Chinese Journal of Physiology 54(1): 55-66, 2011
DOI: 10.4077/CJP.2011.AMM086

Effect of Passive Repetitive Isokinetic Training on Cytokines and Hormonal Changes

Shu-Lin Lee[1], Kenny Wen-Chyuan Chen[2], Szu-Tah Chen[3], Po-Ju Chu[4], Chuan-Show Chen[5], Mei-Chich Hsu[6], Tzyy-Yuang Shiang[7], Mao-Kuan Su[8], Mu-San Chang[9], Ying-Ling Chang[10,*], and Shyi-Wu Wang[11,*]

[1]*Department of Nutrition and Health Science, Chung Chou Institute of Technology, Yuanlin, Changhua*
[2]*Department of Physical Education, Chang Gung Institute of Technology, Kweisan, Taoyuan*
[3]*Division of Endocrinology and Metabolism, Department of Internal Medicine, Chang-Gung Memorial Hospital, Taoyuan*
[4]*Department of Biomedical Sciences, Chang-Gung University, Taoyuan*
[5]*Department of Physical Education, National Taiwan Sports University, Taichung*
[6]*Institute of Sports Science, National Taiwan Sport University, Kweishan, Taoyuan*
[7]*Graduate Institute of Exercise and Sport Science, National Taiwan Normal University, Taipei*
[8]*Physical Education Room, Chang-Gung University, Taoyuan*
[9]*Center of Physical Education, Tzu Chi University, Hualien*
[10]*School of Traditional Chinese Medicine, Chang-Gung University, Taoyuan*
and
[11]*Department of Physiology and Pharmacology, College of Medicine, Chang-Gung University, Taoyuan 33333, Taiwan, Republic of China*

Abstract

It is well known that muscle strength and power are important factors in exercise. Plyometrics is designed to gain muscle strength and power in a shock method. The passive repetitive isokinetic (PRI) machine is developed for plyometrics. The present study aims to understand the effect of ten-week PRI training in different intensities on human plasma concentration cytokines as well as hormonal changes. Thirty young male subjects were enrolled into the ten-week PRI training program and were divided randomly into traditional, low- and high-intensity PRI training groups. Blood samples were obtained before, during, after and 1-, 2- , 3-, 5- and 7-day (D) post-training. The plasma concentrations of cytokines and hormones were measured by an enzyme-linked immunosorbent assay (ELISA). Elevated plasma IL-2 was found in the subjects in all the training programs. Significant increases of proinflammatory cytokines IL-1β and TNF-α were observed at post 7 D in the high-intensity PRI training (29.5 ± 4.4 and 515.8 ± 127.1 pg/ml, respectively). No significance in differences in the plasma concentration of IL-6 was observed in the traditional and low-intensity PRI training. Significant elevation of IL-6 was found at post 5 D in high-intensity PRI training. Higher plasma IL-6 concentration was observed at post 3 and 5 D in high-intensity PRI training compared to low-intensity PRI training ($P < 0.05$). Significant elevation of plasma IL-15 during (week 6) and after (post 0 D) was observed in low-intensity PRI training. Also, there were differences between low-intensity PRI training and traditional training at post 0, 2, 3, and 5 D. The plasma concentration of cortisol was decreased to the lowest value (118.0 ± 17.3 ng/ml) at post 0 D in traditional training, then returned to the baseline (220.5 ± 19.1 ng/ml). In the high-intensity PRI training, but not in the low-intensity PRI training, the cortisol level dropped from 224.9 ± 25.8 ng/ml at post 0 D down to the 123.2 ± 22.6 ng/ml at post 1 D. Significant differences

Corresponding author: Shyi-Wu Wang, Ph.D., Department of Physiology and Pharmacology, Chang Gung University, Taoyuan 33333, Taiwan, Republic of China. Tel: +886-3-211-8800 ext. 5253, Fax: +886-3-3288448, E-mail: swwang@mail.cgu.edu.tw
*These authors contributed equally to this work.
Received: August 30, 2010; Revised: November 12, 2010; Accepted: November 16, 2010.
 ISSN : 0304-4920. http://www.cps.org.tw

were found at post 1 and 5 D between low- and high-intensity PRI training, and post 0, 1, 2, and 3 D between traditional and high-intensity PRI training. Significant increased testosterone was found post 0, 1, 2, and 3 D in traditional training. Higher plasma testosterone was observed during and the recovery period in low-intensity, but not in high-intensity, PRI training. In conclusion, high-intensity PRI training could induce the proinflammatory cytokines, *i.e.* IL-1β and TNF-α, and decrease plasma cortisol in the recovery period.

Key Words: passive repetitive isokinetic training, PRI, cytokine, hormone, human

Introduction

For athletes, muscle strength and power are considered as very important components in sports competition. Large muscle strength can produce enough joint torque to accomplish the movement requiring heavy load, and also provide better base foundation for muscle power. In addition to strength, muscle power is essential to most sporting activities involving striking, throwing, jumping and rapid acceleration movements.

There are two contractions for muscle movement, *i.e*. isometric and isotonic contraction, or eccentric and concentric muscle contractions. In most cases, muscle contractions, stretch and shortening cycle (SSC) are involved in the real movement (36). Therefore, it is well known that successful athletic performance depends on leg-muscle power and vertical-jump performance (4, 69). During the past years, vibration training (8), electrostimulation training (46), heavy-resistance training (92) and explosive type (93) have been widely and effectively applied for the improvement of vertical jump performance. However, studies have indicated that plyometric training (PT) seems to be a better method for enhancement of vertical jump ability and leg muscle power (16, 48). Plyometric training, advocated by Verkhoshanski in Russia in 1969 (90), is the SSC movements involving a rapid, powerful concentric contraction and high-intensity eccentric contraction (47). Passive repetitive isokinetic (PRI) training methods were developed based on the theory of high speed contraction, stretch shortening cycle and isokinetic contraction. The PRI training machine has also been designed to achieve the above mentioned functions. A previous study has shown that in augmentation of muscle power and strength, the effect of PRI training on PRI machine is significantly better than the traditional isotonic weight training (43). Also, long term PRI training (10 weeks) could affect the reflex modulation of spinal cord and plasticity of spinal circuitry (42).

It is well known that the immune function can be influenced by exercise, including strenuous and eccentric exercises. Cytokines are a group of small peptides released from white blood and other body cells in response to different stimuli. Cytokines are considered as immunoregulatory molecules for regulation of immune function and other body responses (83). Physical training and exercise are considered as stressors which induce local or systemic responses including cytokine changes (5). Many reviews have indicated that heavy exertion such as ultramarathon races not only increased the risk of upper respiratory tract infection (URTI) but also the augmented plasma interleukin (IL)-6 levels. In addition, the proinflammatory cytokines, TNF-α and IL-1β, have been reported to be increased during and after exercises. Moreover, mRNAs of IL-6 and IL-15 have been demonstrated to be expressed in muscle tissues, and these three cytokines are classified as "myokines" (61). However, most studies concerning the exercise effects on the plasma levels of cytokines are limited to the short period during and after training or race. Changes of plasma levels of cytokines during and after PRI training are still unknown.

The present investigation was to explore the cytokines change pre-, during and after PRI training. We also investigated the cytokines levels in the recovery period up to 7 days. In addition, we measured the plasma testosterone and cortisol levels which represent the anabolic and catabolic hormones.

Materials and Methods

Subjects

Thirty-two healthy male college students with a mean age of 22 ± 1 years old, height 176 ± 2 cm, weight 70 ± 2 kg, were enrolled in this experiment. All subjects were asked to maintain their usual food intake and no food supplements and medication were taken. The experimental protocol was approved by the Institutional Review Board/Chang-Gung Memorial Hospital, and all subjects gave written informed consent. Two subjects were excluded because of cold disorder. Ten-week training was applied to all subjects who were randomly divided into three groups: traditional weight training control group (n = 10), low-intensity (0.5 Hz) PRI training group (n = 10), and

Table 1. Detection range, sensitivity, intraassay and interassay coefficients of variation (CV) for the cytokines and hormones ELISA

Cytokines/ Hormones	Detection range	Sensitivity	Intraassay CV	Interassay CV
IL-1β	3.91-250 pg/ml	1 pg	4.0%	6.9%
IL-2	31.25-2,000 pg/ml	7 pg	6.4%	10.2%
IL-6	9.375-600 pg/ml	0.7 pg	4.2%	10.8%
IL-15	7.8-500 pg/ml	1 pg	7.8%	11.7%
TNF-α	15.6-1,000 pg/ml	1.6 pg	5.0%	9.6%
Cortisol	50-230 ng/ml	2.5 ng	4.4%	7.1%
Testosterone	7.8-500 pg/ml	6 pg	4.4%	7.7%

high-intensity (2.5 Hz) PRI training group (n = 10).

