serum cortisol concentration, expressed as a mean of both groups ($n = 20$), behaved differently between the two exercises (time x exercise interaction, $P = 0.008$). After the first 20 min of exercising it increased by 17% in IR ($P < 0.05$) but was unchanged in TR. After 40 minutes of exercising, it was elevated by 30% in IR ($P < 0.001$) and by 23% in TR ($P < 0.01$). Ten minutes after the end of exercise, s-cortisol levels still increased and were 39% and 51% and above the pre-exercise level in IR and TR ($P < 0.001$ in both), respectively. In the case of TR, 90 minutes after the end of exercise it was still 16% ($P < 0.05$) higher than the pre-exercise level, but in the case of IR s-cortisol decreased back to pre-exercise level by 90 minutes. At the same, s-testosterone behaved similarly during the both exercises. The only difference was found 90 min after the end of exercise when s-testosterone decreased back to the pre-exercise level in the case of TR, but 20% under the pre-exercise level in the case of IR ($P < 0.001$).

TABLE 5 Acute hormonal responses to continuous running (TR = 40 min continuous run) and intermittent running (IR = intermittent run, 10 x 2 min with 2 min recovery) in marathon (MAR) and middle distance (MID) runners

	TR		IR	
Running intensity (% vVO ₂ max)	80%		100%	
Duration of running (total time)	40 min (40 min)		20 min (40 min)	
Subjects (n)	MAR (10)	MID (10)	MAR (10)	MID (10)
Testosterone response	45% increase 1 min after E ^{***}	39% increase 1 min after E ^{***}	24% increase 1 min after E ^{***} 26% decrease 90 min after E	50% increase 1 min after E ^{***}
LH response	no sig. changes	no sig. changes	no sig. changes	no sig. changes
FSH response	no sig. changes	no sig. changes	no sig. changes	no sig. changes
Cortisol response	33% increase 10 min after E ^{***}	69% increase 10 min after E ^{***} 26% increase 90 min after E ^{**}	41% increase 10 min after E ^{***}	36% increase 10 min after E ^{***}

E = exercise

Difference between MAR and MID; [□]P < 0.05

Difference between pre and post exercise measurements; **P < 0.01, ***P < 0.001

5.4 Lipid and lipoprotein responses to prolonged exercise (V, VI)

The baseline diene conjugation of LDL (LDL-BDC) remained unchanged during the 31-km run and the marathon (V) but decreased by 17% (P<0.01) after the 6-h walking (VI) (table 6). During the 2-day walking exercise protocol the total decrease in LDL-BDC was 25% (ANOVA time effect P=0.001).

TABLE 6 Acute LDL-DC, S-TRAP and S- α -tocopherol responses to prolonged exercise

		Study V	Study V	Study VI
Subjects		keep-fit marathon runners (n = 22)	competitive long distance runners (n = 8)	endurance athletes (n = 10)
Exercise	Mode	running	running	walking
	Duration	2h 42min - 5h10min	2h	6h
	Distance	42,2km	31km	38-40km
	Intensity (% vVO ₂ max)	marathon race (80%)	hard exercise run (70-85%)	higher than running (35-45%)
Response	LDL-BDC	no significant changes	no significant changes	17% decrease**
	S-TRAP	16% increase**	22% increase **	14% increase*
	S- α -tocopherol	7% increse*	29% increase*	no significant changes

*P < 0.05

**P < 0.01

Serum triglycerides, serum total cholesterol and LDL-cholesterol decreased also during the walking exercise. The reduction of triglyceride concentration during the 2-day PLA trial was 22% (P=0.001), of total cholesterol 3% (P=0.017), and of LDL-cholesterol 14% (P=0.045). At the same time, HDL cholesterol rose by 9% (P=0.001).

S-TRAP increased during the 31-km run, during the marathon and during the 2-day walking exercise by 22% (P=0.001), 16% (P=0.001) and 22% (P=0.018), respectively. LDL-TRAP was not affected by the exercises while the ratio of LDL-TRAP to LDL-cholesterol rose during the 2-day walking protocol by 15% (P=0.034). The serum concentration of α -tocopherol rose during the 31-km run (by 29%, P=0.011) and during the marathon (by 7%, P=0.031) but not during the 2-day walking exercise. No changes were seen in the serum concentrations of ubiquinol, retinol (in study V) and β -carotene (in study VI). The serum concentration of γ -tocopherol decreased during the 2-day exercise by 20% (P=0.049), while the ratio to serum lipids was unchanged (Table 6).

6 DISCUSSION

6.1 Energy release in intermittent running exercises

The relative aerobic energy release, determined by the accumulated oxygen deficit (AOD) method, was high during the 28-min intermittent running exercises. It was over 70% in IR₆₀ and over 80% in IR₁₂₀. Previous studies have shown, in line with the present findings, that during short maximal muscle work decreasing the intensity from supramaximal to maximal capacity (100 % VO_{2max}) and concomitantly prolonging the duration of muscle work increases the relative aerobic energy release (Hermansen and Medbo 1984, Medbo and Tabata 1986, Withers et al. 1991, Gatin and Lawson 1994). It has been documented, that in 800 and 1500-m maximal runs, in which the calculated running intensities were 110% and 102% VO_{2max}, respectively, the relative aerobic energy release determined by the AOD method were 70% and 83% (Spencer and Gatin 2001). These AODs were approximately the same as recorded in IR₆₀ and IR₁₂₀ in the present study.

Changing the duration of high intensity muscle work may influence the metabolic responses in several ways. The shorter the duration, the greater is the proportion of total energy produced from the intracellular phosphocreatine stores (Hermansen and Medbo 1984, Hirvonen et al. 1987, Nummela and Rusko 1995) and the more oxygen is delivered from the myoglobin bound oxygen stores for aerobic metabolism (Åstrand et al. 1960a,b, Christensen et al. 1960). Prolonging the duration of runs at V_{max} may cause opposite changes. In the present study, the peak oxygen uptake, the sum oxygen consumption and the relative aerobic energy release during the runs and the accumulated blood lactate increased when the duration of running bouts was doubled.

The findings of the present study also confirm that in well-trained runners reducing the duration of the recovery periods in connection with intermittent training has the same effect on AOD and relative aerobic energy release as prolonging the runs. The mean AOD of IR₆₀ was significantly smaller (relative aerobic energy release was higher) than the mean AOD of the first 60 seconds in 120-s runs after 120-s recovery in IR₁₂₀ (Figure 3). If the running bouts of 60-s

duration were performed with 120-s recovery, with AOD obviously being enhanced, relative aerobic energy release would be decreased even more.

In both 28-min intermittent exercises, the recovery periods between the runs were complete rest. By using active recovery, e.g. running at the intensity of 50% VO_{2max} , like in a recent interval training study (Billat et al. 1999b), the AODs of the present IRs would have decreased because the initial VO_2 level at the end of the recovery period affects the AOD of the next run. In this study, the recovery VO_2 at the end of 120-s recovery bouts (IR_{120}) was significantly lower and the first minute AOD during 120-s runs significantly higher than the corresponding values before and during the 60-s runs (Figure 3).

The subjects performed the 28-min running exercises intermittently whereas the original AOD method was used for determining the total AOD in single maximal or submaximal performances (Gastin 1994, Medbo et al. 1988, Medbo and Tabata 1989, Spencer and Gastin 2001). This may, at least partly, have increased the aerobic energy release, because it has been indicated that the rate of lactate accumulation and the anaerobic energy release tend to decrease when the same exercise bout is repeated (Spriet et al. 1989, 1990, Bangsbo et al. 1992a,b). Our results also support the recent findings of a relatively small AOD and high aerobic energy release found during high-intensity intermittent running and bicycling (Nummela and Rusko 1995, Tabata et al. 1997).

The physical characteristics of the present subjects may also have affected the measured AOD values during the intermittent exercises. Our subjects were well-trained middle distance runners, and previous studies (Nummela and Rusko 1995, Cooper and Barstow 1996) have shown that endurance athletes with a high percentage of ST muscle fibers do have a smaller AOD if performing the same high-intensity exercise when compared to sprinters and athletes with a high percentage of FT fibers.

During IR_{60} a greater proportion of total oxygen was consumed during recovery and evidently used for recharging the myoglobin bound oxygen stores and for the resynthesis of phosphocreatine (Åstrand et al. 1992). The significantly lower peak oxygen consumption, peak heart rate and blood lactate level during IR_{60} in comparison to IR_{120} suggests that a 60 second recovery time after a 60 second run is enough for these processes to assist intermittent running at V_{max} .

During IR_{120} blood lactate accumulated similarly as in the VO_{2max} test, and the aerobic energy releasing system was taxed maximally during the second half of each 120-s repetition except for the first repetition. Previous studies indicate that the high blood lactate level during prolonged aerobic-anaerobic muscle work may have, on one hand, a negative and, on the other hand, a positive influence on performance. High blood lactate is known to reflect a decreased muscle pH -level and to relate with a concomitant fall in force output of muscle contraction (Kavinsky and Meyer 1977, Chasiotis 1983, Mainwood and Renaud 1985, Hultman and Spriet 1986), although the exercise induced fall in force output is suggested to result mainly from the regulation of the central nervous system (Noakes 2001). Especially during intermittent muscle work, a

relatively high blood lactate level (up to 10mM) may also indicate effective “lactate shuttle” and high lactate consumption in working skeletal muscles which, in fact, may enhance muscular performance (Brooks 1986, 1991). In the present study there were no differences in the striding or in the vertical jump height, either during or between the exercises despite the significantly higher bLa accumulation during IR₁₂₀ than during IR₆₀. Our findings in studies I and III suggest that the ability to maintain or even improve the muscular power performance and to tolerate fatigue during and immediately after high-intensity intermittent running seems to be typical especially for well-trained middle and long distance runners. Rusko et al. (1993) have reported that during intermittent running consisting of 20-s repetitions CMJ of middle distance runners does not decrease until blood lactate level is about 10 mM.

6.2 Muscle power and muscle activation responses to intermittent and continuous running

It was a somewhat unexpected finding that an intensive 40-min running exercise resulted in an acute improvement in the power performance of the lower extremities in well-trained long distance runners. An acute improvement was observed in two different vertical leg extension exercises, counter movement jumps and half squats, performed immediately after the exercise. The acute improvement in the power performance took place after both, a continuous and an intermittent running exercise. The present findings are quite surprising, since prolonged running has usually been shown to induce acute neuromuscular perturbation and strength loss in working muscles (Davies and Thompson 1986, Nicol et al. 1991b, Paavolainen et al. 1999b, Lepers et al. 2000b, Millet et al. 2002, 2003).

The improved power performance in the half squats was associated with a decrease in the EMG activity of leg extensor muscles. This was also an unexpected finding, since an acute reduction in EMG activity is usually associated with muscle fatigue and a decrease in muscle strength (Bigland-Ritchie 1983, Häkkinen and Komi 1986, Nicol et al. 1991b, Häkkinen 1993, Strojnik and Komi 2000). In the present study, the only evidence of muscle fatigue was observed during the set of the ten half squats performed after MR; both Δ mechanical P% and Δ EMGrms% (a relative change within the set) decreased compared to the values before MR (Figure 4). Muscle fatigue associated reduction in surface EMG has been documented to result from decreased neural activity at the central level or from depression of the action potential transport in peripheral neuromuscular function (Asmussen 1979, Bigland-Ritchie et al. 1983, Gandevia 1998, Gandevia 2001). Since surface EMG in this study was not normalized by the maximal M-wave for the same muscle, it was not known whether the decreases in EMG activity were explained by changes of action potentials propagation along the sarcolemma or if neural

input reaching the neuromuscular junction was decreased after the three exercises. Furthermore, to what extent the intra-set reduction in \square mechanical P% is associated with a running-induced reduction in neural activity at the central level and / or to a depression of the action potential transport in peripheral neuromuscular function and / or to a depletion of ATP and CP stores is difficult to establish (Hirvonen et al. 1992, Millet and Lepers 2004). Anyway, the present data confirms that a running exercise-induced fatigue may be highly specific in well-trained long distance runners. Even an exhaustive running exercise does not reduce the vertical jumping and half squat performance in athletes who are used to prolonged and exhaustive runs but not to explosive types of performances. Under these conditions fatigue does not take place until during the last 3-4 half squat repetitions of the 10 repetitions.

A decrease in EMG activity measured on the surface of the knee extensor muscles may also result from exercise-induced increase in coactivation between the muscles of the lower extremities (Psek and Cafarelli 1993, Enoka 1994). During fatiguing tasks EMG may appear to rotate within and between muscles (Gandevia 2001). The latter effect presumably reflects an attempt to use a different mechanical or muscle strategy to perform the same task (Clark and Carter 1985, Gandevia 2001). In the present study we investigated EMG changes on the surface of the agonist muscles vastus medialis and lateralis and gastrocnemius, as well as of the antagonist muscle biceps femoris, which in a multi joint action, e.g. the half squat, also acts as a synergist muscle. It is obvious that after the intensive runs a different coordination strategy between the agonist and synergist muscles was used in the leg extension exercises, since EMG activities on the surfaces of vastus medialis and lateralis decreased, while no significant change on the surface of biceps femoris was found (Table 3). This new coordination strategy may have maintained or even improved the leg extension power performances of the high calibre long distance runners in the present study, although under some circumstances, e.g. after a prolonged exercise the increased coactivation of synergist muscles has also been connected to the strength loss in the active muscles (Psek and Cafarelli 1993, Millet and Lepers 2004).

The exercise-induced changes in the muscular performances were of similar quantity after the continuous type and intermittent type of the non-exhaustive running exercises. However, some differences were found in the ratio between EMGrms and P, which has been used as a parameter of neuromuscular efficiency in these types of muscle actions (Bosco et al. 2000). The decrease in EMGrms: P ratio was greater after TR (but not after IR) when compared to MR. This may partly result from greater changes in coordination strategy after TR than after IR.

It has also been shown that the contractile mechanisms of a muscle, especially the series elastic component, may be altered during an exercise (Vigreux et al. 1980) and it is assumed that the contractile response of a muscle depends to a great extent on the exercise activation (Rassier and MacIntosh 2000). A brief period of repetitive stimulation results in enhanced contractile

response (potentiation) while continued stimulation results in impairment or attenuated response (fatigue) (Rassier and MacIntosh 2000). Further, counteracting fatigue by increasing twitch tension is not necessarily associated with enhanced neuromuscular function (Millet and Lepers 2004). Exercise-induced increase in muscle temperature may also have affected on the power output and efficiency in the present study. However, it is obvious that the neuromuscular performance has not been changed differently between TR and IR and between different types of runners because the warming up procedure and the duration of intensive exercising was equal in both exercises. According to these findings and since neuromuscular functions in the present study were evaluated only by using surface EMG, the improved power performance may not be explained by an enhanced contractile response of the muscles.

6.3. Acute effects of running exercises on maximal vertical jumping

An acute improvement in maximal vertical jumping height (CMJ) was found immediately after the VO_{2max} test run, after two different submaximal running exercises of 40 min duration, and after the first phases of the longer maximal anaerobic running test modification (MART₃) in well-trained endurance runners. However, CMJ decreased after the short anaerobic running test modification (MART₁) in sprinters and during MART₃ in middle distance runners. In addition, in study II, CMJ did not change during the last phases of the submaximal intermittent exercises, of 28 min duration, in well-trained middle distance runners. (Table 3). The difference between the findings in studies II and III may be explained mostly because of the timing of the CMJ measurements. In study II, CMJ was not measured before the exercise, as in study III, but only 5-6 minutes after the start of the exercise. On the basis of these findings, it is obvious that the running-induced acute changes in CMJ depends on the training background of the runners and the intensity and duration of the exercise

An exercise-induced acute improvement in jumping performance, as well as in half squat performance, is quite a surprising finding considering the conventional fatigue-associated impairment in neuromuscular performance of leg muscles (MacIntosh and Rassier 2002, Millet and Lepers 2004), e.g. a decreased jumping performance after prolonged exercises (Ferretti et al. 1987, Millet et al. 2000). Enhancement of strength in the leg extensor muscles is known to follow adaptation to training, e.g., after several weeks of heavy resistance training (Häkkinen et al. 1985, Aagaard et al. 2002). This type of adaptation has been attributed to improvement in neuromuscular behaviour caused by the increasing activity of the higher motor centers (Aagaard 2003), and by changes in the interaction between neuromotoric, hypertrophic and mechanical factors and the function of the stretch-shortening cycle (Komi 1986,

Aagaard et al. 2001). On the other hand, the acute decrease in muscular strength is suggested to depend upon the type of muscular contraction, with a greater loss occurring in eccentric and isometric actions than in concentric ones, but is independent of the angular velocity of the movement (Lepers et al. 2000b). In the present study, the fact that the improved performance was demonstrated both in the maximal half squats starting from the static position and maximal vertical jumps with a counter movement suggests that the improved muscular power performance is not only associated with better use of the stretch-shortening cycle but also with other neuromuscular mechanisms. However, CMJ tended to increase more (10-12%) than the mechanical P in half squats (3-4%) and increased also in MR, while the half squat performance remained unchanged. Although it is tentative to compare the exercise-induced changes in half squats with additional load to the changes in CMJ without any additional load, the results suggest that the increased utilization of elastic energy may, at least partly, explain the acute exercise-induced improvement in the power performance of the lower extremities. The decreased surface EMG of leg extensor muscles also supports this assumption.

6.4 Factors relating with running-induced acute changes in power performance

In the VO_{2max} test run the improvement in CMJ correlated negatively with the accumulation of lactate in blood, but in the submaximal intermittent run the correlation was positive. During the submaximal tempo run the blood lactate increased only slightly and no relationship between blood lactate increase and CMJ improvement was found. Correspondingly, CMJ performance in marathon runners improved during the early phases of MART₃ but not during MART₁ where blood lactate increased less. A similar tendency was not found in middle distance or sprint runners who achieved the same maximal blood lactate level in both MART modifications. Thus, in non-exhaustive intermittent running, an increment of the exercise intensity, the concentration of blood lactate and possibly also the increased muscle temperature seem to be associated with improved rather than impaired explosive strength output of long distance runners. Recently, Lattier et al. (2004) reported slightly decreased isometric maximal voluntary contraction values after repeated high intensity uphill runs with high blood lactate levels. That was suggested to be due to significant alteration in excitation-contracting coupling failure and it was concluded that this type of exercise, of shorter duration but of higher blood lactate response as in the present study, does not induce significant central fatigue or changes at the crossbridge level (Lattier et al. 2004).

The acute improvement in mechanical P correlated significantly with the maximal running performance of the subjects. Surprisingly, this correlation was positive in TR but negative in IR suggesting that the higher caliber long

distance runners may obtain more neuromuscular benefit and/or resist fatigue more efficiently during continuous type of running exercises. A possible explanation for this may be related to the differences in the training background of the subjects and their adaptation to different types of running exercises. In the present study, the higher the yearly training volume of the runners the better their performances in MR ($r = 0.66$; $P < 0.001$). It is obvious, that the higher caliber long-distance runners resist fatigue and produce power more efficiently not only after maximal but also after continuous type of prolonged running; high calibre long distance runners have previously been reported to resist neuromuscular perturbation during a 10-km time trial better than low calibre runners (Paavolainen et al. 1999b). Consequently, the lower caliber long distance runners (by $v\text{VO}_{2\text{max}}$) in the present study may have been better adapted to intermittent and fast running exercise of the IR type and / or they may have become more easily fatigued during TR compared to the higher caliber and the more trained runners. This finding suggests that in training long distance runners who have good endurance but poor explosive strength output, as in our marathon runners, tempo runs (continuous type of running) would be preferred instead of interval runs, with regard to the ability to produce strength after a single exercise.

6.5 Factors underlying running power in maximal anaerobic interval run

The results of the present study are in agreement with the findings of Rusko et al. (1993), which showed that anaerobic running power in the shorter MART modification ($n \times 1 \times 20$ s) is affected by the same factors as the sprint running performance. In the present study, this was supported by the correlation analyses, which showed that the maximal sprinting speed and CMJ, but not the $\text{VO}_{2\text{max}}$, correlated positively with the maximal anaerobic running power in MART_1 . Furthermore, the sprinters and the middle distance runners attained significantly higher power values than the marathon runners in MART_1 . In the 400 m run the estimated oxygen demand is about $120 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (ACSM 1986, Noakes 1988) as was the P_{max} of the 400m runners in the study of Rusko et al. (1993) and of the sprinters and middle distance runners in the present study.

In MART_1 the sprinters were not able to reach higher P_{max} than the middle distance runners, suggesting that the limiting factors in this test may not be exactly the same as in 100 and 200-m sprinting. The sprinters have well-developed force-velocity characteristics and alactic power of leg muscles (good results in sprinting and jumping tests) while the lactic power and capacity as well as the ability to resist fatigue are well-developed in middle-distance runners (Denis et al. 1992). According to Noakes (2001) the high maximal running power of middle distance runners in MART_1 could be explained by the muscle power model and /or by the central governor model. The previous

predicts that changes in exercise performance may result from increased muscle contractile function caused by biochemical adaptations in muscle. This may increase force production or the rate of sarcomere shortening, or both, independent of changes in neural recruitment by the brain. The latter emphasize the inhibitive role of central nervous system in strenuous physical exercises. On the base of this theory, "the central governor" may have prevented, especially in the case of sprinters, the recruitment of additional muscle fibres necessary to further increase the work output in the end of MART₁.

The large number of 20-s runs and counter movement jumps obviously had more detrimental effect on the P_{max} in sprinters compared with middle-distance runners who have better aerobic capacity and lactate removal (Dennis et al. 1992, Paavolainen et al. 1994). Therefore, shorter than 20-s runs should be used if the main interest is to test alactic and neuromuscular components of maximal anaerobic power in runners trained both for short and long distances. According to Christensen (1960) and Åstrand et al. (1960a) the interval work becomes more alactic by shortening the 20-s repetitions when the intensity level is greater than 100% VO_{2max} , such as in the MART. In order to decrease the effects of fatigue on the P_{max} , the recovery period between the runs can also be prolonged (e.g. from 40 up to 100 s).

The P_{max} in each group was lower as the number of runs per set increased in the MART protocol. However, while middle distance and sprint runners attained almost the same peak blood lactate in each of the test modifications, marathon runners could not reach as high values in the MART₁ as in MART₃ and MART₅. According to Noakes (2001) this shows that it was the marathon runners' inability to recruit fast twitch muscle fibers that produced the low power output in MART₁. Due to this and the fact that the marathon runners were unable to sprint fast and to obtain high performance in CMJ, it is obvious that the factors, which limited the maximal anaerobic running power of marathon runners, were associated with the function of the neuromuscular system and especially in the MART₁. Despite this, the marathon runners attained the highest P_{max} in MART₁. Thus, the only reason to recommend the longer modifications of the MART for marathon runners is associated with blood lactate accumulation. In testing the maximal anaerobic running power, marathon runners were able to accumulate higher lactate as the number of runs per set increased in the MART protocol. There are also several arguments why the longer MART modifications may not be useful for short distance runners. First, the sprinters could not perform MART₅ at all. Second, both middle distance runners and sprinters had a significantly higher P_{max} in the MART₁ than in the other test modifications. Moreover, the sprinters and the middle distance runners could not accumulate more blood lactate in MART₃ and MART₅ than in MART₁.

Explosive strength of the leg muscles of the sprinters was superior over the middle-distance and marathon runners. Bosco et al. (1987) and Mero et al. (1990) have suggested that a 10-cm difference in CMJ (such as in the present

study) affects the maximal running speed by increasing the ability to move the legs fast and to use longer strides. In the present MART study (I) and in one previous study, (Paavolainen et al. 1994) CMJ decreased in runners specialized for shorter distances but was at exhaustion still higher in the sprinters and middle-distance runners than in the marathon runners. Therefore, the decreased ability of the neuromuscular system to produce explosive strength due to fatigue cannot be the only reason for exhaustion in the sprinters and middle distance runners. After the longer MART modifications their P_{\max} did not differ significantly from the P_{\max} of the marathon runners. Other reasons for exhaustion could be central fatigue (Paavolainen et al. 1994) that is in connection with a limited ability to release anaerobic energy (Noakes 2001). The latter possibility is supported by the similar peak blood lactate in the different MART modifications in sprinters and middle-distance runners.

6.6 Different adrenal and testicular responses to continuous and intermittent runs between marathon and middle distance runners

The present results showed that the acute exercise-induced testosterone and cortisol responses were partly different in high level endurance runners who had event specific differences in their physical characteristics and training background. Marathon runners had lower cortisol response to intensive continuous running compared to middle distance runners while middle distance runners had higher testosterone response to intensive intermittent running compared to marathon runners. Previously, differences in serum cortisol and testosterone responses to acute prolonged exercise have been documented between endurance athletes and non-athletes (e.g. Vasankari et al. 1993a) and between high and low level endurance athletes (Snegovskaya and Viru 1993). Based on the present results, an acute shift to a more anabolic and less catabolic hormone environment within the body results from continuous type running exercise in marathon runners and intermittent type running exercise in middle distance runners. These differences in acute hormone responses are obviously due to differences in the subjects' genetic background and / or adaptation to marathon type (aerobic) or middle distance type (anaerobic) of training.

The endocrinological responses to intensive continuous and intermittent running were studied by changes in serum testosterone, LH, FSH and cortisol concentrations and in the molar ratio of testosterone to cortisol in 20 well-trained middle and long distance runners. Because testosterone is the main anabolic hormone and cortisol is the main stress / catabolic hormone, the molar ratio of testosterone to cortisol was used to evaluate if two exercises were a different anabolic or catabolic stimuli for athletes (Kenefick et al. 1998). In the present study, changes in plasma volume were not measured and thus, the

absolute changes in the measured hormone concentrations may be overestimated. However, the total exercise time was only 40 min of duration and no changes in the body weight were found after the exercises. Thus, the possible decrease in the plasma volume may have been minimal in both exercises. In addition to this, the total exercise time during both exercises was equal so that the comparisons between the groups could also be conducted without major influences of the possible plasma volume changes.

After the first 20 min of exercising s-cortisol remained at the pre-exercise level in TR while in IR it was elevated by 17%. Thereafter, s-cortisol increased also in TR reaching, immediately after 40 min of exercising, the same level as after IR. S-testosterone increased similarly in both exercises. Tremblay et al. (2005) reported partly similar testosterone and cortisol changes after continuous type runs at 50% of VO_{2max} . At that intensity, exercise duration had an independent effect on the hormonal responses; longer duration was necessary to stimulate increased levels of testosterone, DHEAS (dehydroepiandrosterone sulfate) and cortisol. Beyond 80 min of running there was a shift to a more catabolic hormonal environment. In TR, which was performed at an intensity of 80% vVO_{2max} , the duration of 20 minutes was enough to increase the concentration of serum testosterone but not cortisol. However, it is possible that the concentration of serum cortisol may also have increased after 20 minutes if the exercise was stopped at that point. Serum cortisol concentration continued to increase after 40 minutes, during the first ten minutes of recovery (Table 5) similarly as it has been shown to behave after exhaustive running at the ventilatory threshold (85 min) in trained runners (Daly et al. 2005) and for example after heavy-resistance exercise protocols (Kraemer et al. 1993). The differences in the serum cortisol concentrations after 20 and 40 minutes of exercising between TR and IR suggests that in continuous type of running the duration of exercise is the stress factor, which induces elevated levels of s-cortisol.

The present study agrees with previous ones that exercise intensity has an affect on the endocrine responses to acute prolonged exercise (Kindermann et al. 1982, Snegovskaya and Viru 1993, Duclos et al. 1996, Tremblay 2005). In continuous (aerobic) type of running the shift to more catabolic hormonal environment seems to take place earlier if the intensity is higher. In the present study, 40 min of running at 80% of vVO_{2max} resulted in a shift to more catabolic hormonal environment (increase in serum cortisol concentration) compared to the hormonal environment after 20 min of running. A similar tendency is not found when running at 50% of VO_{2max} (Galbo 1981) or it is found not earlier than after 120 min of running (Tremblay et al. 2005). Previously, a single bout of anaerobic running (leading to exhaustion within 1.5 min) has been shown to increase serum cortisol concentration more compared to 50 min of aerobic running at the 4mM blood lactate level (Kindermann et al. 1982). In the present study, the 40-min IR, which was performed at an intensity of 100%, did not increase serum cortisol concentration more but did it earlier compared to TR, which was performed at an intensity of 80% vVO_{2max} . The total time but not the

total work output of TR and IR were equated resulting in similar subjective strain (RPE) measured at the end of the 40-min exercises. If the total work output were to be equated, the serum cortisol response to the more intense IR might have been greater compared to TR, as in a previous study where the effects of equal anaerobic (110% $\text{VO}_{2\text{max}}$) and aerobic (65% $\text{VO}_{2\text{max}}$) total work outputs on the cortisol response was studied (Hackney et al. 1995).