Experimental Design

All subjects underwent a 10-week regiment of training 3 days/week and maintained their habitual physical activities but performed no additional strength training. Exercise was performed at a resistance that was initially 70% of one repetition maximum (1 RM) on the PRI machine or the traditional squat training method (Smith Press, Cybex, NY, USA). The subjects of low intensity group performed training for 20 sec at a frequency of 0.5 Hz, while high intensity group performed training for 20 sec at a frequency of 2.5 Hz. Resistance used in this study was determined during the training sessions every week for the 10-week training period using the 1 RM approach. Subjects performed 10 repetitions set and performed 5 set with 2-min rest periods.

Blood Collection

Plasma was separated by centrifugation at 10,000 × *g* for 1 min and stored at -20°C. To measure the concentrations of cytokines and hormones, including interleukin-1β (IL-1β), interleukin-2 (IL-2), interleukin-6 (IL-6), interleukin-15 (IL-15), tumor necrosis factor-α (TNF-α) and testosterone and cortisol, 0.1 ml plasma was used. The concentrations of plasma cytokines and hormones were measured by enzyme-linked immunosorbent assays (ELISA).

ELISA of Cytokines and Hormones

Plasma concentrations of cytokines and hormones were determined by ELISA (Enzyme-linked Immunosorbent Assay) as described previously (10, 25, 94, 95) with some modifications. Briefly, 0.1 ml of capture antibodies (R & D systems, Minneapolis, MN, USA) were coated on the polystyrene microtiter plates (NUNC, U16 Maxisorp type, Rochester, NY, USA) and incubated at room temperature overnight. The plates were blocked the next day and then incubated for 1 h. Then, 0.1 ml standard/sample was added and incubated for 2 h. After washing 3 times, 0.1 ml of detection antibody (R & D systems) was applied for 2 h. One hundred micorliters streptavidin horseradish peroxidase (R & D systems) and then 0.1 ml tetramethylbenzidine substrate (Clinical Science Products Inc., Mansfield, MA, USA) followed this incubation. The reaction was stopped using 2 N sulfuric acid and optical density (OD) reading was taken at 450 nm (BioTek, Winooski, VT, USA). All samples were run in duplicates. The results were expressed as concentration of cytokines (pg/ml) and hormones (ng/ml) read from standard curves. The detection range, sensitivity, the intraassay and interassay coefficients of variation for the ELISA are presented in Table 1.

Statistical Analysis

All values are given as the means ± SEM. Differences between the pre, during and post exercise values of the measured variables were tested for homogeneity by one-way ANOVA and the differences between specific means were tested for significance by the Duncan multiple range test (79). A difference between two means was considered statistically significant when $P < 0.05$.

Results

Changes of Plasma Levels of IL-2 before and after PRI Training

Traditional training significantly enhanced plasma concentrations of IL-2 during the recovery period (57.0 ± 10.8 pg/ml at post 0 D to 50.5 ± 11.3 pg/ml at post 7 D), reaching the highest level at post 0 D compared to pre-training (29.7 ± 5.8 pg/ml) (Fig. 1, upper panel). There was a significant gradual increase in plasma IL-2 level during the recovery period (21.5 ± 2.0 pg/ml at post 0 D to 35.1 ± 2.7 pg/ml at post

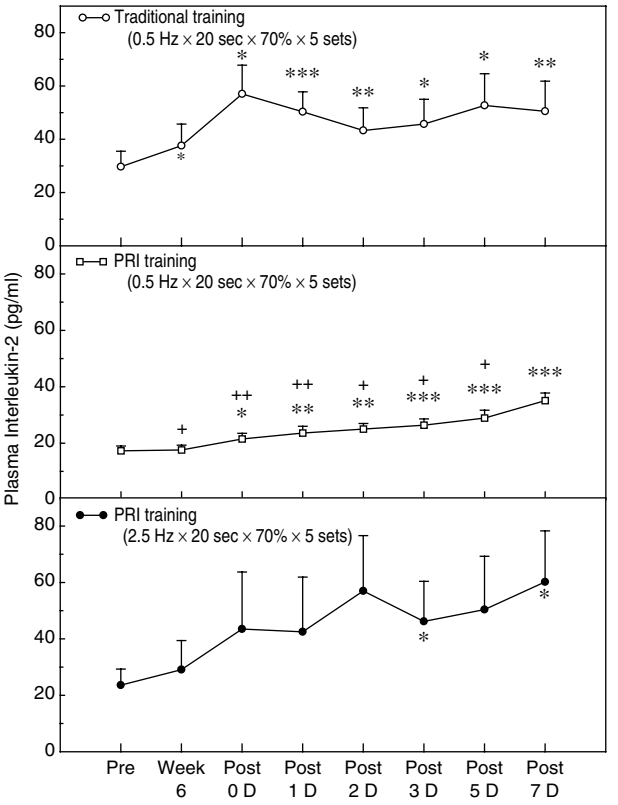

Fig. 1. Plasma concentrations of interleukin-2 (IL-2) before and after PRI training. Before and after PRI training, blood samples were obtained from subjects and separated by centrifugation. Collected plasma was stored in -20°C and the plasma IL-2 concentration was measured by ELISA. N = 10. *, **, *** $P < 0.05$, $P < 0.01$ and $P < 0.001$ compared to the values before training (designated as pre). +, ++ $P < 0.05$ and $P < 0.01$, values in PRI (low- or high-intensity) training, respectively, compared to the value in traditional training.

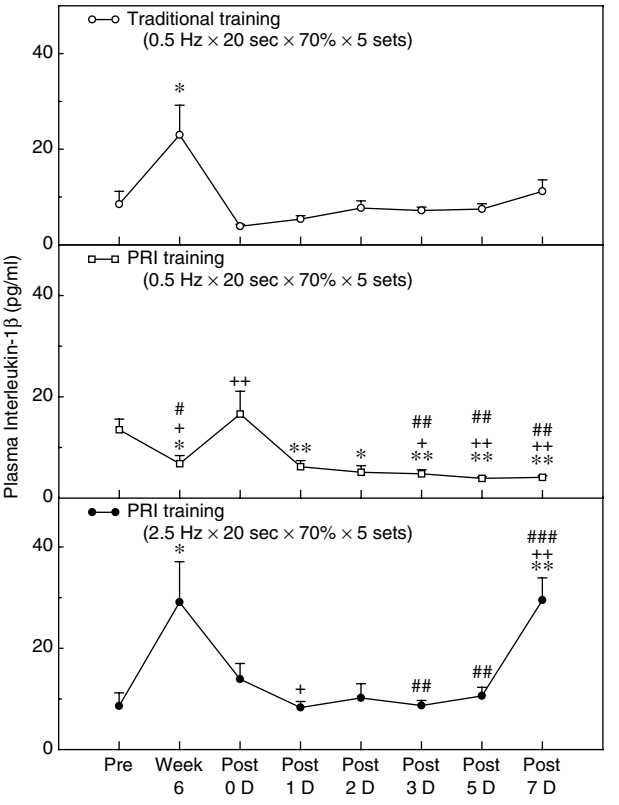

Fig. 2. Plasma concentrations of interleukin-1β before and after PRI training. See legend to Fig. 1. #, ##, ### $P < 0.05$, $P < 0.01$, and $P < 0.001$ values in low-intensity PRI training compared to the value in high-intensity PRI training. Each value represents means ± SEM.

7 D) compared to the value of pre-training (17.3 ± 1.7 pg/ml) after low-intensity PRI training (0.5 Hz × 20 sec × 70% × 5 sets) and high-intensity PRI training (2.5 Hz × 20 sec × 70% × 5 sets) during the recovery period (43.5 ± 20.2 pg/ml at post 0 D to 60.2 ± 18.1 pg/ml at post 7 D) compared to the value of pre-training (23.6 ± 5.7 pg/ml) (Fig. 1, middle and lower panels). A lower plasma IL-2 concentration was found in the low-intensity PRI training during the recover period compared to the plasma IL-2 concentration in traditional training.

Changes of Plasma Interleukin-1β (IL-1β) Levels before and after PRI Training

Fig. 2 shows the concentration of plasma IL-1β before and after PRI training. The highest level of plasma IL-1β was observed at week 6 (23.0 ± 6.2 pg/ml) for traditional training, but not significant compared to the pre-training (8.5 ± 2.4 pg/ml) (Fig. 2, upper panel). Then, the plasma IL-1β dropped to the lowest level at post 0 D (3.9 ± 0.4 pg/ml) and remained at low levels during the recovery period (post 1 D to post 7 D). In the low-intensity PRI training, the highest concentration of plasma IL-1β was observed at post 0 D (16.6 ± 4.5 pg/ml), then declined gradually to the significantly lower levels during the recovery period compared to the value of pre-training (13.5 ± 2.1 pg/ml) (Fig. 2, middle panel). The plasma IL-1β level reached the peak level (29.1 ± 8.0 pg/ml) at week 6 in the high-intensity PRI training period, then dropped to the level similar to that of pre-training (8.6 ± 2.6 pg/ml). The highest plasma IL-1β concentration, however, was observed at post 7 D (29.5 ± 4.4 pg/ml) after the high-intensity PRI training (Fig. 2, lower panel). Significantly low plasma IL-1β level in the low-intensity PRI training was observed at week 6, post 3, 5 and 7 D compared to the level of the traditional training. In contrast, higher plasma IL-1β level in high-intensity PRI training was found at post 3, 5 and 7 D compared to the level of the low-intensity PRI training.

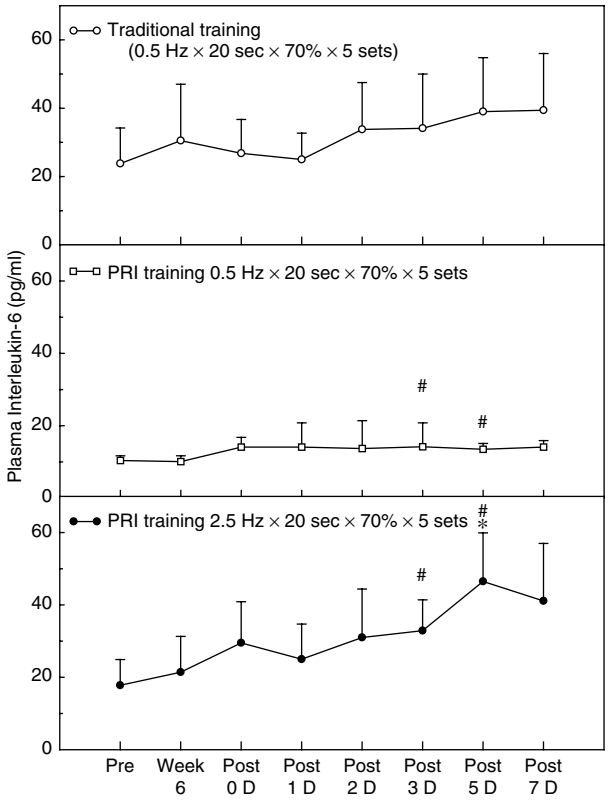

Fig. 3. Plasma concentrations of interleukin-6 before and after PRI training. See legends to Figs. 1 and 2.

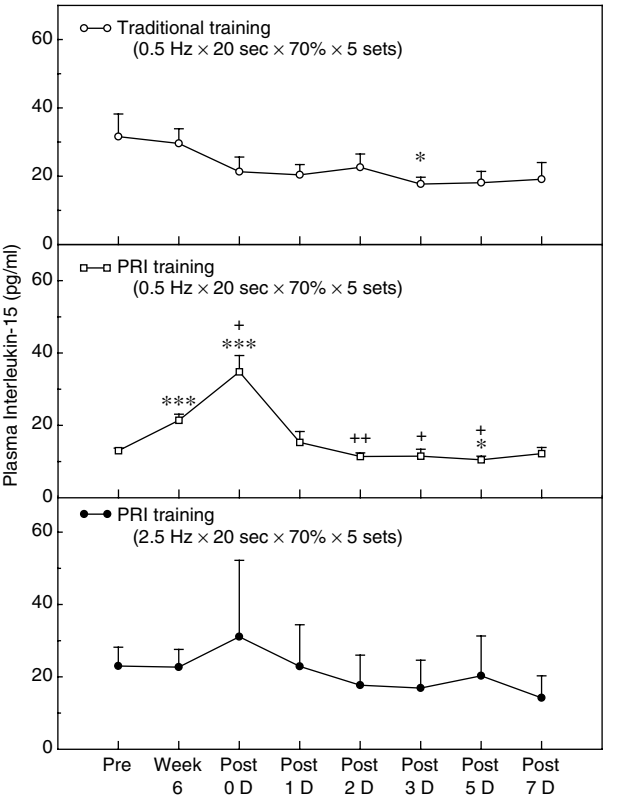

Fig. 4. Plasma concentrations of interleukin-15 before and after PRI training. See legend to Fig. 1.

Effect of PRI Training on the Concentration of IL-6 before and after Exercise

Gradually increased IL-6 level was observed after the traditional training (26.8 ± 9.9 pg/ml at post 0 D to 39.4 ± 16.6 pg/ml at post 7 D), however, the level was not significant compared to the value from pre-training (23.8 ± 10.4 pg/ml) (Fig. 3, upper panel). After the low-intensity PRI-training, the plasma IL-6 increased, but not significantly, during the recovery period (14.1 ± 2.7 pg/ml at post 0 D to 14.1 ± 1.8 pg/ml at post 7 D compared to 10.4 ± 1.3 pg/ml at pre-training) (Fig. 3, middle panel). Significant level of plasma IL-6 was found after high-intensity PRI-training (post 5 d, 46.5 ± 13.4 pg/ml, respectively) compared to the value from pre-training (17.8 ± 7.1 pg/ml) (Fig. 3, lower panel). In addition, significant difference was observed at post 3 and 5 D between low- and high-intensity PRI training. Higher plasma IL-6 level in high-intensity PRI training was found at post 3 and 5 D compared to the level of low-intensity PRI training.

Plasma Concentrations of IL-15 before and after PRI Training

After traditional training, the plasma IL-15 concentration decreased gradually to a lowest value at post 3 D (17.7 ± 2.0 pg/ml) (Fig. 4, upper panel). In contrast, gradually increased IL-15 level was observed during exercise and to a highest level at post 0 D after low-intensity PRI-training. In the recovery period, the plasma IL-15 level decreased gradually (Fig. 4, middle panel). A similar pattern was found in high-intensity PRI-training. Highest values of plasma IL-15, which was not significant compared to pre-training, was found at post 0 D (31.1 ± 21.1 *vs*. 23.0 ± 5.2 pg/ml), then declined gradually during the recovery period (Fig. 4, lower panel). There were differences between traditional training and low-intensity PRI training at post 0, 2, 3, and 5 D.

Plasma Concentrations of TNF-α before and after PRI Training

In the traditional training, the plasma TNF-α remained constant during and at recovery period until post 3 D (229.2 to 276.1 pg/ml), then declined gradually at post 5 and 7 D (218.6 ± 73.1 and 152.2 ± 62.3 pg/ml, respectively) (Fig. 5, upper panel).

There was no significant change for the plasma TNF-α concentration after the low-intensity PRI training during exercise and in the recovery period, ranging

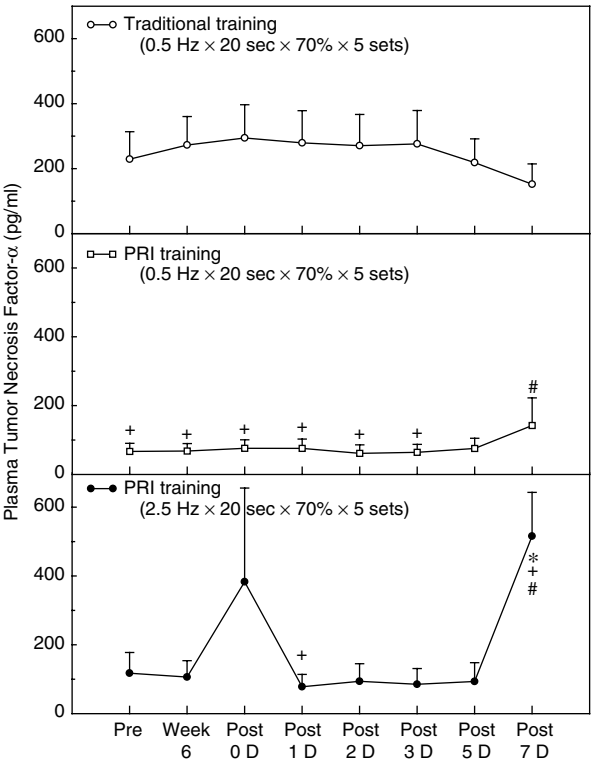

Fig. 5. Plasma concentrations of tumor necrosis factor-α before and after PRI training. See legends to Figs. 1 and 2.