Exercise induced acute increases in serum testosterone concentration have been suggested to be caused by the influence of increased circulation in the testicles, activation of the sympathetic nervous system, increased lactate accumulation and / or luteinizing hormone concentrations (Eik-Nes 1969, Jezova and Vigas 1981, Fahrner and Hackney 1998). In the present study, the exercise-induced rise of testosterone took place without any change in LH or FSH suggesting that the acute rise in serum testosterone levels was not of pituitary origin. On the contrary, the exercise-induced decrease in serum testosterone has been found after prolonged exercise and it has been concluded to be caused by reduced hypothalamus-pituitary stimuli (Vasankari et al. 1993b). During short-term intense exercise, as IR in the present study, the circulating testosterone level is probably influenced by the sympathetic nervous system or by circulating catecholamines (Sutton and Lazarus 1974, Jezova and Vigas 1981). After IR, serum testosterone increased more in middle distance runners compared to marathon runners and a positive correlation between the change in serum testosterone concentration after IR and the maximal blood lactate concentration achieved after the exhaustive $\text{VO}_{2\text{max}}$ test run was found. However, no difference in post-IR blood lactate concentration was found between middle distance and marathon runners, and neither any correlation between the serum testosterone and blood lactate increases after IR. Hence, after intense intermittent type running, some physical characteristics, e.g. high maximal lactic energy producing capacity but not the blood lactate accumulation during the exercise, seems to be connected with the high serum testosterone increase. The individual differences in physical characteristics may be either of genetic origin or developed as adaptation to training.

In the present study, the intensive continuous running induced serum cortisol to increase more in middle distance runners compared to marathon runners. In addition to this, the increase in serum cortisol concentration correlated positively with the runners' maximal running speed and maximal explosive strength (maximal vertical jump) but negatively with $\text{VO}_{2\text{max}}$ as well as with the total running training amount during the last year before the study. A positive correlation was also found between the runners' $\text{VO}_{2\text{max}}$ and the TR-induced change in the molar ratio of testosterone to cortisol. These findings suggest that in hard continuous type of running cortisol secretion is stimulated more in endurance runners who have done less (endurance) running training and have a low $\text{VO}_{2\text{max}}$, but are fast and have a high level of explosive strength.

High-level middle distance runners and marathon runners both do, at least in some training periods, high mileages in their daily training and are supposedly well adapted to many different types of long distance running

exercises. However, their training also includes some basic differences. Middle distance runners do more sprint and muscle power training and fast intermittent running exercises. In the present study (III, IV) the proportion of intermittent running was about 8.5% in middle distance runners and only 4.5% in marathon runners. This is intentional because intermittent runs at a velocity that yields maximal oxygen uptake have been suggested to be especially effective for enhancing middle distance running performance (e.g. Hill and Rowell 1997). Marathon runners prefer slower continuous type of running and have higher VO_{2max} and decreased maximal running speed and explosive strength as shown in study I. High running mileage and adaptation to marathon type of training and related training-induced changes in physical performance, e.g. high VO_{2max} , seem to explain, at least partly, the differences in exercise-induced serum cortisol changes in hard continuous running. Because marathon runners had lower cortisol response to TR, when compared to middle distance runners, together with a high testosterone response, it may be argued that they use TR-type of exercises in their training. Correspondingly, it may be favourable that middle distance runners use IR-type of exercises in their training, because their testosterone response to IR was higher compared to marathon runners. These suggestions are supported by the findings of study III that TR-type of exercise induces acute positive changes in muscular performance especially in runners who have a high vVO_{2max} (mostly marathon and long distance runners) and IR-type of exercise in runners who have lower vVO_{2max} (mostly middle distance runners).

Although cortisol is the main catabolic hormone and elevated serum cortisol levels may indicate a shift to a more catabolic hormone environment, unfavourable catabolism may not have existed in the present study, even in MID after TR. After both types of exercises, serum testosterone concentration increased in line with cortisol in both groups and the ratio of testosterone to cortisol did not decrease when compared to the pre-exercise level. In addition to this, the increase in serum cortisol, in both groups after both exercises, was of the same amount as reported earlier after prolonged exercise of well-trained athletes (Kuoppasalmi et al. 1980, Vasankari et al. 1991). Furthermore, it should be noted that the exercise response of adrenal cortex activity may also be triggered by the central motor command and the responses may be further supported by positive feedback influences from proprio- and metaboreceptors in muscles (Kjaer 1992). In our earlier study (Vuorimaa et al. 1999) we showed that, despite the well documented acute changes in serum hormone concentrations, a single IR type of intensive running exercise does not induce changes in serum cortisol and testosterone of well-trained runners during recovery over 3 days. In less fit or untrained subjects, hard prolonged exercises have led to decreased levels of serum testosterone (Vasankari et al. 1993a,b). Thus, in spite of the higher cortisol response, TR type of continuous running exercises may be favourable for middle distance runners when training-induced improvement in performance is expected. Similarly, the total hormone response

to IR in marathon runners was great, which is a mark of altering the homeostasis and may result in a good training effect.

6.7 Acute lipid and lipoprotein responses to prolonged exercise

It has been shown in earlier studies that under some circumstances intensive endurance exercises and increased release of pro-oxidants may cause oxidative stress, e.g. oxidation of circulating lipids (Davies et al. 1982, Jenkins 1988). In the present study prolonged and low intensity walking with trained men, decreased the level of conjugated dienes extracted from LDL (LDL-BDC), which are a determinant of circulating oxidized LDL (Ahotupa et al. 1996, 1998, 1999). LDL oxidation seemed to decrease already during the first day of walking. Since oxidized LDL is a risk factor for cardiovascular disease (Steinberg 1994, Witztum 1994), this change may be beneficial. This finding indicates that the positive changes in the concentrations of the conventional lipids after prolonged exercise also occur in the concentration of oxidized LDL, measured "in vivo". Our finding is in accordance with previous observations according to which several years of endurance training (Kujala et al. 1996) and a training period of 10 months (Vasankari et al. 1998) result in a reduction of the concentration of moderately oxidized LDL. Further, a single bout of ultra-endurance exercise reduced the susceptibility of LDL to peroxidation "in vitro" in well-trained triathletes (Ginsburg et al. 1996). Contradictory findings have, however, also been reported: increased serum autoantibodies against oxidized LDL have been recorded in specific athlete groups like professional basketball and football players (Pincemail et al. 2000).

In study VI, the lack of a non-exercise control group lead us to speculate that the reduced LDL oxidation, as well as the differences in other variables, may partly be caused by factors other than the exercise. However, since in study VI, the results of the baseline measurements of the two trials did not differ, and the results of both trials were comparable, a true exercise effect is rather obvious.

In study V, acute exercise induced no changes in LDL oxidation. The difference between studies V and VI may be explained by the difference in the intensity and nature of the exercise. The exercise in study VI was of long duration but the intensity of the walking was low, around 50% VO_{2max} , as judged on the basis of the exercise heart rate. This is lower than the subjects had used in their daily training before study VI and also lower than in study V, in which the exercise used was running and which resulted in no change in LDL oxidation. Nor did the exercise schedule in study VI include intermittent fast and anaerobic working sessions, which is the case for basketball and football players (Pincemail et al. 2000). The present walking exercise reduced the subjects' ability to run 1000 m as fast as possible while the submaximal running ability (on the basis of blood lactate level in submaximal running) did not

change. This indicates that the physiological strain of our prolonged exercise protocol, consisting of two consecutive days, causes fatigue in the running muscles, but is not exhausting for trained men. In study V, the exercises were more strenuous and/or led to exhaustion.

The degree of LDL oxidation may be associated with the levels of malonaldehyde in the plasma, since increased levels have also been said to reflect exercise-induced oxidative stress (Jenkins 1988). The decreased concentration of oxidized LDL in study VI on low intensity exercise is supported by an earlier study (Lovlin et al. 1987), which showed that plasma levels of malonaldehyde were dependent on exercise intensity. In that study, the plasma malonaldehyde levels rose when the subjects performed at maximal intensity (100% VO_{2max}), was unchanged at exercise of short duration at an intensity of 70% VO_{2max} , and sank at an intensity of 40% VO_{2max} . Obviously, the exercise performed at an intensity of 40% VO_{2max} , which comes close to the walk exercise performed at an intensity of 50% VO_{2max} in the present study, is light enough to avoid such oxidative stress, which increases both the levels of plasma malonaldehyde and LDL oxidation.

A good training background may also be one reason for the lack of an acute LDL oxidation response to the 2-day exercise protocol in study VI. The subjects were well trained for the exercise in this study. They had a training history of endurance sports, and during the last 9 weeks before the study they had prepared especially well for the walking exercise of long duration. Regular participation in intensive physical exercise training is associated with reduced levels of circulating oxidized LDL, which is reflected by enhanced LDL diene conjugation (Kujala et al. 1996). Also, experimental studies show that physical training and adaptation to muscle work of long duration may alter the response of lipid peroxidation to exercise in humans (Viinikka et al. 1984, Alessio and Goldfarb 1988, Krezchmar et al. 1992) and in vitro at least in rat muscle (Salminen and Vihko 1983).

In study VI, the finding of an exercise-associated increase in serum antioxidant potential (S-TRAP), but not in the antioxidant capacity of LDL (LDL-TRAP), accords with study V where S-TRAP, but not LDL-TRAP, increased in subjects who ran 31 km and 42 km. However, in study VI, the increase in S-TRAP can not be explained directly by an effect of a single antioxidant, like α -tocopherol in study V, because not any of the measured serum antioxidant concentrations increased during the 2-day walking exercise. It is obvious that the extremely long duration of exercise (12h in two days) has led to the increased antioxidant consumption in working tissues, and caused the decrease in serum γ -tocopherol and the absence of the acute increase in circulating antioxidants seen in exercise of shorter duration (Pincemail et al. 1988). Further, the 2-day exercise-induced remarkable decrease in serum lipids may have been the reason why the α -tocopherol: serum lipids ratio increased (Thurnam et al. 1986). However, some other factors, like elevated levels of plasma proteins (e.g. uric acid, albumin, bilirubin, ceruloplasmin, transferritin

and haptoglobin) may have led to the exercise-induced increase in S-TRAP (Davies et al. 1982, Haramäki and Packer 1994).

Carbohydrate ingestion during the 2-day exercise protocol had no further effect on the antioxidant capacity nor on the plasma lipid and lipoprotein levels. Rather, the increase in serum antioxidant capacity was somewhat lower in the trial where the subjects received carbohydrate. This is not what one would expect on the basis of earlier studies, which have implied that carbohydrate ingestion strengthens the antioxidant defence system during heavy exercise training (Vasankari et al. 1998, Mooradian et al. 1994). The 2-day exercise was performed at low intensity and our subjects were healthy and well trained. Carbohydrates may enhance antioxidant capacity more in situations where the subjects are not adapted to exercises of long duration and not pre-protected against exercise-induced lipid oxidation and oxidative stress.

7 PRIMARY FINDINGS AND CONCLUSIONS

The main findings and conclusions of the present study can be summarized as follows:

1) **Blood lactate accumulation and oxygen uptake during intermittent running**

The physiological strain, as evidenced by blood lactate accumulation, oxygen consumption and heart rate, increased significantly when the duration of running bouts was doubled from 1 to 2 min (with 1 and 2 min recovery, respectively) in a 28-min intermittent exercises performed at the velocity associated with VO_{2max} (vVO_{2max}). The aerobic energy release, determined by the accumulated oxygen deficit method, increased from about 70% to 82%. This is about the same level as documented in 800 and 1500-m races, respectively.

2) **Muscle power and muscle activation responses to single endurance running exercises**

a) Three different endurance running exercises led to a significant acute improvement in the vertical jumping and half squat performance in elite middle and long distance runners. The only evidence of muscle fatigue was found, when the maximal test induced an acute decrease in mechanical power, expressed as a relative change within the set of half squats. The capacity to produce power with both legs was improved, although a reduction in the EMG activity of leg extensor muscles took place. An acute improvement in power performance was observed after continuous and intermittent type of running exercises in both jumping and half squat performances. It is likely, that after these types of intensive running in elite endurance runners, the use of a different coordination strategy counteracts strength loss and even improves power in extension exercises performed vertically with both legs.

b) Intermittent running consisting of $n \times 3 \times 20$ -s repetitions, improved vertical jumping performance in marathon runners but decreased vertical jumping performance in middle distance runners. The improvement in the half squat power after the intensive continuous type of running correlated positively with the $v\text{VO}_{2\text{max}}$ of the well-trained endurance runners. The corresponding correlation in the case of the 40-min intermittent running exercise was negative. On the basis of these findings, it is likely, that runners with different training backgrounds have differences in their muscular responses between different type of intermittent running and between continuous and intermittent running. These differences could result from adaptation to different type of training. Middle distance runners are obviously better adapted to intermittent running where the explosive muscle contractions and performance in different power performance tasks could be better maintained. Marathon runners are obviously similarly adapted, either genetically or as a result of training, to continuous type of running exercises. Measuring maximal vertical jump height on a contact mat may be a useful and simple way to follow the acute neuromuscular responses to endurance running exercises. However, it should be noted that these changes do not necessarily reflect the neuromuscular changes and fatigue in running action.