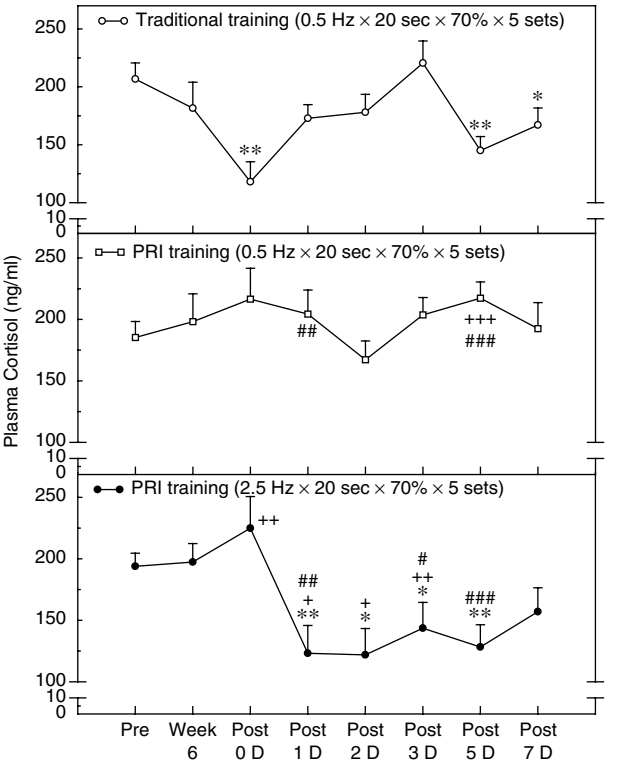

Fig. 6. Plasma concentrations of cortisol before and after PRI training. See legends to Figs. 1 and 2.

from 66.8 ± 23.7 to 142.3 ± 80.3 pg/ml (Fig. 5, middle panel). In the high-intensity PRI training, an initial plasma TNF-α concentration of 117.6 ± 60.1 pg/ml decreased to 78.1 ± 35.8 pg/ml at post 1 D, then peaked to 515.8 ± 127.1 pg/ml ($P < 0.05$ compared to pre-training) (Fig. 5, lower panel). A lower plasma TNF-α concentration was found in low-intensity PRI training compared to the level in the traditional training. However, the plasma TNF-α concentration at post 7 D in high-intensity PRI training was significant higher than the plasma TNF-α levels in traditional and low-intensity PRI training.

Plasma Concentrations of Cortisol before and after PRI Training

In response to the traditional training, plasma cortisol reduced from 206.7 ± 13.9 at pre-training to 118.0 ± 17.3 ng/ml at post 1 D ($P < 0.01$). However, plasma cortisol levels rose up to 220.5 ± 19.1 ng/ml, then declined to 145.0 ± 12.1 ($P < 0.01$ *vs*. Pre) and 167.0 ± 14.7 ng/ml ($P < 0.05$ *vs*. Pre) at post 5 and 7 D, respectively (Fig. 6, upper panel). In the low-intensity PRI training, the highest values of plasma cortisol were found at post 5 D (217.3 ± 13.2 ng/ml) and the lowest value was observed at post 2 D (167.2 ± 15.3 ng/ml). No significant difference was found compared to the pre-training (185.3 ± 13.0 ng/ml) (Fig. 6, middle panel). After the high-intensity PRI training, the plasma cortisol level rose from 193.9 ± 1.07 at pre-training to 224.9 ± 25.8 ng/ml at post 0 D ($P = 0.197$), then decreased significantly ($P < 0.01$) to 123.2 ± 22.6 ng/ml at post 1 D, increased slowly to 157.0 ± 19.5 ng/ml at post 7 D (Fig. 6, lower panel). Significant difference in the plasma cortisol concentration between traditional and low-intensity PRI training was found at post 0 and 5D. Between high-intensity PRI and traditional training, the significant difference was observed at post 0, 1, 2, and 3 D. Also, there was significant difference in the plasma cortisol concentration at post 1, 3, and 5 D between the low- and high-intensity PRI training.

Plasma Concentrations of Testosterone before and after PRI Training

In traditional training, significantly increased plasma concentrations of testosterone were found between post 0 D (10.7 ± 1.4 ng/ml) and post 3 D (6.0 ± 1.2 ng/ml) compared to pre-training (6.2 ± 0.7 ng/ml) (Fig. 7, upper panel). There was a gradual and significant increase in the plasma testosterone concentrations during and after the low-intensity PRI training (7.3 ± 1.2 and 9.8 ± 1.6 ng/ml, respectively) (Fig. 7, middle panel). After the high-intensity PRI

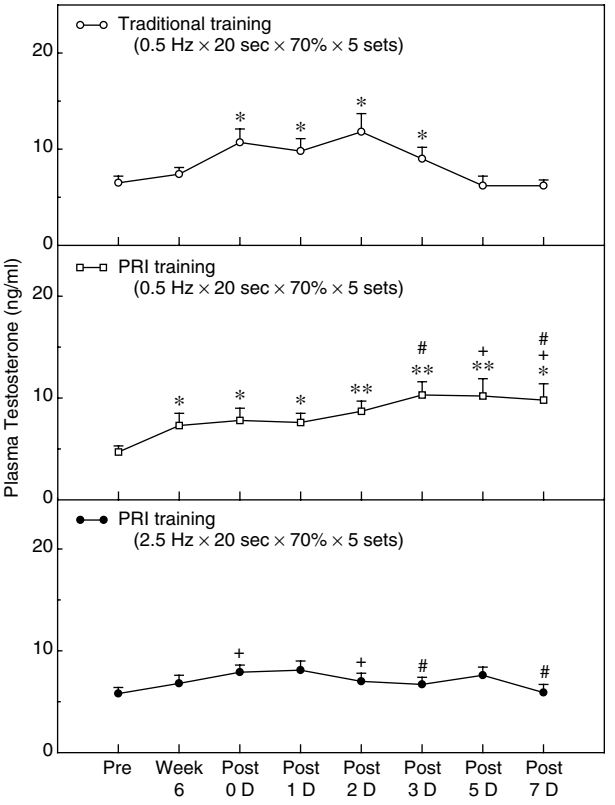

Fig. 7. Plasma concentrations of testosterone before and after PRI training. See legends to Figs. 1 and 2.

training, the plasma testosterone concentrations increased from 6.2 ± 0.7 ng/ml at pre-training to 7.9 ± 0.8 ng/ml at post 1 D, then decreased to 6.4 ± 0.8 ng/ml at post 7 D (Fig. 7, lower panel). No significant difference was found in the plasma testosterone concentration in the high-intensity PRI training. At post 5 and 7 D, there were significant differences in plasma testosterone concentrations between the traditional and low-intensity PRI training. Between the high-intensity PRI and traditional training, significant difference in plasma testosterone concentrations was observed at post 0 and 2 D. Also, there were significant differences between low- and high-intensity PRI training at post 3 and 7 D.

Discussion

The objective of this study was to determine if the plasma cytokine and hormone (testosterone and cortisol) levels would vary with different intensities of PRI training compared to traditional training with similar time and duration. The present study provides information on changes in the plasma concentrations of IL-1β, IL-2, IL-6, IL-15, TNF-α, testosterone and cortisol before, during and after PRI training. Also, the recovery period had been extended to 7 days.

IL-1β and tumor necrosis factor TNF-α are two classic proinflammatory cytokines which are mainly produced from monocytes, macrophages and neutrophils (65). In 1983, Cannon and Kluger detected an endogenous pyrogen release from human mononuclear leukocytes *in vitro* during exercise (7). Evans *et al*. found that plasma IL-1 activities, measured using the thymocyte proliferation method, increased 3 h after exercise in all untrained men, but not in trained people (18). In a later study, ELISA was used to show that there was no detectable IL-1 in plasma after exercise (58). In contrast, two- to three–fold increase of plasma IL-1β concentrations was found after exercise (59, 86).

Increased plasma TNF-α levels were observed at the end of exercise and remained elevated at 24 h post-exercise (51, 62, 80, 86). In contrast, undectable, unchanged or decreased plasma TNF-α levels were found during and after exercise (25, 30, 84, 85). In addition, the TNF-α mRNA has been detected in muscle tissues (2, 76) and white blood cells (WBC). In frail obese elderly persons, significant decrease in TNF-α mRNA levels from the vastus lateralis muscle was observed after exercise (41). However, other studies found increased TNF-α mRNA levels in obesity (21), elderly (26) and in patients with type II diabetes mellitus (73). The TNF-α mRNA in WBC or monocytes after exercise training may be unchanged (51), decreased (74) or increased (71, 96). These divergent results on plasma IL-1β and TNF-α in relation to exercise could be due to the type, mode, intensity and duration of physical activity. Also, the sensitivity and specificity of assays might be another important factor (5). Our study showed that there were no differences in plasma levels of TNF-α and IL-1β compared to the pre-values in traditional training. Decreased levels of plasma IL-1β, but not TNF-α, were found in low intensity PRI training. Significantly increased levels of plasma IL-1β and TNF-α levels were found in high intensity PRI training at post 7 D. It seems that the intensity of exercise could enhance the plasma IL-1β and TNF-α levels after exercise.

IL-2 is a cytokine produced primarily from activated T-lymphocytes. This cytokine plays important roles in immunoregulatory responses after stimulation, and enhances the function of T-cells for host defense (13). Results from previous studies indicated that the plasma concentration of IL-2 elevated, decreased, or unchanged after exercise. Suzuki *et al*. showed that the plasma IL-2 concentration was significantly reduced by 32% after the Marathon race (82). Espersen *et al*. found that, compared to the pre-exercise values, IL-2 was significantly decreased immediately after a five-kilometer race and significantly increased after 24 h (17). In contrast, Neiman

et al. showed unchanged plasma IL-2 concentrations after exercise compared to pre-exercise values (56). Also, there are many factors that affect the immune system during and after exercise. Early studies have documented different findings from immunosuppression to immunostimulation. Therefore, the IL-2 mRNA expression was detected in the PHA-stimulated $CD4^+/CD8^+$ lymphocytes or peripheral blood mononuclear (PBMN) cells collected after exercise. Bacurau *et al*. reported that exercise induced a reduction by 35% in the production of IL-2 by cultured PBMN (1). In addition, the percentage of IL-2-producing $CD4^+$ and $CD8^+$ T-cells was suppressed at the end of exercise and 2 h after exercise, but returned to pre-exercise values 24 h after exercise (77). In contrast, some studies indicated that the IL-2 expressing $CD4^+$ and $CD8^+$ T cells did not change, or increased, after exercise compared to pre-exercise values (32, 96). In our study, although we did not examine the expression of IL-2 in $CD4^+$ and $CD8^+$ T-cells, exercise induced elevation of plasma IL-2 concentration in traditional PRI training was nearly two times at post 0 D compared to the pre-exercise values. The same result was observed in light PRI-training. However, in heavy PRI-training, the highest plasma IL-2 concentration was found at post 7 D, 2.6 times compared to the pre-exercise values.

Of the measured cytokines in response to an exercise-induced stimulation, IL-6 increase appeared to be the reliable and significant marker (82). The increased magnitude of IL-6 during and after exercise depends on the exercise intensity (60). IL-6 is considered to be the first cytokine changed in the plasma during exercise. The plasma IL-6 level increases from 10- to 100-fold in response to exercise and declines after exercise and recovery period (19, 64, 66, 81). In addition, muscle damage is considered to be the major source of increased plasma IL-6 during exercise. Many studies have demonstrated that elevated IL-6 mRNA is expressed in contracted muscle (20, 34, 54, 59, 75, 76) and that IL-6 is secreted from the skeletal muscle during and after exercise (31, 68). Even a moderate exercise can induce a 20-fold elevation of plasma IL-6 and a 16-fold increase of IL-6 mRNA in working muscles (22). Although numerous studies have suggested that IL-6 is produced from damaged muscle tissue during exercise (20, 56), other studies demonstrated IL-6 could be elevated by undamaged muscle (49) and other factor unrelated to exercise such as neutrophils under lipopolysaccharide (LPS) stimulation (50). In the present study, although we did not determine the IL-6 mRNA expression level in the skeletal muscle, the plasma IL-6 only increased 1.4-fold in low intensity PRI-training and 2.6-fold in high-intensity PRI-training. This result indicates that the elevation of plasma IL-6 in low- and high-intensity PRI-training would not be so large compared to other studies.

IL-15 is a 14-kDa cytokine identified as a T-cell growth factor (70). Few studies are concerned with plasma IL-15 concentrations during and after exercise. Significantly increased plasma IL-15 protein levels were observed immediately after acute resistance exercise in young participants, including men and women (72). Another study showed unchanged plasma IL-15 protein during and after exercise in male people (52). In contrast, a recent study showed decreased circulating IL-15 levels in obese people with low energy diet and diet-induced weight-loss groups after aerobic exercise for 12 weeks (11). Also, evidences showed that increased expression of IL-15 mRNA was detected in the skeletal muscle (15, 52). Therefore, IL-15 is considered as a myokine; cytokines and other peptides that are produced, expressed and secreted from muscle fibres and exert paracrine or endocrine effects (61-63, 67). IL-15 exerts its effect on muscle protein dynamics, mainly on the decrease of muscle protein degradation induced by proteolysis factors, lipid oxidation stimulation and increase of glucose uptake (70). In our study, we found significantly increase of plasma IL-15 concentration at post 0 D in light-intensive PRI training, but not in traditional or in heavy-intensive PRI training. It is unknown whether these results indicate significant depression in muscle protein degradation, increased lipid oxidation, or glucose uptake. Further studies are needed to clarify this question.

Accumulated evidences show that the intensity and/or duration of exercise may be a form of stress which mediates cortisol secretion through the CRH and ACTH (33). Many studies have indicated that the plasma and salivary cortisol concentrations increase after prolonged exercise at high intensity (3, 24, 45, 53, 55, 87, 88). Therefore, short-duration and low-intensity physical training would not increase the plasma concentrations of cortisol (24). In addition, no change of serum cortisol concentrations was observed in a pilot study with 50 min of high intensity exercise (9). The augmentation of plasma cortisol after prolonged exercise with high intensity has been in relation to lipolysis, ketogenesis, proteolysis, and glucose homeostasis (12). Moreover, cortisol has been hypothesized as an important factor in maintaining lymphopenia and neutrophilia after high-intensity and prolonged exercise (64). Kanaley *et al*. reported that both baseline (pre-exercise) and peak cortisol concentrations were significantly higher at 7 AM that at 7 PM or midnight (35). In contrast, decreased cortisol concentrations overnight were observed after 90 min of exercise at 70% Vo_{2max} (27). Although we did not collect blood samples at 7 AM, the schedule for the sample collection were the same.

In our study, increased plasma cortisol levels were, although not significantly, found after low and high intensity PRI training at post 0 D. However, significantly decreased cortisol level was observed from post 1 D to post 7 D after high intensity PRI training, not in low intensity PRI training. In contrast, in traditional training, the lowest cortisol was found at post 0 D. It is not known what factor(s) could affect the plasma cortisol levels in traditional training and PRI training; the training type, duration and intensity are considered possible factors to influence plasma hormone and cytokines levels (64).

As a potent anabolic hormone which stimulates muscle protein synthesis (40), testosterone is mainly synthesized and secreted from testicular Leydig cells in men and ovaries and adrenal cortex in women (44). Studies have indicated that exercise results in acute increases of plasma testosterone concentrations during and 5 minutes after training (23, 29, 37-39, 89). Then the elevated testosterone level returned to the basal level. In contrast, decreased plasma testosterone during exercise was found in other studies (14, 28). These studies indicate that the elevated or decreased plasma testosterone levels seem to be depended on the training mode, duration of exercise bout and intensity of training. The physiological basis for these data (*i.e.* elevated or decreased testosterone levels) could be due to reduced metabolic clearance rate (6) and hemoconcentration (91) for increased testosterone concentration and blunted luteinizing hormone (LH) production rate (57) or reduced responsiveness of Leydig cells to LH for decreased testosterone concentration. Moreover, dietary nutrients employed by the trained people have been shown to affect hormonal changes during and after exercise (89). Low percentage of dietary fat (93) or high protein-to-carbohydrate ratio dietary (89) is associated with low plasma testosterone concentration in healthy active men. In this study, elevated plasma testosterone concentration was observed post-1 to 3 D in the three training modes. Although we did not measure the plasma LH, these increased testosterone levels might be due to the increased LH production from pituitary gland or elevated responsiveness of Leydig cells to LH. However, the exact physiological mechanisms responsible for the increased testosterone post-exercise in our study remain to be fully elucidated.

The relationship between cytokines (*e.g.* interleukins, interferons, and growth factors) and hormones (including steroid and protein hormones) has been well recognized (78). Exercise or training mainly enhances muscle strength and power. Numerous reviews have indicated that exercise could alter the immune and endocrine status during and after training period (44). In the past decade, muscle has been speculated and demonstrated to be the main source of IL-6 synthesis (63). Also, hormones, including growth hormone and insulin-like growth factors, have been associated with muscle strength and power during training period (78). Although we measured the plasma testosterone and cortisol concentrations before, during and after training, we did not find a relationship between cortisol and testosterone with interleukins.