3) Acute endocrinological responses to intermittent and continuous running in middle distance and marathon runners

The TR and IR exercise protocols, both of which were rated as hard or very hard on the Borg scale, led to greater acute testosterone and cortisol responses in both middle distance and marathon runners. The continuous type of running increased serum cortisol later than the intermittent type of running. Serum testosterone response to IR and serum cortisol response to TR were higher in middle distance runners compared to marathon runners. The tempo running - induced increase in serum cortisol was the lower the higher the runners' $\text{VO}_{2\text{max}}$. Further, the change in the ratio of testosterone to cortisol after TR correlated positively with the $\text{VO}_{2\text{max}}$ of the runners and after IR it correlated positively with the maximal blood lactate measured after the exhaustive test run. On the basis of these findings, testosterone and cortisol responses to continuous versus intermittent running are specific regarding the training background and physical characteristics of the runner. Runners who have high aerobic capacity seem to have higher acute anabolic response to intensive tempo running whereas runners who have high anaerobic capacity seem to have similar response to intensive intermittent running.

4) Acute oxidative stress response to endurance running exercises of long duration

a) The antioxidant capacity (S-TRAP) was shown to increase immediately after three different endurance exercises of long duration in trained endurance athletes. This paradoxical exercise-associated increase in S-TRAP is, at least

partly, explained by a simultaneous increase in the serum concentration of α -tocopherol after an intensive 31-42-km run, but not after a 2-day walking exercise at low intensity.

b) An unexpected acute decrease in the oxidative stress marker, LDL-BDC, was found after a low intensity 6h walking exercise but not after a 31-km intensive run or after a competitive marathon run. This shows that an endurance exercise of unusually long duration and low intensity may decrease the concentration of moderately oxidized LDL and increase serum antioxidant potential in athletes adapted to exercises of long duration. These findings also imply that similar exercise-induced beneficial changes, as seen in the conventional lipid risk factors of atherosclerosis, also take place in circulating moderately oxidized LDL.

5) Training recommendations for middle and long distance runners

The results of this study emphasize the importance of individuality in programming the training of middle and long distance runners. It is useful to recognize the differences in training background and physical performance when selecting running exercise models, which would result in expected muscular and hormonal responses. It seems that the runners who are fast and specialized to middle distances will have more favourable acute neuromuscular and hormonal responses to fast intermittent running compared to intensive continuous running. On the contrary, the marathon type of runners, who have high maximal aerobic power, seem to respond better to continuous type of running. Thus, at least during a short period of time, e.g. when preparing to important competitions, middle distance runners may benefit more from intermittent running exercises and marathon runners more from continuous type of exercises. On the other hand, during a long period of time, e.g. during a basic training period, there may also be a need to train by another way. This means that middle distance runners should try to adapt for longer continuous type of running and marathon runners for faster intermittent type of running.

YHTEENVETO

Neuromuskulaariset, hormonaaliset ja hapettumisstressiin liittyvät vasteet kestävyysjuoksuharjoituksiin hyvin harjoitelleilla juoksijoilla

Kun pyritään kehittämään kilpaurheilussa käytettäviä harjoitusmenetelmiä, on tärkeää tuntea sekä lajissa käytettävien pääharjoitusten että harjoittelujaksojen aikaansaamat muutokset urheilijan elimistössä. Kestävyysjuoksussa yksi harjoittelun pääelementtejä on matalatehoinen kestojuoksuharjoittelu, jota painotetaan kaikilla kestävyysjuoksijoilla peruskuntoharjoittelussa ja maratonjuoksijoilla läpi koko harjoituskauden. Kestävyysjuoksuharjoittelun toinen pääelementti on kovatehoisempi intervalliharjoittelu, jota erityisesti keskimatkojen juoksijat painottavat kilpailuun valmistavssa harjoittelussa.

Tässä tutkimuksessa tutkittiin eri tyyppisten, kesto- ja intervalliperiaatteella toteutettujen, kestävyysjuoksuharjoitusten vaikutuksia tarkastelemalla hyvin harjoitelleiden urheilijoiden hermolihajärjestelmään, hormonieritykseen ja oksidatiiviseen stressiin liittyviä akuutteja muutoksia. Tavoitteena oli selvittää ovatko jalkalihasten voimantuottotehossa ja seerumin hormonipitoisuuksissa tapahtuvat akuutit harjoitusperäiset muutokset erilaisia kesto- ja intervalliperiaatteella suoritettavissa kestävyysjuoksuharjoituksissa ja riippuvatko muutokset juoksijan fyysisistä ominaisuuksista ja harjoitustaustasta. Lisäksi pyrittiin selvittämään aikaansaako pitkäkestoinen kestävyysharjoitus hyvin harjoitelleilla urheilijoilla oksidatiivista stressiä ja onko akuutti oksidatiivinen stressi yhteydessä harjoitusten tehoon.

Tutkimukseen osallistui kaikkiaan 88 mieskestävyysurheilijaa. Akuutteja harjoitusvasteita tutkittiin kaikkiaan yhdeksässä eri juoksusuorituksessa ja yhdessä pitkässä kävelysuorituksessa. Juoksusuorituksista lyhin ja nopein oli 20 s toistoista 100 s palautuksella koostunut nousujohteinen intervallijuoksu ja pisin 42,2 km:n maratonjuoksukilpailu. Harjoitusten aikaansaamia välittömiä voimantuottokyvyn muutoksia mitattiin ennen ja jälkeen harjoitusten suoritetuin vertikaalisuuntaisin kontaktimattohypyyn ja lisäpainon kanssa suoritettuihin puolikykykkysarjoin. Puolikykykyissä seurattiin myös lihasaktiivisuutta pinta EMG - mittauksin neljästä eri alaraajan lihasryhmästä. Intervalli- ja kestoharjoituksen aikaansaamia endokrinologisia muutoksia seurattiin mittaamalla seerumin testosteroni-, LH-, FSH- ja kortisoli pitoisuuksia ennen harjoitusta, harjoitusten puolessa välin ja välittömästi harjoitusten jälkeen. Oksidatiivisen stressin ilmenemistä ja antioksidanttipuolustusta seurattiin mittaamalla lipoproteiineissa tapahtuvia hapettumismuutoksia ja seerumin antioksidanttipitoisuuksissa ja kokonaisantioksidanttikapasiteetissa (S-TRAP) tapahtuvia muutoksia.

Sekä intervalli- että kestoperiaatteella toteutettujen juoksuharjoitusten jälkeen suoritetuissa puolikykykkysarjoissa reiden ojentajalihasten lihasaktiivisuuden havaittiin laskevan, samalla kun kyykkysuorituksista mitattu voimantuotto ja kontaktimattohypyjen nousukorkeus parani. Muutokset

olivat osittain erilaisia keskimatkoille ja pitkille juoksumatkoille erikoistuneilla juoksijoilla. Nousujohteisista 3 x 20 s sarjoista koostuva intervallijuoksu paransi maksimaalista vertikaalista kevennyshyppyä maratonjuoksijoilla, mutta ko. suoritus heikkeni keskimatkanjuoksijoilla. Kansainvälisen tason ja kansallisen kärkitason keskimatkanjuoksijoista ja maratonjuoksijoista koostuvassa ryhmässä kolme erityyppistä juoksumattoharjoitusta, 40 min kovavauhtinen intervallijuoksu, 40 min kovavauhtinen kestojuoksu ja nousujohteinen noin 20 min kestoinen testijuoksu, paransivat kaikki vertikaalihyppysuoritusta ja voimantuottotehoa lisäpainoin suoritetuissa puolikykyissä. Samanaikaisesti reiden etuosan lihasten, vastus medialis ja vastus lateralis, lihasaktiivisuus laski. Kestojuoksun aikaansaama voimantuottotehon parantuminen korreloi positiivisesti juoksijoiden maksimaalisen aerobisen juoksunopeuden (nopeus, jolla nousujohteisessa maksimaalinen hapenottokyky saavutetaan) kanssa. Subjektiviiselta kuormitukseltaan yhtä raskaaksi koetun intervalliharjoituksen yhteydessä vastaava korrelaatio oli negatiivinen. Tulokset osoittavat, että hyvin harjoitelleilla kestävyysjuoksijoilla kovien juoksuharjoitusten aikana voi tapahtua muutoksia jalkalihasten koordinaatiostrategiassa. Muutokset voivat ehkäistä voimantuottokyvyn heikkenemistä ja johtaa jopa suorituskyvyn paranemiseen nopeata voimantuottoa vaativissa jalkaliikkeissä. Näin näyttäisi voivan tapahtua ainakin, mikäli suoritettavat liikkeet ovat vertikaalisuuntaisissa ja ei-juoksunomaisia.

Kestoltaan 40 min mittainen intervalliharjoitus ja samanmittainen kestoarjoitus nostivat sekä seerumin testosteroni- että kortisolipitoisuutta. Intervalliharjoituksen aikaansaama seerumin testosteronitason nousu ja toisaalta kestoarjoituksen aikaansaama kortisolin nousu oli suurempi keskimatkanjuoksijoilla kuin maratonjuoksijoilla. Kestoarjoituksen jälkeen mitattu testosteronin ja kortisolin molaarisuhteen muutos korreloi positiivisesti juoksijan maksimaaliseen hapenottokykyyn.

Kolme erityyppistä pitkää kestävyysuoritusta, kuuden tunnin kävelysuoritus, 31 km:n kova juoksuharjoitus ja maratonjuoksukilpailu, aikaansaiivat kaikki hyvin harjoitelleiden kestävyysurheilijoiden seerumin antioksidanttikapasiteetin akuuttia kasvua. Molempien pitkien kestävyysjuoksuuoritusten jälkeen hapettuneen LDL:n määrä pysyi suorituksia edeltävällä tasolla ja jopa odottamattomasti laski kuuden tunnin matalatehoisen kävelysuorituksen jälkeen.

Tämän tutkimuksen tulokset korostavat urheilijan harjoitustaustan ja yksilöllisten ominaisuuksien tunnistamisen tärkeyttä suunniteltaessa akuuttien voimantuotto- ja hormonimuutosten kannalta mahdollisimman hyödyllistä kestävyysarjoittelua. Näyttää siltä, että jalkalihasten voimantuottokyvyn ja seerumin hormonimuutosten perusteella keskimatkoille erikoistuneet nopeat juoksijat reagoivat akuutisti paremmin nopearytmiseen intervallijuoksuun ja pitkille juoksumatkoille erikoistuneet kestävämmät juoksijat puolestaan akuutisti paremmin hidarytmiseen kestojuoksuun. Ainakin lyhyellä aikavälillä, esim. kilpailukaudella ennen pääkilpailuja, tuntuisi tämän vuoksi tarkoituksenmukaiselta, että mailerityyppinen (nopea) juoksija harjoittelisi

intervallivoittoisesti ja maratontyyppinen (hidas) juoksija kestovoittoisesti. Toisaalta, pidemmällä aikavälillä, esim. peruskuntokauden aikana, lienee tarpeellista totuttautua myös harjoituksiin, jotka erityisesti kuormittavat hermolihasjärjestelmää ja hormoniaineenvaihduntaa, mutta eivät aikaansaa yhtä positiivisia, jalkojen voimantuottoon ja hormonimuutoksiin liittyviä, akuutteja harjoitusvasteita.

Tämän tutkimuksen perusteella näyttää myös siltä, että mikäli kestävyysurheilija on hyväkuntoinen ja tottunut pitkäkestoisiin kestävyysharjoituksiin, hän pystyy vahvan antioksidanttikapasiteettinsa avulla vastustamaan akuuttia oksidatiivista stressiä, jota yleensä ilmenee kestävyystyyppisten kuormitusten yhteydessä. Hyvin harjoitellut urheilijat näyttävät kykenevän vastustamaan oksidatiivista stressiä ainakin kestoiltaan pitkissä mutta urheilijan suorituskykyyn nähden matalatehoisissa harjoituksissa.

REFERENCES

- Aagaard, P. (2003). Training-Induced Changes in Neural Function. *Exerc. Sport Sci. Rev.* 31, 61-67.
- Aagaard, P., Andersen, J. L., Dyhre-Poulsen, P., Leffers, A. M., Wagner, A., Magnusson, S. P., Halkjaer-Kristensen, J. and Simonsen, E. B. (2001). A Mechanism for Increased Contractile Strength of Human Pennate Muscle in Response to Strength Training: Changes in Muscle Architecture. *J. Physiol.* 534, 613-623.
- Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, P. and Dyhre-Poulsen, P. (2002). Increased Rate of Force Development and Neural Drive of Human Skeletal Muscle Following Resistance Training. *J. Appl. Physiol.* 93, 1318-1326.
- Abe, D., Yanagawa, K., Yamanobe, K. and Tamura, K. (1998). Assessment of Middle-Distance Running Performance in Sub-Elite Young Runners using Energy Cost of Running. *Eur. J. Appl. Physiol. Occup. Physiol.* 77, 320-325.
- Abe, T., Brown, J. B. and Brechue, W. F. (1999). Architectural Characteristics of Muscle in Black and White College Football Players. *Med. Sci. Sports Exerc.* 31, 1448-1452.
- Abe, T., Kumagai, K. and Brechue, W. F. (2000). Fascicle Length of Leg Muscles is Greater in Sprinters than Distance Runners. *Med. Sci. Sports Exerc.* 32, 1125-1129.
- ACSM (1986). American College of Sports Medicine. Guidelines for graded exercise testing and exercise prescription, 3rd ed. Lea and Febiger, Philadelphia, p. 168
- Ahotupa, M., Marniemi, J., Lehtimäki, T., Talvinen, K., Raitakari, O. T., Vasankari, T., Viikari, J., Luoma, J. and Ylä-Herttuala, S. (1998). Baseline Diene Conjugation in LDL Lipids as a Direct Measure of in Vivo LDL Oxidation. *Clin. Biochem.* 31, 257-261.
- Ahotupa, M., Ruutu, M. and Mantyla, E. (1996). Simple Methods of Quantifying Oxidation Products and Antioxidant Potential of Low Density Lipoproteins. *Clin. Biochem.* 29, 139-144.
- Ahotupa, M. and Vasankari, T. J. (1999). Baseline Diene Conjugation in LDL Lipids: An Indicator of Circulating Oxidized LDL. *Free Radic. Biol. Med.* 27, 1141-1150.
- Alessio, H. M. (1993). Exercise-Induced Oxidative Stress. *Med. Sci. Sports Exerc.* 25, 218-224.
- Alessio, H. M. and Goldfarb, A. H. (1988). Lipid Peroxidation and Scavenger Enzymes during Exercise: Adaptive Response to Training. *J. Appl. Physiol.* 64, 1333-1336.
- Allen, D. G., Westerblad, H., Lee, J. A. and Lannergren, J. (1992). Role of Excitation-Contraction Coupling in Muscle Fatigue. *Sports Med.* 13, 116-126.