In summary, the present study provides information on changes in the plasma concentrations of IL-1β, IL-2, IL-6, IL-15, TNF-α, testosterone and cortisol during different intensities of PRI training compared to traditional training with similar duration. Also, the recovery period had been extended to 7 days. Our result indicated that high-intensity PRI training could induce the proinflammatory cytokines, IL-1β and TNF-α, and decrease plasma cortisol levels in the recovery period.

Acknowledgments

This study was supported by the Grant NSC92-2413-H-182-004, NSC93-2413-H-182-003, NSC94-2413-H-179-010, and NSC96-2413-H-182-003 from the National Science Council. The technical assistance provided by Miss I-Hsiao Kao and Sama Chen is appreciated.

References

1. Bacurau, R.F.P., Bassit, R.A., Sawada, L., Navarro, F., Martins Jr, E. and Costa Rosa, L.F.B.P. Carbohydrate supplementation during intense exercise and the immune response of cyclists. *Clin. Nutr.* 21: 423-429, 2002.
2. Batista Jr, M.L., Rosa, J.C., Lopes, R.D., Lira, F.S., Martins Jr, E., Yamashita, A.S., Brum, P.C., Lancha Jr, A.H., Lopes, A.C. and Seelaender, M. Exercise training changes IL-10/TNF-α ratio in the skeletal muscle of post-MI rats. *Cytokine* 49: 102-108, 2010.
3. Berk, L.S., Nieman, D.C., Youngberg, W.S., Arabatzis, K., Simpson-Westerberg, M., Lee, J.W., Tan, S.A. and Eby, W.C. The effect of long endurance running on natural killer cells in marathoners. *Med. Sci. Sports Exerc.* 22: 207-212, 1990.
4. Bobbert, M.F. Drop jumping as a training method for jumping ability. *Sports Med.* 9: 7-22, 1990.
5. Bruunsgaard, H. Physical activity and modulation of systemic low-level inflammation. *J. Leukoc. Biol.* 78: 819-835, 2005.
6. Cadoux-Hudson, T.A., Few, J.D. and Imms, F.J. The effect of exercise on the production and clearance of testosterone in well trained young men. *Eur. J. Appl. Physiol. Occup. Physiol.* 54: 321-325, 1985.
7. Cannon, J.G. and Kluger, M.J. Endogenous pyrogen activity in human plasma after exercise. *Science* 220: 617-619, 1983.
8. Cardinale, M. and Bosco, C. The use of vibration as an exercise intervention. *Exerc. Sport Sci. Rev.* 31: 3-7, 2003.
9. Chatzitheodorou, D., Kabitsis, C., Malliou, P. and Mougios, V. A pilot study of the effects of high-intensity aerobic exercise versus passive interventions on pain, disability, psychological strain, and serum cortisol concentrations in people with chronic low back pain. *Phys. Ther.* 87: 304-312, 2007.
10. Cheng, H.M., Hsiang, C.Y., Chen, G.W., Wu, S.L., Chen, J.C., Huang, C.Y., Bau, D.T. and Ho, T.Y. Inhibition of lipopolysaccharide-induced interleukin-β and tumor necorsis factor-α production by menthone through nuclear factor-κB signaling pathway in HaCat

cells. *Chinese J. Physiol*. 51: 160-166, 2008.
11. Christiansen, T., Paulsen, S.K., Bruun, J.M., Pedersen, S.B. and Richelsen, B. Exercise training versus diet-induced weight-loss on metabolic risk factors and inflammatory markers in obese subjects: a 12-week randomized intervention study. *Am. J. Physiol. Endocrinol. Metab*. 298: E824-E831, 2010.
12. Corral, P.D., Howley, E.T., Hartsell, M., Ashraf, M. and Younger, M.S. Metabolic effects of low cortisol during exercise in humans. *J. Appl. Physiol*. 84: 939-947, 1998.
13. De Maeyer, E. and De Maeyer-Guignard, J. The Cytokine Handbook. San Diego, CA: Academic, 1998, pp. 491-516.
14. Dessypris, A., Kuoppasalmi, K. and Adlercreutz, H. Plasma cortisol, testosterone, androstenedione and luteinizing hormone (LH) in a non-competitive marathon run. *J. Steroid Biochem*. 7: 33-37, 1976.
15. Dieli-Conwright, C.M., Spektor, T.M., Rice, J.C. and Schroeder, E.T. Hormone therapy attenuates exercise-induced skeletal muscle damage in postmenopausal women. *J. Appl. Physiol*. 107: 853-858, 2009.
16. Ebben, W.P. and Blackard, D.O. Strength and conditioning practices of National Football League strength and conditioning coaches. *J. Strength Cond. Res*. 15: 48-58, 2001.
17. Espersen, G.T., Elbaek, A., Ernst, E., Toft, E., Kaalund, S., Jersild, C. and Grunnet, N. Effect of physical exercise on cytokines and lymphocyte subpopulations in human peripheral blood. *APMIS* 98: 395-400, 1990.
18. Evans, W.J., Meredith, C.N., Cannon, J.G., Dinarello, C.A., Frontera, W.R., Hughes, V.A., Jones, B.H. and Knuttgen, H.G. Metabolic changes following eccentric exercise in trained and untrained men. *J. Appl. Physiol*., 61: 1864-1868, 1986.
19. Febbraio, M.A. and Pedersen, B.K. Muscle-derived interleukin-6: mechanisms for activation and possible biological roles. *FASEB J*. 16: 1335-1347, 2002.
20. Febbraio, M.A., Steensberg, A., Keller, C., Starkie, R.I., Krustrup, P., Ott, P., Secher, N.H. and Pedersen, B.K. Glucose ingestion attenuates interleukin-6 release from contracting skeletal muscle in humans. *J. Physiol*. 549: 607-612, 2003.
21. Ferrier, K.E., Nestel, P., Taylor, A., Drew, B.G. and Kingwell, B.A. Diet but not aerobic exercise training reduces skeletal muscle TNF-α in overweight humans. *Diabetologia* 47: 630-637, 2004.
22. Fischer, C.P., Hiscock, N.J., Penkowa, M., Basu, S., Vessby, B., Kallner, A., Sjoberg, L.B. and Pedersen, B.K. Supplementation with vitamins C and E inhibits the release of interleukin-6 from contracting human skeletal muscle. *J. Physiol*. 558: 633-645, 2004.
23. Fry, A.C., Kraemer, W.J. and Ramsey, L.T. Pituitary-adrenal-gonadal responses to high-intensity resistance exercise overtraining. *J. Appl. Physiol*. 85: 2352-2359, 1998.
24. Galbo, H. Hormoneal and metabolic adaptation to exercise. New York: Thieme Verlag, 1983.
25. Gokhale, R., Chandrashekara, S. and Vasanthakumar, K.C. Cytokine response to strenuous exercise in athletes and non-athletes-an adaptive response. *Cytokine* 40: 123-127, 2007.
26. Greiwe, J.S., Cheng, B., Rubin, D.C., Yarasheski, K.E. and Semenkovich, C.F. Resistance exercise decreases skeletal muscle tumor necrosis factor-α in frail elderly humans. *FASEB J*. 15: 475-482, 2001.
27. Hackney, A.C. The male reproductive system and endurance exercise. *Med. Sci. Sports Exerc*. 28: 180-189, 1996.
28. Hackney, A.C., Fahrner, C.L. and Gulledge, T.P. Basal reproductive hormonal profiles are altered in endurance trained men. *J. Sports Med. Phys. Fitness* 38: 138-141, 1998.
29. Hakkinen, K., Pakarinen, A., Kraemer, W.J., Newton, R.U. and Alen, M. Basal concentrations and acute responses of serum hormones and strength development during heavy resistance training in middle-aged and elderly men and women. *J. Gerontol. A Biol. Sci. Med. Sci*. 55: B95-B105, 2000.
30. Hirose, L., Nosaka, K., Newton, M., Laveder, A., Kano, M., Peake, J. and Suzuki, K. Changes in inflammatory mediators following eccentric exercise of the elbow flexors. *Exerc. Immunol. Rev*. 10: 75-90, 2004.
31. Hiscock, N., Chan, M.H., Bisucci, T., Darby, I.A. and Febbraio, M.A. Skeletal myocytes are a source of interleukin-6 mRNA expression and protein release during contraction: evidence of fiber type specificity. *FASEB J*. 18: 992-994, 2004.
32. Ibfelt, T., Petersen, E.W., Bruunsgaard, H., Sandmand, M. and Pedersen, B.K. Exercise-induced change in type 1 cytokine-producing $CD8^+$ T cells is related to a decrease in memory T cells. *J. Appl. Physiol*. 93: 645-648, 2002.
33. Inder, W.J., Hellemans, J., Swanney, M.P., Prickett, T.C.R. and Donald, R.A. Prolonged exercise increases peripheral plasma ACTH, CRH, and AVP in male athletes. *J. Appl. Physiol*. 85: 835-841, 1998.
34. Jonsdottir, I.H., Schjerling, P., Ostrowski, K., Asp, S., Richter, E.A. and Pedersen, B.K. Muscle contractions induce interleukin-6 mRNA production in rat skeletal muscles. *J. Physiol*. 528: 157-163, 2000.
35. Kanaley, J.A., Weltman, J.Y., Pieper, K.S., Weltman, A. and Hartman, M. L. Cortisol and growth hormone responses to exercise at different times of day. *J. Clin. Endocrinol. Metab*. 86: 2881-2889, 2001.
36. Komi, P.V. Stretch-shortening cycle: a powerful model to study normal and fatigyued muscle. *J. Biomec*. 33: 1197-1206, 2000.
37. Kraemer, W.J., Gordon, S.E., Fleck, S.J., Marchitelli, L.J., Mello, R., Dziados, J.E., Friedl, K., Harman, E., Maresh, C. and Fry, A.C. Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. *Int. J. Sports Med*. 12: 228-235, 1991.
38. Kraemer, W.J., Häkkinen, K., Newton, R.U., Nindl, B.C., Volek, J.S., McCormick, M., Gotshalk, L.A., Gordon, S.E., Fleck, S.J., Campbell, W.W., Putukian, M. and Evans, W.J. Effects of heavy-resistance training on hormonal response patterns in younger *vs*. older men. *J. Appl. Physiol*. 87: 982-992, 1999.
39. Kraemer, W.J., Marchitelli, L., Gordon, S.E., Harman, E., Dziados, J.E., Mello, R., Frykman, P., McCurry, D. and Fleck, S.J. Hormonal and growth factor responses to heavy resistance exercise protocols. *J. Appl. Physiol*. 69: 1442-1450, 1990.
40. Kvorning, T., Andersen, M., Brixen, K. and Madsen, K. Suppression of endogenous testosterone production attenuates the response to strength training: a randomized, placebo-controlled, and blinded intervention study. *Am. J. Physiol. Endocrinol. Metab*. 291: E1325-E1332, 2006.
41. Lambert, C.P., Wright, N.R., Finck, B.N. and Villareal, D.T. Exercise but not diet-induced weight loss decreases skeletal muscle inflammatory gene expression in frail obese elderly persons. *J. Appl. Physiol*. 105: 473-478, 2008.
42. Liu, C., Chang, Y.J., Chen, C.S. and Shiang, T.Y. Reflex modulation after long term passive repeated plyometric training. International Society of Biomechanics XXth Congress- ASB 29th Annual Meeting, Cleveland, Ohio, USA, 2005, p. 381.
43. Liu, C, Liu, T.C., Chen, C.S. and Shiang, T.Y. The study of training effect of passive repeated plyometric training machine. XIX International Symposia on Biomechanics in Sports, San Francisco, USA, 2001, pp. 147-150.
44. Liu, T.C., Kao, C.H. and Wang, P.S. Exercise and testosterone. *Adapt. Med*. 1: 20-31, 2009.
45. Luigi, L.D., Baldari, C., Sgrò, P., Emerenziani, G.P., Gallotta, M. C., Bianchini, S., Romanelli, F., Pigozzi, F., Lenzi, A. and Guidetti, L. The type 5 phosphodiesterase inhibitor tadalafil influences salivary cortisol, testosterone, and dehydroepiandrosterone sulphate responses to maximal exercise in healthy men. *J. Clin. Endocrinol. Metab*. 93: 3510-3514, 2008.
46. Malatesta, D., Cattaneo, F., Dugnani, S. and Maffiuletti, N.A. Effects of electromyostimulation training and volleyball practice on jumping ability. *J. Strength Cond. Res*. 17: 573-579, 2003.

47. Malisoux, L., Francaux, M., Nielens, H. and Theisenl, D. Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers. *J. Appl. Physiol*. 100: 771-779, 2006.
48. Markovic, G., Jukic, I., Milanovic, D. and Metikos, D. Effects of sprint and plyometric training on muscle function and athletic performance. *J. Strength Cond. Res*. 21: 543-549, 2007.
49. Mazzeo, R.S., Donovan, D., Fleshner, M., Butterfield, G.E., Zamudio, S., Wolfel, E.E. and Moore, L.G. Interleukin-6 response to exercise and high-altitude exposure: influence of α-adrenergic blockade. *J. Appl. Physiol*. 91: 2143-2149, 2001.
50. Meksawan, K., Venkatraman, J.T., Awad, A.B. and Pendergast, D.R. Effect of dietary fat intake and exercise on inflammatory mediators of the immune system in sedentary men and women. *J. Am. Coll. Nutr*. 23: 331-340, 2004.
51. Moldoveanu, A.I., Shephard, R.J. and Shek, P.N. Exercise elevates plasma levels but not gene expression of IL-1β, IL-6, and TNF-α in blood mononuclear cells. *J. Appl. Physiol*. 89: 1499-1504, 2000.
52. Nielsen, A.R., Mounier, R., Plomgaard, P., Mortensen, O.H., Penkowa, M., Speerschneider, T., Pilegaard, H. and Pedersen, B.K. Expression of interleukin-15 in human skeletal muscle–effect of exercise and muscle fibre type composition. *J. Physiol*. 584: 305-312, 2007.
53. Nieman, D.C., Ahle, J.C., Henson, D.A., Warren, B.J., Suttles, J., Davis, J.M., Buckley, K.S., Simandle, S., Butterworth, D.E., Fagoaga, O.R. and Nehlsen-Cannarella, S.L. Indomethacin does not alter natural killer cell response to 2.5 h of running. *J. Appl. Physiol*. 79: 748-755, 1995.
54. Nieman, D.C., Davis, J.M., Henson, D.A., Walberg-Rankin, J., Shute, M. Dumke, C.L., Utter, A.C., Vinci, D.M., Carson, J.A., Brown, A., Lee, W.J. McAnulty, S.R. and McAnulty, L.S. Carbohydrate ingestion influences skeletal muscle cytokine mRNA and plasma cytokine levels after a 3-h run. *J. Appl. Physiol*. 94: 1917-1925, 2003.
55. Nieman, D.C., Fagoaga, O., Butterworth, D.E., Warren, B.J., Utter, A., Davis, J., Henson, D.A. and Nehlsen-Cannarella, S.L. Carbohydrate supplementation affects blood granulocyte and monocyte trafficking but not function after 2.5 h of running. *Am. J. Clin. Nutri.* 66: 153-159, 1997.
56. Nieman, D.C., Henson, D.A., Smith, L.L., Utter, A.C., Vinci, D.M., Davis, J.M., Kaminsky, D.E. and Shute, M. Cytokine changes after a marathon race. *J. Appl. Physiol*. 91: 109-114, 2001.
57. Nindl, B.C., Kraemer, W.J., Deaver, D.R., Peters, J.L., Marx, J.O., Heckman, J.T. and Loomis, G.A. LH secretion and testosterone concentrations are blunted after resistance exercise in men. *J. Appl. Physiol*. 91: 1251-1258, 2001.
58. Northoff, H. and Berg, A. Immunologic mediators as parameters of the reaction to strenuous exercise. *Int. J. Sports Med*. 12 Suppl 1: S9-S15, 1991.
59. Ostrowski, K., Rohde, T., Asp, S., Schjerling, P. and Pedersen, B.K. Pro- and anti-inflammatory cytokine balance in strenuous exercise in humans. *J. Physiol. (Lond.)* 515: 271-291, 1999.
60. Ostrowski, K., Rohde, T., Zacho, M., Asp, S. and Pedersen, B.K. Evidence that IL-6 is produced in skeletal muscle during prolonged running. *J. Physiol*. 508: 949-953, 1998.
61. Pedersem, B.K. The diseasome of physical inactivity-and the role of myokines in muscle—fat cross talk. *J. Physiol*. 587: 5559-5568, 2009.
62. Pedersen, B.K. Special feature for the Olympics: effects of exercise on the immune system: exercise and cytokines. *Immunol. Cell. Biol*. 78: 532-535, 2000.
63. Pedersen, B.K. and Febbraio, M.A. Muscle as an endocrine organ: focus on muscle-derived interleukin-6. *Physiol. Rev*. 88: 1379-1406, 2008.
64. Pedersen, B.K. and Hoffman-Goetz, L. Exercise and immune system: regulation, integration and adaptation. *Physiol. Rev*. 80: 1055-1081, 2000.
65. Pedersen, B.K., Ostrowski, K., Rohde, T. and Bruunsgaard, H. The cytokine response to strenuous exercise. *Can. J. Physiol. Pharmacol*. 76: 505-511, 1998.
66. Pedersen, B.K., Steensberg, A. and Schjerling, P. Muscle-derived interleukin-6: possible biological effects. *J. Physiol*. 536: 329-337, 2001.
67. Pedersen, B.K., Thorbjörn, C.A., Åkerström, T.C.A., Nielsen, A.R. and Fischer, C.P. Role of myokines in exercise and metabolism. *J. Appl. Physiol*. 103: 1093-1098, 2007.
68. Penkowa, M., Keller, C., Keller, P., Jauffred, S. and Pedersen, B.K. Immunohistochemical detection of interleukin-6 in human skeletal muscle fibers following exercise. *FASEB J*. 17: 2166-2168, 2003.
69. Potteiger, J.A., Lockwood, R.H., Haub, M.D., Dolezal, B.A., Almuzaini, K.S., Schroeder, J.M. and Zebas, C.J. Muscle power and fiber characteristics following 8 weeks of plyometric training. *J. Strength Cond. Res*. 13: 275-279, 1999.
70. Quinn, L.S. Interleukin-15: A muscle-derived cytokine regulating fat-to-lean body composition. *J. Anim. Sc*. 86: E75-E83, 2008.
71. Rhind, S.G., Castellani, J.W., Brenner, I.K.M., Shephard, R.J., Zamecnik, J., Montain, S.J., Young, A.J. and Shek, P.N. Intracellular monocyte and serum cytokine expression is modulated by exhausting exercise and cold exposure. *Am. J. Physiol. Regul. Integr. Comp. Physiol*. 281: R66-R75, 2001.
72. Riechman, S.E., Balasekaran, G., Roth, S.M. and Ferrell, R.E. Association of interleukin-15 protein and interleukin-15 receptor genetic variation with resistance exercise training responses. *J. Appl. Physiol*. 97: 2214-2219, 2004.
73. Saghizadeh, M., Ong, J.M., Garvey, W.T., Henry, R.R. and Kern, P.A. The expression of TNF α by human muscle. Relationship to insulin resistance. *J. Clin. Invest*. 97: 1111-1116, 1996.
74. Sloan, R.P., Shapiro, P.A., DeMeersman, R.E., McKinley, P.S., Tracey, K.J., Slavov, I., Fang, Y. and Flood, P.D. Aerobic exercise attenuates inducible TNF production in humans. *J. Appl. Physiol*. 103: 1007-1011, 2007.
75. Steensberg, A., Febbraio, M.A., Osada, T., Schjerling, P., van Hall, G., Saltin, B. and Pedersen, B.K. Interleukin-6 production in contracting human skeletal muscle is influenced by pre-exercise muscle glycogen content. *J. Physiol*. 537: 633-639, 2001.
76. Steensberg, A., Keller, C., Starkie, R.L., Osada, T., Febbraio, M.A. and Pedersen, B.K. IL-6 and TNF-α expression in, and release from, contracting human skeletal muscle. *Am. J. Physiol. Endocrinol. Metab*. 283: E1272-E1278, 2002.
77. Steensberg, A., Toft, A.D., Bruunsgaard, H., Sandmand, M., Halkjær-Kristensen, J. and Pedersen, B.K. Strenuous exercise decreases the percentage of type 1 T cells in the circulation. *J. Appl. Physiol*. 91: 1708-1712, 2001.
78. Steinacker, J.M., Lormes, W., Reissnecker, S. and Liu, Y. New aspects of the hormone and cytokine response to training. *Eur. J. Appl. Physiol*. 91: 382-391, 2004.
79. Stell, R.D. and Torrie, J.H. Principals and Procedures of Statistics, 2nd edn., New York, McGraw-Hill, 1981.
80. Stewart, L.K., Flynn, M.G., Campbell, W.W., Craig, B.A., Robinson, J.P., Timmerman, K.L., McFarlin, B.K., Coen, P.M. and Talbert, E. The influence of exercise training on inflammatory cytokines and C-reactive protein. *Med. Sci. Sports Exerc*. 39: 1714-1719, 2007.
81. Suzuki, K., Nakaji, S., Yamada, M., Totsuka, M., Sato, K. and Sugawara, K. Systemic inflammatory response to exhaustive exercise. Cytokine kinetics. *Exerc. Immunol. Rev*. 8: 6-48, 2002.
82. Suzuki, K., Yamada, M., Kurakake, S., Okamura, N., Yamaya, K., Liu, Q., Kudoh, S., Kowatari, K., Nakaji, S. and Sugawara, K. Circulating cytokines and hormones with immunosuppressive but neutrophil-priming potentials rise after endurance exercise in humans. *Eur. J. Appl. Physiol*. 81: 281-287, 2000.
83. Thomson, A.W. The cytokine handbook. London, Norfolk, England: Academic Press, 1994
84. Uchida, M.C., Nosaka, K., Ugrinowitsch, C., Yamashita, A.,