- Alway, S. E., Hughson, R. L., Green, H. J., Patla, A. E. and Frank, J. S. (1987). Twitch Potentiation After Fatiguing Exercise in Man. *Eur. J. Appl. Physiol. Occup. Physiol.* 56, 461-466.
- Anderson, T. (1996). Biomechanics and Running Economy. *Sports Med.* 22, 76-89.
- Arrese, A. L., Munguia, I., Urdiales, D. M. (2005). A Review of the Maximal Oxygen Uptake Values Necessary for Different Performance Levels. *New Studies in Athletics* 3: 7-20.
- Asmussen, E. (1979). Muscle Fatigue. *Med. Sci. Sports* 11, 313-321.
- Åstrand, I., Åstrand, P. O., Christensen, E. H. and Hedman, R. (1960a). Intermittent Muscular Work. *Acta Physiol. Scand.* 48, 448-453.
- Åstrand, I., Åstrand, P. O., Christensen, E. H. and Hedman, R. (1960b). Myohemoglobin as an Oxygen-store in Man. *Acta Physiol. Scand.* 48, 454-460.
- Åstrand, P. O. (1992). Endurance Sports. In: Shephard, R. J. and Åstrand, P. O (eds.) *Endurance in Sports*, Blackwell, Oxford, pp. 8-12.
- Åstrand, P. O. and Rodahl, K. (1997). *Textbook of Work Physiology*, McGraw-Hill, New York.
- Aunola, S. and Rusko, H. (1986). Aerobic and Anaerobic Thresholds Determined from Venous Lactate Or from Ventilation and Gas Exchange in Relation to Muscle Fiber Composition. *Int. J. Sports Med.* 7, 161-166.
- Aura, O. and Komi, P. V. (1986). Effects of Prestretch Intensity on Mechanical Efficiency of Positive Work and on Elastic Behavior of Skeletal Muscle in Stretch-Shortening Cycle Exercise. *Int. J. Sports Med.* 7, 137-143.
- Bangsbo, J., Gollnick, P. D., Graham, T. E., Juel, C., Kiens, B., Mizuno, M. and Saltin, B. (1990). Anaerobic Energy Production and O₂ Deficit-Debt Relationship during Exhaustive Exercise in Humans. *J. Physiol.* 422, 539-559.
- Bangsbo, J., Graham, T., Johansen, L., Strange, S., Christensen, C. and Saltin, B. (1992a). Elevated Muscle Acidity and Energy Production during Exhaustive Exercise in Humans. *Am. J. Physiol.* 263, R891-9.
- Bangsbo, J., Graham, T. E., Kiens, B. and Saltin, B. (1992b). Elevated Muscle Glycogen and Anaerobic Energy Production during Exhaustive Exercise in Man. *J. Physiol.* 451, 205-227.
- Bassett, D. R., Jr and Howley, E. T. (1997). Maximal Oxygen Uptake: "Classical" Versus "Contemporary" Viewpoints. *Med. Sci. Sports Exerc.* 29, 591-603.
- Bassett, D. R., Jr and Howley, E. T. (2000). Limiting Factors for Maximum Oxygen Uptake and Determinants of Endurance Performance. *Med. Sci. Sports Exerc.* 32, 70-84.
- Berson, S. A. and Yalow, R. S. (1968). Radioimmunoassay of ACTH in Plasma. *J. Clin. Invest.* 47, 2725-2751.
- Bigland-Ritchie, B., Johansson, R., Lippold, O. C. and Woods, J. J. (1983). Contractile Speed and EMG Changes during Fatigue of Sustained Maximal Voluntary Contractions. *J. Neurophysiol.* 50, 313-324.

- Billat, L. V. (2001). Interval Training for Performance: A Scientific and Empirical Practice. Special Recommendations for Middle- and Long-Distance Running. Part I: Aerobic Interval Training. *Sports Med.* 31, 13-31.
- Billat, L. V. and Koralsztein, J. P. (1996). Significance of the Velocity at VO₂max and Time to Exhaustion at this Velocity. *Sports Med.* 22, 90-108.
- Billat, V., Renoux, J. C., Pinoteau, J., Petit, B. and Koralsztein, J. P. (1994a). Reproducibility of Running Time to Exhaustion at VO₂max in Subelite Runners. *Med. Sci. Sports Exerc.* 26, 254-257.
- Billat, V., Renoux, J. C., Pinoteau, J., Petit, B. and Koralsztein, J. P. (1994b). Times to Exhaustion at 100% of Velocity at VO₂max and Modelling of the Time-limit/velocity Relationship in Elite Long-Distance Runners. *Eur. J. Appl. Physiol. Occup. Physiol.* 69, 271-273.
- Billat, V., Renoux, J. C., Pinoteau, J., Petit, B. and Koralsztein, J. P. (1995). Times to Exhaustion at 90, 100 and 105% of Velocity at VO₂ Max (Maximal Aerobic Speed) and Critical Speed in Elite Long-Distance Runners. *Arch. Physiol. Biochem.* 103, 129-135.
- Billat, V. L., Blondel, N. and Berthoin, S. (1999a). Determination of the Velocity Associated with the Longest Time to Exhaustion at Maximal Oxygen Uptake. *Eur. J. Appl. Physiol. Occup. Physiol.* 80, 159-161.
- Billat, V. L., Flechet, B., Petit, B., Muriaux, G. and Koralsztein, J. P. (1999b). Interval Training at VO₂max: Effects on Aerobic Performance and Overtraining Markers. *Med. Sci. Sports Exerc.* 31, 156-163.
- Billat, V. L., Hill, D. W., Pinoteau, J., Petit, B. and Koralsztein, J. P. (1996). Effect of Protocol on Determination of Velocity at VO₂ Max and on its Time to Exhaustion. *Arch. Physiol. Biochem.* 104, 313-321.
- Billat, V. L., Slawinski, J., Bocquet, V., Demarle, A., Lafitte, L., Chassaing, P. and Koralsztein, J. P. (2000). Intermittent Runs at the Velocity Associated with Maximal Oxygen Uptake Enables Subjects to Remain at Maximal Oxygen Uptake for a Longer Time than Intense but Submaximal Runs. *Eur. J. Appl. Physiol.* 81, 188-196.
- Bosco, C., Belli, A., Astrua, M., Tihanyi, J., Pozzo, R., Kellis, S., Tsarpela, O., Foti, C., Manno, R. and Tranquilli, C. (1995). A Dynamometer for Evaluation of Dynamic Muscle Work. *Eur. J. Appl. Physiol. Occup. Physiol.* 70, 379-386.
- Bosco, C., Colli, R., Bonomi, R., von Duvillard, S. P. and Viru, A. (2000). Monitoring Strength Training: Neuromuscular and Hormonal Profile. *Med. Sci. Sports Exerc.* 32, 202-208.
- Bosco, C., Komi, P. V. and Ito, A. (1981). Prestretch Potentiation of Human Skeletal Muscle during Ballistic Movement. *Acta Physiol. Scand.* 111, 135-140.
- Bosco, C., Montanari, G., Ribacchi, R., Giovenali, P., Latteri, F., Iachelli, G., Faina, M., Colli, R., Dal Monte, A. and La Rosa, M. (1987). Relationship between the Efficiency of Muscular Work during Jumping and the Energetics of Running. *Eur. J. Appl. Physiol. Occup. Physiol.* 56, 138-143.

- Bosco, C., Tihanyi, J., Komi, P. V., Fekete, G. and Apor, P. (1982). Store and Recoil of Elastic Energy in Slow and Fast Types of Human Skeletal Muscles. *Acta Physiol. Scand.* 116, 343-349.
- Bosco, C. and Viitasalo, J. T. (1982). Potentiation of Myoelectrical Activity of Human Muscles in Vertical Jumps. *Electromyogr. Clin. Neurophysiol.* 22, 549-562.
- Brooks, G. A. (1986). The Lactate Shuttle during Exercise and Recovery. *Med. Sci. Sports Exerc.* 18, 360-368.
- Brooks, G. A. (1991). Current Concepts in Lactate Exchange. *Med. Sci. Sports Exerc.* 23, 895-906.
- Brooks, G. A. and Fahey, T. (1984). *Exercise Physiology. Human Bioenergetics and its Application*, Macmillan, New York.
- Burfoot, A. and Billing, B. (1999). The Perfect Pace. *Runner's World* 20, 39-83.
- Cavanagh, P. R. and Williams, K. R. (1982). The Effect of Stride Length Variation on Oxygen Uptake during Distance Running. *Med. Sci. Sports Exerc.* 14, 30-35.
- Chasiotis, D. (1983). The Regulation of Glycogen Phosphorylase and Glycogen Breakdown in Human Skeletal Muscle. *Acta Physiol. Scand. Suppl.* 518, 1-68.
- Christensen, E. H., Hedman, R. and Saltin, B. (1960). Intermittent and Continuous Running. (A further Contribution to the Physiology of Intermittent Work.). *Acta Physiol. Scand.* 50, 269-286.
- Clark, G. T. and Carter, M. C. (1985). Electromyographic Study of Human Jaw-Closing Muscle Endurance, Fatigue and Recovery at various Isometric Force Levels. *Arch. Oral Biol.* 30, 563-569.
- Conley, D. L. and Krahenbuhl, G. S. (1980). Running Economy and Distance Running Performance of Highly Trained Athletes. *Med. Sci. Sports Exerc.* 12, 357-360.
- Cooper, D. M. and Barstow, T. J. (1996). Magnetic Resonance Imaging and Spectroscopy in Studying Exercise in Children. *Exerc. Sport Sci. Rev.* 24, 475-499.
- Costill, D. L. (1967). The Relationship between Selected Physiological Variables and Distance Running Performance. *J. Sports Med. Phys. Fitness* 7, 61-66.
- Costill, D. L., Daniels, J., Evans, W., Fink, W., Krahenbuhl, G. and Saltin, B. (1976). Skeletal Muscle Enzymes and Fiber Composition in Male and Female Track Athletes. *J. Appl. Physiol.* 40, 149-154.
- Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R. and Park, S. H. (1988). Effects of Repeated Days of Intensified Training on Muscle Glycogen and Swimming Performance. *Med. Sci. Sports Exerc.* 20, 249-254.
- Costill, D. L., Thomason, H. and Roberts, E. (1973). Fractional Utilization of the Aerobic Capacity during Distance Running. *Med. Sci. Sports* 5, 248-252.
- Costill, D. L. and Winrow, E. (1970a). A Comparison of Two Middle-Aged Ultramarathon Runners. *Res. Q.* 41, 135-139.

- Costill, D. L. and Winrow, E. (1970b). Maximal Oxygen Intake among Marathon Runners. *Arch. Phys. Med. Rehabil.* 51, 317-320.
- Cumming, D. C., Brunsting, L. A., 3rd, Strich, G., Ries, A. L. and Rebar, R. W. (1986). Reproductive Hormone Increases in Response to Acute Exercise in Men. *Med. Sci. Sports Exerc.* 18, 369-373.
- Cumming, D. C., Wheeler, G. D. and McColl, E. M. (1989). The Effects of Exercise on Reproductive Function in Men. *Sports Med.* 7, 1-17.
- Daly, W., Seegers, C. A., Rubin, D. A., Dobridge, J. D. and Hackney, A. C. (2005). Relationship between Stress Hormones and Testosterone with Prolonged Endurance Exercise. *Eur. J. Appl. Physiol.* 93, 375-380.
- Daniels, J. (1974). Physiological Characteristics of Champion Male Athletes. *Res. Q.* 45, 342-348.
- Daniels, J. (1998). Daniels's Running Formula. Human Kinetics, Champaign, IL.
- Daniels, J. and Daniels, N. (1992). Running Economy of Elite Male and Elite Female Runners. *Med. Sci. Sports Exerc.* 24, 483-489.
- Daniels, J. and Scardina, N. (1984). Interval Training and Performance. *Sports Med.* 1, 327-334.
- Daniels, J. T., Scardina, N. and Hayes, J. (1986). Elite and Subelite Female Middle- and Long-Distance Runners. In: Landers, D. M. (ed.) *Sport and Elite Performers*, Human Kinetics, Champaign, IL, pp. 57-72.
- Daniels, J. T., Yarbrough, R. A. and Foster, C. (1978). Changes in VO₂ Max and Running Performance with Training. *Eur. J. Appl. Physiol. Occup. Physiol.* 39, 249-254.
- Davies, C. T. and Thompson, M. W. (1979a). Aerobic Performance of Female Marathon and Male Ultramarathon Athletes. *Eur. J. Appl. Physiol. Occup. Physiol.* 41, 233-245.
- Davies, C. T. and Thompson, M. W. (1979b). Estimated Aerobic Performance and Energy Cost of Severe Exercise of 24 h Duration. *Ergonomics* 22, 1249-1255.
- Davies, C. T. and Thompson, M. W. (1986). Physiological Responses to Prolonged Exercise in Ultramarathon Athletes. *J. Appl. Physiol.* 61, 611-617.
- Davies, K. J., Quintanilha, A. T., Brooks, G. A. and Packer, L. (1982). Free Radicals and Tissue Damage Produced by Exercise. *Biochem. Biophys. Res. Commun.* 107, 1198-1205.
- Del Maestro, R. F. (1980). An Approach to Free Radicals in Medicine and Biology. *Acta Physiol. Scand. Suppl.* 492, 153-168.
- Denis, C., Linnossier, M. T., Dormois, D., Padilla, S., Geysant, A., Lacour, J. R. and Inbar, O. (1992). Power and Metabolic Responses during Supramaximal Exercise in 100-m and 800-m Runners. *Scand. J. Med. Sci. Sports* 2, 62-69.
- Di Prampero, P. E., Capelli, C., Pagliaro, P., Antonutto, G., Girardis, M., Zamparo, P. and Soule, R. G. (1993). Energetics of Best Performances in Middle-Distance Running. *J. Appl. Physiol.* 74, 2318-2324.

- Dietz, V., Schmidtbleicher, D. and Noth, J. (1979). Neuronal Mechanisms of Human Locomotion. *J. Neurophysiol.* 42, 1212-1222.
- Dillard, C. J., Litov, R. E., Savin, W. M., Dumelin, E. E. and Tappel, A. L. (1978). Effects of Exercise, Vitamin E, and Ozone on Pulmonary Function and Lipid Peroxidation. *J. Appl. Physiol.* 45, 927-932.
- Duclos, M., Corcuff, J. B., Rashedi, M., Fougere, V. and Manier, G. (1996). Does Functional Alteration of the Gonadotropic Axis Occur in Endurance Trained Athletes during and After Exercise? A Preliminary Study. *Eur. J. Appl. Physiol. Occup. Physiol.* 73, 427-433.
- Durstine, J. L. and Haskell, W. L. (1994). Effects of Exercise Training on Plasma Lipids and Lipoproteins. *Exerc. Sport Sci. Rev.* 22, 477-521.
- Edwards, R. H. (1981). Human Muscle Function and Fatigue. In: *Human Muscle Fatigue: Physiological Mechanisms*, Pitman Medical, London, pp. 1-18
- Eik-Nes, K. B. (1969). An Effect of Isoproterenol on Rates of Synthesis and Secretion of Testosterone. *Am. J. Physiol.* 217, 1764-1770.
- Enoka, R. M. (1988a). Muscle Strength and its Development. *New Perspectives. Sports Med.* 6, 146-168.
- Enoka, R. M. (1988b). *Neuromechanical Basis of Kinesiology, Human Kinetics*, Champaign, Ill, pp. 201-206, 336.
- Fahrner, C. L. and Hackney, A. C. (1998). Effects of Endurance Exercise on Free Testosterone Concentration and the Binding Affinity of Sex Hormone Binding Globulin (SHBG). *Int. J. Sports Med.* 19, 12-15.
- Farrell, P. A., Wilmore, J. H., Coyle, E. F., Billing, J. E. and Costill, D. L. (1979). Plasma Lactate Accumulation and Distance Running Performance. *Med. Sci. Sports* 11, 338-344.
- Ferretti, G., Gussoni, M., Di Prampero, P. E. and Cerretelli, P. (1987). Effects of Exercise on Maximal Instantaneous Muscular Power of Humans. *J. Appl. Physiol.* 62, 2288-2294.
- Foster, C. (1983). VO₂max and Training Indices as Determinants of Competitive Running Performance. *J. Sport Sci.* 1, 13-22.
- Foster, C., Costill, D. L., Daniels, J. T. and Fink, W. J. (1978). Skeletal Muscle Enzyme Activity, Fiber Composition and VO₂ Max in Relation to Distance Running Performance. *Eur. J. Appl. Physiol. Occup. Physiol.* 39, 73-80.
- Galbo, H. (1981). Endocrinology and Metabolism in Exercise. *Int. J. Sports Med.* 2, 203-211.
- Galbo, H., Hummer, L., Peterson, I. B., Christensen, N. J. and Bie, N. (1977). Thyroid and Testicular Hormone Responses to Graded and Prolonged Exercise in Man. *Eur. J. Appl. Physiol. Occup. Physiol.* 36, 101-106.
- Gandevia, S. C. (1998). Neural Control in Human Muscle Fatigue: Changes in Muscle Afferents, Motoneurons and Motor Cortical Drive [Corrected]. *Acta Physiol. Scand.* 162, 275-283.
- Gandevia, S. C. (2001). Spinal and Supraspinal Factors in Human Muscle Fatigue. *Physiol. Rev.* 81, 1725-1789.

- Gastin, P. B. and Lawson, D. L. (1994). Influence of Training Status on Maximal Accumulated Oxygen Deficit during all-Out Cycle Exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 69, 321-330.
- Gibson, H. and Edwards, R. H. (1985). Muscular Exercise and Fatigue. *Sports Med.* 2, 120-132.
- Ginsburg, G. S., Agil, A., O'Toole, M., Rimm, E., Douglas, P. S. and Rifai, N. (1996). Effects of a Single Bout of Ultraendurance Exercise on Lipid Levels and Susceptibility of Lipids to Peroxidation in Triathletes. *JAMA* 276, 221-225.
- Gollhofer, A., Komi, P. V., Fujitsuka, N. and Miyashita, M. (1987a). Fatigue during Stretch-Shortening Cycle Exercises. II. Changes in Neuromuscular Activation Patterns of Human Skeletal Muscle. *Int. J. Sports Med.* 8 Suppl. 1, 38-47.
- Gollhofer, A., Komi, P. V., Miyashita, M. and Aura, O. (1987b). Fatigue during Stretch-Shortening Cycle Exercises: Changes in Mechanical Performance of Human Skeletal Muscle. *Int. J. Sports Med.* 8, 71-78.
- Gollnick, P. D., Armstrong, R. B., Saubert, C. W., Piehl, K. and Saltin, B. (1972). Enzyme Activity and Fiber Composition in Skeletal Muscle of Untrained and Trained Men. *J. Appl. Physiol.* 33, 312-319.
- Gorostiaga, E. M., Izquierdo, M., Ruesta M., Iribarren J., Gonzalez-Badillo J. J., Ibanez, J. (2004). Strength training effects on physical performance and serum hormones in young soccer players. *Eur. J. Appl. Physiol.* 91, 698-707.
- Grant, S., Craig, I., Wilson, J. and Aitchison, T. (1997). The Relationship between 3 km Running Performance and Selected Physiological Variables. *J. Sports Sci.* 15, 403-410.
- Green, H. J. (1997). Mechanisms of Muscle Fatigue in Intense Exercise. *J. Sports Sci.* 15, 247-256.
- Green, H. J., Coates, G., Sutton, J. R. and Jones, S. (1991a). Early Adaptations in Gas Exchange, Cardiac Function and Haematology to Prolonged Exercise Training in Man. *Eur. J. Appl. Physiol. Occup. Physiol.* 63, 17-23.
- Green, H. J., Helyar, R., Ball-Burnett, M., Kowalchuk, N., Symon, S. and Farrance, B. (1992). Metabolic Adaptations to Training Precede Changes in Muscle Mitochondrial Capacity. *J. Appl. Physiol.* 72, 484-491.
- Green, H. J., Jones, L. L., Houston, M. E., Ball-Burnett, M. E. and Farrance, B. W. (1989). Muscle Energetics during Prolonged Cycling After Exercise Hypervolemia. *J. Appl. Physiol.* 66, 622-631.
- Green, H. J., Jones, L. L., Hughson, R. L., Painter, D. C. and Farrance, B. W. (1987). Training-Induced Hypervolemia: Lack of an Effect on Oxygen Utilization during Exercise. *Med. Sci. Sports Exerc.* 19, 202-206.
- Green, H. J., Jones, L. L. and Painter, D. C. (1990). Effects of Short-Term Training on Cardiac Function during Prolonged Exercise. *Med. Sci. Sports Exerc.* 22, 488-493.

- Green, H. J., Jones, S., Ball-Burnett, M. and Fraser, I. (1991b). Early Adaptations in Blood Substrates, Metabolites, and Hormones to Prolonged Exercise Training in Man. *Can. J. Physiol. Pharmacol.* 69, 1222-1229.
- Green, H. J. and Patla, A. E. (1992). Maximal Aerobic Power: Neuromuscular and Metabolic Considerations. *Med. Sci. Sports Exerc.* 24, 38-46.
- Gullstrand, L. and Lawrence, S. (1987). Heart Rate and Blood Lactate Response to Short Intermittent Work at Race Pace in Highly Trained Swimmers. *Aust. J. Sci. Med. Sport* 19, 10-14.
- Hackney, A. C., Premo, M. C. and McMurray, R. G. (1995). Influence of Aerobic Versus Anaerobic Exercise on the Relationship between Reproductive Hormones in Men. *J. Sports Sci.* 13, 305-311.
- Hakkinen, K. (1993). Neuromuscular Fatigue and Recovery in Male and Female Athletes during Heavy Resistance Exercise. *Int. J. Sports Med.* 14, 53-59.
- Hakkinen, K., Alen, M., Kallinen, M., Newton, R. U. and Kraemer, W. J. (2000). Neuromuscular Adaptation during Prolonged Strength Training, Detraining and Re-Strength-Training in Middle-Aged and Elderly People. *Eur. J. Appl. Physiol.* 83, 51-62.
- Hakkinen, K. and Komi, P. V. (1983). Changes in Neuromuscular Performance in Voluntary and Reflex Contraction during Strength Training in Man. *Int. J. Sports Med.* 4, 282-288.
- Hakkinen, K. and Komi, P. V. (1986). Effects of Fatigue and Recovery on Electromyographic and Isometric Force- and Relaxation-Time Characteristics of Human Skeletal Muscle. *Eur. J. Appl. Physiol. Occup. Physiol.* 55, 588-596.
- Hakkinen, K., Komi, P. V. and Alen, M. (1985). Effect of Explosive Type Strength Training on Isometric Force- and Relaxation-Time, Electromyographic and Muscle Fibre Characteristics of Leg Extensor Muscles. *Acta Physiol. Scand.* 125, 587-600.
- Hakkinen, K. and Pakarinen, A. (1993). Acute Hormonal Responses to Two Different Fatiguing Heavy-Resistance Protocols in Male Athletes. *J. Appl. Physiol.* 74, 882-887.
- Halliwell, B. (1994). Free Radicals, Antioxidants, and Human Disease: Curiosity, Cause, Or Consequence? *Lancet* 344, 721-724.
- Haramäki, N. and Packer, L. (1994). Oxidative Stress Indices in Exercise. In: Sen, C. K., Packer, L., and Hänninen, O. (eds.) *Exercise and Oxygen Toxicity*, Elsevier, Amsterdam, pp. 77-87.
- Hawley, J. A., Myburgh, K. H., Noakes, T. D. and Dennis, S. C. (1997). Training Techniques to Improve Fatigue Resistance and Enhance Endurance Performance. *J. Sports Sci.* 15, 325-333.
- Henriksson, J. (1992). Effects of Physical Training on the Metabolism of Skeletal Muscle. *Diabetes Care* 15, 1701-1711.
- Hill, A. V. and Lupton, H. (1923). Muscular Exercise, Lactic Acid and the Supply and Utilization of Oxygen. *Qart. Med. J.* 16, 135-71.
- Hill, D. W. and Rowell, A. L. (1997). Responses to Exercise at the Velocity Associated with VO₂max. *Med. Sci. Sports Exerc.* 29, 113-116.

- Hirvonen, J., Nummela, A., Rusko, H., Rehunen, S. and Harkonen, M. (1992). Fatigue and Changes of ATP, Creatine Phosphate, and Lactate during the 400-m Sprint. *Can. J. Sport Sci.* 17, 141-144.
- Hirvonen, J., Rehunen, S., Rusko, H. and Harkonen, M. (1987). Breakdown of High-Energy Phosphate Compounds and Lactate Accumulation during Short Supramaximal Exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 56, 253-259.
- Hultman, E. and Spriet, L. L. (1986). Skeletal Muscle Metabolism, Contraction Force and Glycogen Utilization during Prolonged Electrical Stimulation in Humans. *J. Physiol.* 374, 493-501.
- Izquierdo M., Gonzalez-Badillo, J. J., Häkkinen, K., Ibanez, J., Kraemer, W. J., Altadill, A. (2006). Effect of loading on unintentional lifting velocity declines during single sets of repetitions to failure during upper and lower extremity muscle actions. *Int. J. Sports Med.* 29, 718-724.
- James, D. V. and Doust, J. H. (1998). Oxygen Uptake during Moderate Intensity Running: Response Following a Single Bout of Interval Training. *Eur. J. Appl. Physiol. Occup. Physiol.* 77, 551-555.
- James, D. V. and Doust, J. H. (1999). Oxygen Uptake during High-Intensity Running: Response Following a Single Bout of Interval Training. *Eur. J. Appl. Physiol. Occup. Physiol.* 79, 237-243.
- Jenkins, R. R. (1988). Free Radical Chemistry. Relationship to Exercise. *Sports Med.* 5, 156-170.
- Jensen, J., Oftebro, H., Breigan, B., Johnsson, A., Ohlin, K., Meen, H. D., Stromme, S. B. and Dahl, H. A. (1991). Comparison of Changes in Testosterone Concentrations After Strength and Endurance Exercise in Well Trained Men. *Eur. J. Appl. Physiol. Occup. Physiol.* 63, 467-471.
- Jezova, D. and Vigas, M. (1981). Testosterone Response to Exercise during Blockade and Stimulation of Adrenergic Receptors in Man. *Horm. Res.* 15, 141-147.
- Ji, L. L. (1995). Oxidative Stress during Exercise: Implication of Antioxidant Nutrients. *Free Radic. Biol. Med.* 18, 1079-1086.
- Ji, L. L. (1996). Exercise, Oxidative Stress, and Antioxidants. *Am. J. Sports Med.* 24, S20-4.
- Joyner, M. J. (1991). Modeling: Optimal Marathon Performance on the Basis of Physiological Factors. *J. Appl. Physiol.* 70, 683-687.
- Kargotich, S., Goodman, C., Keast, D., Fry, R. W., Garcia-Webb, P., Crawford, P. M. and Morton, A. R. (1997). Influence of Exercise-Induced Plasma Volume Changes on the Interpretation of Biochemical Data Following High-Intensity Exercise. *Clin. J. Sport Med.* 7, 185-191.
- Kasvinsky, P. J. and Meyer, W. L. (1977). The Effect of pH and Temperature on the Kinetics of Native and Altered Glycogen Phosphorylase. *Arch. Biochem. Biophys.* 181, 616-631.
- Kayser, B., Narici, M., Binzoni, T., Grassi, B. and Cerretelli, P. (1994). Fatigue and Exhaustion in Chronic Hypobaric Hypoxia: Influence of Exercising Muscle Mass. *J. Appl. Physiol.* 76, 634-640.