Martins Jr, E., Moriscot, A.S. and Aoki, M.S. Effect of bench press exercise intensity on muscle soreness and inflammatory mediators. *J. Sports Sci*. 27: 499-507, 2009.

85. Ullum, H., Haahr, P.M., Diamant, M., Palmo, J., Halkjaer-Kristensen, J. and Pedersen, B.K. Bicycle exercise enhances plasma IL-6 but does not change IL-1 α, IL-1 β, IL-6, or TNF-α pre-mRNA in BMNC. *J. Appl. Physiol*. 77: 93-97, 1994.
86. Vassilakopoulos, T., Karatza, M.H., Katsaounou, P., Kollintza, A., Zakynthinos, S. and Roussos, C. Antioxidants attenuate the plasma cytokine response to exercise in humans. *J. Appl. Physiol*. 94: 1025-1032, 2003.
87. Viru, A., Karelson, K. and Smirnova, T. Stability and variability in hormonal responses to prolonged exercise. *Int. J. Sports Med*. 13: 230-235, 1992.
88. Viru, A. and Viru, M. Cortisol—essential adaptation hormone in exercise. *Int. J. Sports Med*. 25: 461-464, 2004.
89. Volek, J.S., Kraemer, W.J., Bush, J.A., Incledon, T. and Boetes, M. Testosterone and cortisol in relationship to dietary nutrients and resistance exercise. *J. Appl. Physiol*. 82: 49-54, 1997.
90. Wilk, K.E., Voight, M.L., Keirns, M.A., Gambetta, V., Andrews, J.R. and Dillman, C.I. Stretch-shortening drills for the upper extremities: theory and clinical application. *J. Orthop. Sports Phys. Ther*. 17: 225-239, 1993.
91. Wilkerson, J.E., Horvath, S.M. and Gutin, B. Plasma testosterone during treadmill exercise. *J. Appl. Physiol*. 49: 249-253, 1980.
92. Wilson, G.J., Murphy, A.J. and Giorgi, A. Weight and plyometric training: effects on eccentric and concentric force production. *Can. J. Appl. Physiol*. 21: 301-315, 1996.
93. Wilson, G.J., Newton, R.U., Murphy, A.J. and Humphries, B.J. The optimal training load for the development of dynamic athletic performance. *Med. Sci. Sports Exerc*. 25: 1279-1286, 1993.
94. Wu, C.T., Yu, H.P., Chung, C.Y. Lau, Y.T. and Liao, S.K. Attenuation of lung inflammation and pro-inflammatory cytokine production by resveratrol following trauma-hemorrhage. *Chinese J. Physiol*. 51: 363-368, 2008.
95. Yang, A.L., Su, C.T., Lin, K.L. and Lee, S.D. Enhancement of vascular function mediated by insulin and insulin-like growth factor-1 following single exercise session. *Chinese J. Physiol*. 51: 71-77, 2008.
96. Zaldivar, F., Wang-Rodriguez, J., Nemet, D., Schwindt, C., Galassetti, P., Mills, P.J., Wilson, L.D. and Cooper, D.M. Constitutive pro- and anti-inflammatory cytokine and growth factor response to exercise in leukocytes. *J. Appl. Physiol*. 100: 1124-1133, 2006.