- Kenefick, R. W., Maresh, C. M., Armstrong, L. E., Castellani, J. W., Whittlesey, M., Hoffman, J. R. and Bergeron, M. F. (1998). Plasma Testosterone and Cortisol Responses to Training-Intensity Exercise in Mild and Hot Environments. *Int. J. Sports Med.* 19, 177-181.
- Kindermann, W., Schnabel, A., Schmitt, W. M., Biro, G., Cassens, J. and Weber, F. (1982). Catecholamines, Growth Hormone, Cortisol, Insulin, and Sex Hormones in Anaerobic and Aerobic Exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 49, 389-399.
- Kjaer, M. (1992). Regulation of hormonal and metabolic responses during exercise in humans. *Exerc. Sport Sci. Rev.* 20, 161-184.
- Komi, P. V. (1984) Physiological and Biomechanical Correlates of Muscle Function: Effects of Muscle Structure and Stretch-shortening Cycle on Force and Speed. In: Terjung, R. L. (ed.) *Exercise and Sport Science Reviews*. Collamore, Lexington, Mass, pp. 81-121.
- Komi, P. V. (1984). Biomechanics and Neuromuscular Performance. *Med. Sci. Sports Exerc.* 16, 26-28.
- Komi, P. V. (1986). Training of Muscle Strength and Power: Interaction of Neuromotoric, Hypertrophic, and Mechanical Factors. *Int. J. Sports Med.* 7 Suppl 1, 10-15.
- Komi, P. V. and Karlsson, J. (1979). Physical Performance, Skeletal Muscle Enzyme Activities, and Fibre Types in Monozygous and Dizygous Twins of both Sexes. *Acta Physiol. Scand. Suppl.* 462, 1-28.
- Komi, P. V., Viitasalo, J. H., Havu, M., Thorstensson, A., Sjodin, B. and Karlsson, J. (1977). Skeletal Muscle Fibres and Muscle Enzyme Activities in Monozygous and Dizygous Twins of both Sexes. *Acta Physiol. Scand.* 100, 385-392.
- Komi, P. V. (1991) (ed.) *Strength and Power in Sport*, Blackwell Scientific Publications, Oxford, pp. 169-179, 404.
- Kraemer, W. J., Dziados, J. E., Marchitelli, L. J., Gordon, S. E., Harman, E. A., Mello, R., Fleck, S. J., Frykman, P. N. and Triplett, N. T. (1993). Effects of Different Heavy-Resistance Exercise Protocols on Plasma Beta-Endorphin Concentrations. *J. Appl. Physiol.* 74, 450-459.
- Kraemer, W. J., Marchitelli, L., Gordon, S. E., Harman, E., Dziados, J. E., Mello, R., Frykman, P., McCurry, D. and Fleck, S. J. (1990). Hormonal and Growth Factor Responses to Heavy Resistance Exercise Protocols. *J. Appl. Physiol.* 69, 1442-1450.
- Kreuschmar, M., Pfeifer, G., Machnick, G. and Klinger, W. (1992). Glutathione Homeostasis and Turnover in the Totally Hepatectomized Rat: Evidence for a High Glutathione Export Capacity of Extrahepatic Tissues. *Exp. Toxicol. Pathol.* 44, 273-281.
- Kujala, U. M., Ahotupa, M., Vasankari, T., Kaprio, J. and Tikkanen, M. J. (1996). Low LDL Oxidation in Veteran Endurance Athletes. *Scand. J. Med. Sci. Sports* 6, 303-308.

- Kumagai, S., Tanaka, K., Matsuura, Y., Matsuzaka, A., Hirakoba, K. and Asano, K. (1982). Relationships of the Anaerobic Threshold with the 5 km, 10 km, and 10 Mile Races. *Eur. J. Appl. Physiol. Occup. Physiol.* 49, 13-23.
- Kuoppasalmi, K., Naveri, H., Harkonen, M. and Adlercreutz, H. (1980). Plasma Cortisol, Androstenedione, Testosterone and Luteinizing Hormone in Running Exercise of Different Intensities. *Scand. J. Clin. Lab. Invest.* 40, 403-409.
- Kuoppasalmi, K., Naveri, H., Rehunen, S., Harkonen, M. and Adlercreutz, H. (1976). Effect of Strenuous Anaerobic Running Exercise on Plasma Growth Hormone, Cortisol, Luteinizing Hormone, Testosterone, Androstenedione, Estrone and Estradiol. *J. Steroid Biochem.* 7, 823-829.
- Kyrolainen, H., Avela, J. and Komi, P. V. (2005). Changes in Muscle Activity with Increasing Running Speed. *J. Sports Sci.* 23, 1101-1109.
- Lacour, J. R., Padilla-Magunacelaya, S., Barthelemy, J. C. and Dormois, D. (1990). The Energetics of Middle-Distance Running. *Eur. J. Appl. Physiol. Occup. Physiol.* 60, 38-43.
- Lattier, G., Millet, G. Y., Martin, A. and Martin, V. (2004). Fatigue and Recovery After High-Intensity Exercise Part I: Neuromuscular Fatigue. *Int. J. Sports Med.* 25, 450-456.
- Laursen, P. B. and Jenkins, D. G. (2002). The Scientific Basis for High-Intensity Interval Training: Optimising Training Programmes and Maximising Performance in Highly Trained Endurance Athletes. *Sports Med.* 32, 53-73.
- Lepers, R., Hausswirth, C., Maffiuletti, N., Brisswalter, J. and van Hoecke, J. (2000a). Evidence of Neuromuscular Fatigue After Prolonged Cycling Exercise. *Med. Sci. Sports Exerc.* 32, 1880-1886.
- Lepers, R., Pousson, M. L., Maffiuletti, N. A., Martin, A. and Van Hoecke, J. (2000b). The Effects of a Prolonged Running Exercise on Strength Characteristics. *Int. J. Sports Med.* 21, 275-280.
- Lewis, S. F. and Haller, R. G. (1986). The Pathophysiology of McArdle's Disease: Clues to Regulation in Exercise and Fatigue. *J. Appl. Physiol.* 61, 391-401.
- Liefeldt, G., Noakes, T. D. and Dennis, S. C. (1992). Oxygen Delivery does Not Limit Peak Running Speed during Incremental Downhill Running to Exhaustion. *Eur. J. Appl. Physiol. Occup. Physiol.* 64, 493-496.
- Lindsay, F. H., Hawley, J. A., Myburgh, K. H., Schomer, H. H., Noakes, T. D. and Dennis, S. C. (1996). Improved Athletic Performance in Highly Trained Cyclists After Interval Training. *Med. Sci. Sports Exerc.* 28, 1427-1434.
- Londeree, B. R. (1997). Effect of Training on lactate/ventilatory Thresholds: A Meta-Analysis. *Med. Sci. Sports Exerc.* 29, 837-843.
- Lovlin, R., Cottle, W., Pyke, I., Kavanagh, M. and Belcastro, A. N. (1987). Are Indices of Free Radical Damage Related to Exercise Intensity. *Eur. J. Appl. Physiol. Occup. Physiol.* 56, 313-316.
- MacIntosh, B. R. and Rassier, D. E. (2002). What is Fatigue? *Can. J. Appl. Physiol.* 27, 42-55.

- Mainwood, G. W. and Renaud, J. M. (1985). The Effect of Acid-Base Balance on Fatigue of Skeletal Muscle. *Can. J. Physiol. Pharmacol.* 63, 403-416.
- Mathur, D. N., Toriola, A. L. and Dada, O. A. (1986). Serum Cortisol and Testosterone Levels in Conditioned Male Distance Runners and Nonathletes After Maximal Exercise. *J. Sports Med. Phys. Fitness* 26, 245-250.
- Medbo, J. I., Mohn, A. C., Tabata, I., Bahr, R., Vaage, O. and Sejersted, O. M. (1988). Anaerobic Capacity Determined by Maximal Accumulated O₂ Deficit. *J. Appl. Physiol.* 64, 50-60.
- Medbo, J. I. and Tabata, I. (1989). Relative Importance of Aerobic and Anaerobic Energy Release during Short-Lasting Exhausting Bicycle Exercise. *J. Appl. Physiol.* 67, 1881-1886.
- Medbo, J. I. and Tabata, I. (1993). Anaerobic Energy Release in Working Muscle during 30 s to 3 Min of Exhausting Bicycling. *J. Appl. Physiol.* 75, 1654-1660.
- Mero, A., Kauhanen, H., Peltola, E., Vuorimaa, T. and Komi, P. V. (1990). Physiological Performance Capacity in Different Prepubescent Athletic Groups. *J. Sports Med. Phys. Fitness* 30, 57-66.
- Millet, G., Lepers, R., Lattier, G., Martin, V., Babault, N. and Maffiuletti, N. (2000). Influence of Ultra-Long-Term Fatigue on the Oxygen Cost of Two Types of Locomotion. *Eur. J. Appl. Physiol.* 83, 376-380.
- Millet, G. Y. and Lepers, R. (2004). Alterations of Neuromuscular Function After Prolonged Running, Cycling and Skiing Exercises. *Sports Med.* 34, 105-116.
- Millet, G. Y., Lepers, R., Maffiuletti, N. A., Babault, N., Martin, V. and Lattier, G. (2002). Alterations of Neuromuscular Function After an Ultramarathon. *J. Appl. Physiol.* 92, 486-492.
- Millet, G. Y., Martin, V., Lattier, G. and Ballay, Y. (2003). Mechanisms Contributing to Knee Extensor Strength Loss After Prolonged Running Exercise. *J. Appl. Physiol.* 94, 193-198.
- Milne, D. B. and Botnen, J. (1986). Retinol, Alpha-Tocopherol, Lycopene, and Alpha- and Beta-Carotene Simultaneously Determined in Plasma by Isocratic Liquid Chromatography. *Clin. Chem.* 32, 874-876.
- Mooradian, A. D., Habib, M. P. and Dickerson, F. (1994). Effect of Simple Carbohydrates, Casein Hydrolysate, and a Lipid Test Meal on Ethane Exhalation Rate. *J. Appl. Physiol.* 76, 1119-1122.
- Morgan, D. W., Baldini, F. D., Martin, P. E. and Kohrt, W. M. (1989). Ten Kilometer Performance and Predicted Velocity at VO₂max among Well-Trained Male Runners. *Med. Sci. Sports Exerc.* 21, 78-83.
- Moritani, T., Oddson, L. and Thorstensson, A. (1990). Electromyographic Evidence of Selective Fatigue during the Eccentric Phase of stretch/shortening Cycles in Man. *Eur. J. Appl. Physiol. Occup. Physiol.* 60, 425-429.
- Newsholme, E. A. (1976). Carbohydrate Metabolism in Vivo: Regulation of the Blood Glucose Level. *Clin. Endocrinol. Metab.* 5, 543-578.

- Newsholme, E. A. (1978). Substrate Cycles: Their Metabolic, Energetic and Thermic Consequences in Man. *Biochem. Soc. Symp.* (43), 183-205.
- Newsholme, E. A., Zammit, V. A. and Crabtree, B. (1978). The Role of Glucose and Glycogen as Fuels for Muscle. *Biochem. Soc. Trans.* 6, 512-520.
- Nicol, C., Komi, P. V. and Marconnet, P. (1991a). Fatigue Effects of Marathon Running on Neuromuscular Performance. I Changes in Muscular Force and Stiffness Characteristics. *Scand. J. Med. Sci. Sports* 1, 10-17.
- Nicol, C., Komi, P. V. and Marconnet, P. (1991b). Fatigue Effects of Marathon Running on Neuromuscular Performance. II Changes in Force, Integrated Electromyographic Activity and Endurance Capacity. *Scand. J. Med. Sci. Sports* 1, 18-24.
- Noakes, T. (2001). *Lore of Running*, 4th Edition. Human Kinetics, Leeds, pp. 25-47, 297-300, 438-442.
- Noakes, T. D. (1988). Implications of Exercise Testing for Prediction of Athletic Performance: A Contemporary Perspective. *Med. Sci. Sports Exerc.* 20, 319-330.
- Noakes, T. D. (1997). 1996 J.B. Wolffe Memorial Lecture. Challenging Beliefs: Ex Africa Semper Aliquid Novi. *Med. Sci. Sports Exerc.* 29, 571-590.
- Noakes, T. D. (1998). Maximal Oxygen Uptake: "Classical" Versus "Contemporary" Viewpoints: A Rebuttal. *Med. Sci. Sports Exerc.* 30, 1381-1398.
- Noakes, T. D., Myburgh, K. H. and Schall, R. (1990). Peak Treadmill Running Velocity during the VO₂ Max Test Predicts Running Performance. *J. Sports Sci.* 8, 35-45.
- Nummela, A. and Rusko, H. (1995). Time Course of Anaerobic and Aerobic Energy Expenditure during Short-Term Exhaustive Running in Athletes. *Int. J. Sports Med.* 16, 522-527.
- Nummela, A., Rusko, H. and Mero, A. (1994). EMG Activities and Ground Reaction Forces during Fatigued and Nonfatigued Sprinting. *Med. Sci. Sports Exerc.* 26, 605-609.
- Nummela, A., Vuorimaa, T. and Rusko, H. (1992). Changes in Force Production, Blood Lactate and EMG Activity in the 400-m Sprint. *J. Sports Sci.* 10, 217-228.
- Paavolainen, L., Hakkinen, K., Hamalainen, I., Nummela, A. and Rusko, H. (1999a). Explosive-Strength Training Improves 5-km Running Time by Improving Running Economy and Muscle Power. *J. Appl. Physiol.* 86, 1527-1533.
- Paavolainen, L., Hakkinen, K., Nummela, A. and Rusko, H. (1994). Neuromuscular Characteristics and Fatigue in Endurance and Sprint Athletes during a New Anaerobic Power Test. *Eur. J. Appl. Physiol. Occup. Physiol.* 69, 119-126.
- Paavolainen, L., Nummela, A., Rusko, H. and Hakkinen, K. (1999b). Neuromuscular Characteristics and Fatigue during 10 km Running. *Int. J. Sports Med.* 20, 516-521.

- Paavolainen, L. M., Nummela, A. T. and Rusko, H. K. (1999c). Neuromuscular Characteristics and Muscle Power as Determinants of 5-km Running Performance. *Med. Sci. Sports Exerc.* 31, 124-130.
- Padilla, S., Bourdin, M., Barthelemy, J. C. and Lacour, J. R. (1992). Physiological Correlates of Middle-Distance Running Performance. A Comparative Study between Men and Women. *Eur. J. Appl. Physiol. Occup. Physiol.* 65, 561-566.
- Pfiztinger, P. and Douglas, S. (1999). *Road Racing for Serious Runners*. Human Kinetics, Champaign, IL.
- Pincemail, J., Deby, C., Camus, G., Pirnay, F., Bouchez, R., Massaux, L. and Goutier, R. (1988). Tocopherol Mobilization during Intensive Exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 57, 189-191.
- Pincemail, J., Lecomte, J., Castiau, J., Collard, E., Vasankari, T., Cheramy-Bien, J., Limet, R. and Defraigne, J. (2000). Evaluation of Autoantibodies Against Oxidized LDL and Antioxidant Status in Top Soccer and Basketball Players After 4 Months of Competition. *Free Radic. Biol. Med.* 28, 559-565.
- Place, N., Lepers, R., Deley, G. and Millet, G. Y. (2004). Time Course of Neuromuscular Alterations during a Prolonged Running Exercise. *Med. Sci. Sports Exerc.* 36, 1347-1356.
- Pollock, M. L. (1977). Submaximal and Maximal Working Capacity of Elite Distance Runners. Part I: Cardiorespiratory Aspects. *Ann. N. Y. Acad. Sci.* 301, 310-322.
- Psek, J. A. and Cafarelli, E. (1993). Behavior of Coactive Muscles during Fatigue. *J. Appl. Physiol.* 74, 170-175.
- Rassier, D. E. and MacIntosh, B. R. (2000). Coexistence of Potentiation and Fatigue in Skeletal Muscle. *Braz. J. Med. Biol. Res.* 33, 499-508.
- Remes, K., Kuoppasalmi, K. and Adlercreutz, H. (1985). Effect of Physical Exercise and Sleep Deprivation on Plasma Androgen Levels: Modifying Effect of Physical Fitness. *Int. J. Sports Med.* 6, 131-135.
- Rowell, L. B., Saltin, B., Kiens, B. and Christensen, N. J. (1986). Is Peak Quadriceps Blood Flow in Humans Even Higher during Exercise with Hypoxemia? *Am. J. Physiol.* 251, H1038-44.
- Rusko, H., Nummela, A. and Mero, A. (1993). A New Method for the Evaluation of Anaerobic Running Power in Athletes. *Eur. J. Appl. Physiol. Occup. Physiol.* 66, 97-101.
- Rusko, H. K. and Nummela, A. (1996). Measurement of Maximal and Submaximal Anaerobic Performance Capacity: Concluding Chapter. *Int. J. Sports Med.* 17 Suppl 2, S125-9.
- Sale, D. G. (1991). Neural Adaptation to Strength Training. In: Komi (ed.) *Strength and Power in Sports*, The Encyclopedia of Sports Medicine, Blacwell, Oxford, pp 249-265.
- Sale, D. G., MacDougall, J. D., Alway, S. E. and Sutton, J. R. (1987). Voluntary Strength and Muscle Characteristics in Untrained Men and Women and Male Bodybuilders. *J. Appl. Physiol.* 62, 1786-1793.

- Salminen, A. and Vihko, V. (1983). Lipid Peroxidation in Exercise Myopathy. *Exp. Mol. Pathol.* 38, 380-388.
- Schmid, P., Pusch, H. H., Wolf, W., Pilger, E., Pessenhofer, H., Schwabberger, G., Pristautz, H. and Purstner, P. (1982). Serum FSH, LH, and Testosterone in Humans After Physical Exercise. *Int. J. Sports Med.* 3, 84-89.
- Schwarz, L. and Kindermann, W. (1990). Beta-Endorphin, Adrenocorticotrophic Hormone, Cortisol and Catecholamines during Aerobic and Anaerobic Exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 61, 165-171.
- Scrimgeour, A. G., Noakes, T. D., Adams, B. and Myburgh, K. (1986). The Influence of Weekly Training Distance on Fractional Utilization of Maximum Aerobic Capacity in Marathon and Ultramarathon Runners. *Eur. J. Appl. Physiol. Occup. Physiol.* 55, 202-209.
- Sirin, A. V. and Patla, A. E. (1987). Myoelectric Changes in the Triceps Surae Muscles Under Sustained Contractions. Evidence for Synergism. *Eur. J. Appl. Physiol. Occup. Physiol.* 56, 238-244.
- Sjodin, B. and Svedenhag, J. (1985). Applied Physiology of Marathon Running. *Sports Med.* 2, 83-99.
- Slater, T. F. (1985). Overview of Methods Used for Detecting Lipid Peroxidation. *Methods Enzymol.* 105, 283-293.
- Slowinska-Lisowska, M. and Majda, J. (2002). Hormone Plasma Levels from Pituitary-Gonadal Axis in Performance Athletes After the 400 m Run. *J. Sports Med. Phys. Fitness* 42, 243-249.
- Smith, T. P., McNaughton, L. R. and Marshall, K. J. (1999). Effects of 4-Wk Training using Vmax/Tmax on VO₂max and Performance in Athletes. *Med. Sci. Sports Exerc.* 31, 892-896.
- Snegovskaya, V. and Viru, A. (1993). Steroid and Pituitary Hormone Responses to Rowing: Relative Significance of Exercise Intensity and Duration and Performance Level. *Eur. J. Appl. Physiol. Occup. Physiol.* 67, 59-65.
- Spencer, M. R. and Gastin, P. B. (2001). Energy System Contribution during 200- to 1500-m Running in Highly Trained Athletes. *Med. Sci. Sports Exerc.* 33, 157-162.
- Spriet, L. L., Berardinucci, L., Marsh, D. R., Campbell, C. B. and Graham, T. E. (1990). Glycogen Content has no Effect on Skeletal Muscle Glycogenolysis during Short-Term Tetanic Stimulation. *J. Appl. Physiol.* 68, 1883-1888.
- Spriet, L. L., Lindinger, M. I., McKelvie, R. S., Heigenhauser, G. J. and Jones, N. L. (1989). Muscle Glycogenolysis and H⁺ Concentration during Maximal Intermittent Cycling. *J. Appl. Physiol.* 66, 8-13.
- Spriet, L. L., Soderlund, K., Bergstrom, M. and Hultman, E. (1987). Anaerobic Energy Release in Skeletal Muscle during Electrical Stimulation in Men. *J. Appl. Physiol.* 62, 611-615.
- Steinberg, D. (1993). Antioxidant Vitamins and Coronary Heart Disease. *N. Engl. J. Med.* 328, 1487-1489.
- Steinberg, D. (1995). Role of Oxidized LDL and Antioxidants in Atherosclerosis. *Adv. Exp. Med. Biol.* 369, 39-48.

- Strojnik, V. and Komi, P. V. (2000). Fatigue After Submaximal Intensive Stretch-Shortening Cycle Exercise. *Med. Sci. Sports Exerc.* 32, 1314-1319.
- Sutton, J. and Lazarus, L. (1974). Effect of Adrenergic Blocking Agents on Growth Hormone Responses to Physical Exercise. *Horm. Metab. Res.* 6, 428-429.
- Sutton, J. R. (1978). Hormonal and Metabolic Responses to Exercise in Subject of High and Low Work Capacities. *Med. Sci. Sports* 10, 1-6.
- Sutton, J. R., Coleman, M. J., Casey, J. and Lazarus, L. (1973). Androgen Responses during Physical Exercise. *Br. Med. J.* 1, 520-522.
- Sutton, J. R., Coleman, M. J., Casey, J. H. (1974). The Adrenal Cortical Contribution to Serum Androgens in Physical Exercise. *Med. Sci. Sports* 6, 72.
- Tabata, I., Irisawa, K., Kouzaki, M., Nishimura, K., Ogita, F. and Miyachi, M. (1997). Metabolic Profile of High Intensity Intermittent Exercises. *Med. Sci. Sports Exerc.* 29, 390-395.
- Takada, M., Ikenoya, S., Yuzuriha, T. and Katayama, K. (1984). Simultaneous Determination of Reduced and Oxidized Ubiquinones. *Methods Enzymol.* 105, 147-155.
- Tanaka, K. and Matsuura, Y. (1984). Marathon Performance, Anaerobic Threshold, and Onset of Blood Lactate Accumulation. *J. Appl. Physiol.* 57, 640-643.
- Tate, C. A., Hyek, M. F. and Taffet, G. E. (1991). The Role of Calcium in the Energetics of Contracting Skeletal Muscle. *Sports Med.* 12, 208-217.
- Terjung, R. L. (1984). *Exercise and Sport Sciences Reviews*, Collamore, Lexington, Mass, p. 540
- Thurnham, D. I., Davies, J. A., Crump, B. J., Situnayake, R. D. and Davis, M. (1986). The use of Different Lipids to Express Serum Tocopherol: Lipid Ratios for the Measurement of Vitamin E Status. *Ann. Clin. Biochem.* 23 (Pt 5), 514-520.
- Tremblay, M. S., Copeland, J. L. and Van Helder, W. (2005). Influence of Exercise Duration on Post-Exercise Steroid Hormone Responses in Trained Males. *Eur. J. Appl. Physiol.* 94, 505-513.
- Vasankari, T., Kujala, U., Heinonen, O., Kapanen, J. and Ahotupa, M. (1995). Measurement of Serum Lipid Peroxidation during Exercise using Three Different Methods: Diene Conjugation, Thiobarbituric Acid Reactive Material and Fluorescent Chromolipids. *Clin. Chim. Acta* 234, 63-69.
- Vasankari, T., Kujala, U., Sarna, S. and Ahotupa, M. (1998). Effects of Ascorbic Acid and Carbohydrate Ingestion on Exercise Induced Oxidative Stress. *J. Sports Med. Phys. Fitness* 38, 281-285.
- Vasankari, T. J., Kujala, U. M., Heinonen, O. J. and Huhtaniemi, I. T. (1993a). Effects of Endurance Training on Hormonal Responses to Prolonged Physical Exercise in Males. *Acta Endocrinol. (Copenh)* 129, 109-113.
- Vasankari, T. J., Kujala, U. M., Taimela, S. and Huhtaniemi, I. T. (1993b). Pituitary-Gonadal Response to Gonadotropin-Releasing Hormone

- Stimulation is Enhanced in Men After Strenuous Physical Exercise. *Acta Endocrinol. (Copenh)* 129, 9-14.
- Vasankari, T. J., Kujala, U. M., Vasankari, T. M. and Ahotupa, M. (1998). Reduced Oxidized LDL Levels After a 10-Month Exercise Program. *Med. Sci. Sports Exerc.* 30, 1496-1501.
- Vasankari, T. J., Kujala, U. M., Vasankari, T. M., Vuorimaa, T. and Ahotupa, M. (1997). Increased Serum and Low-Density-Lipoprotein Antioxidant Potential After Antioxidant Supplementation in Endurance Athletes. *Am. J. Clin. Nutr.* 65, 1052-1056.
- Vasankari, T. J., Kujala, U. M., Viljanen, T. T. and Huhtaniemi, I. T. (1991). Carbohydrate Ingestion during Prolonged Running Exercise Results in an Increase of Serum Cortisol and Decrease of Gonadotrophins. *Acta Physiol. Scand.* 141, 373-377.
- Vigreux, B., Cnockaert, J. C. and Pertuzon, E. (1980). Effects of Fatigue on the Series Elastic Component of Human Muscle. *Eur. J. Appl. Physiol. Occup. Physiol.* 45, 11-17.
- Viinikka, L., Vuori, J. and Ylikorkala, O. (1984). Lipid Peroxides, Prostacyclin, and Thromboxane A₂ in Runners during Acute Exercise. *Med. Sci. Sports Exerc.* 16, 275-277.
- Viitasalo, J. T. and Bosco, C. (1982). Electromechanical Behaviour of Human Muscles in Vertical Jumps. *Eur. J. Appl. Physiol. Occup. Physiol.* 48, 253-261.
- Viitasalo, J. T. and Komi, P. V. (1981). Interrelationships between Electromyographic, Mechanical, Muscle Structure and Reflex Time Measurements in Man. *Acta Physiol. Scand.* 111, 97-103.
- Viitasalo, J. T., Komi, P. V., Jacobs, I. and Karlsson, J. (1982). Effects of Prolonged Cross-Country Skiing on Neuromuscular Performance. In *Exercise and Sport Biology. International Series on Sport Sciences, Vol. 12* (ed. P. V. Komi), pp. 191-198. Champaign, IL: Human Kinetics.
- Viru, A., Karelson, K. and Smirnova, T. (1992). Stability and Variability in Hormonal Responses to Prolonged Exercise. *Int. J. Sports Med.* 13, 230-235.
- Vuorimaa, T., Vasankari, T., Mattila, K., Heinonen, O., Hakkinen, K. and Rusko, H. (1999). Serum Hormone and Myocellular Protein Recovery After Intermittent Runs at the Velocity Associated with VO₂(max). *Eur. J. Appl. Physiol. Occup. Physiol.* 80, 575-581.
- Weston, A. R., Karamizrak, O., Smith, A., Noakes, T. D. and Myburgh, K. H. (1999). African Runners Exhibit Greater Fatigue Resistance, Lower Lactate Accumulation, and Higher Oxidative Enzyme Activity. *J. Appl. Physiol.* 86, 915-923.
- Weston, A. R., Mbambo, Z. and Myburgh, K. H. (2000). Running Economy of African and Caucasian Distance Runners. *Med. Sci. Sports Exerc.* 32, 1130-1134.
- Wieland, H. and Seidel, D. (1983). A Simple Specific Method for Precipitation of Low Density Lipoproteins. *J. Lipid Res.* 24, 904-909.

- Williams, J. R., Snow, R. and Agruss, C. (1991). Changes in Distance Running Kinematics with Fatigue. *Int. J. Sport. Biom.* 7, 138-162.
- Williams, K. R. (1985). Biomechanics of Running. *Exerc. Sport Sci. Rev.* 13, 389-441.
- Williams, K. R. and Cavanagh, P. R. (1987). Relationship between Distance Running Mechanics, Running Economy, and Performance. *J. Appl. Physiol.* 63, 1236-1245.
- Witztum, J. L. (1994). The Oxidation Hypothesis of Atherosclerosis. *Lancet* 344, 793-795.
- Yoshida, T., Udo, M., Iwai, K. and Yamaguchi, T. (1993). Physiological Characteristics Related to Endurance Running Performance in Female Distance Runners. *J. Sports Sci.* 11, 57-62.