The effect of 30-m repeated sprint exercise on muscle damage indicators, serum insulin-like growth factor-I and cortisol

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Summary

Study aim: The purpose of this study was to examine the effects of a repeated sprint exercise protocol on muscle damage indicators, serum IGF-I and cortisol levels.

Material and methods: Nine trained male subjects (age 23.3 ± 3.6 years) completed a repeated sprint protocol consisting of two sets of 10×30-m maximal sprints with 30 s of active recovery between sprints and 5 min of passive recovery between sets. The isometric strength and flexibility were measured before, immediately after and 24 hours after exercise. 30-m maximal sprint time was measured before and 24 hours after exercise. Blood samples were taken before, immediately after and 24 hours after exercise.

Results: Isometric strength and flexibility were significantly decreased after exercise and 24 hours after exercise ($p < 0.05$). 30-m sprint time was significantly increased 24 hours after exercise ($p < 0.05$). A significant increase in serum lactate dehydrogenase, IGF-I and cortisol were found after exercise ($p < 0.05$). Serum creatine kinase increased significantly immediately after and 24 hours after exercise compared to pre-exercise values ($p < 0.05$).

Conclusion: Our data show that due to increased serum IGF-I level, repeated sprint exercise may have anabolic effects as well as traumatic effects on the muscles.

Key words: Creatine kinase – Anabolic process – Multiple-sprint sports – Eccentric exercise

Introduction

The ability to perform repeated brief maximal sprints interspersed with minimal recovery periods is considered an important fitness component in multiple-sprint sports such as basketball, rugby, soccer and field hockey [34]. In sprinting, eccentric muscle contractions during the landing phase have been reported to result in multiple areas of microscopic damage in the muscles [14, 17, 37]. Efflux of the intramuscular enzymes such as creatine kinase (CK) and lactate dehydrogenase (LDH) into the blood [5, 30] and prolonged deterioration in muscle function as evidenced by reductions in isometric strength [8, 41], range of motion [8], and rapid dynamic muscle functions such as jumping and sprinting [38] are discussed as some of the symptoms of muscle damage in the literature.

Exercise-induced muscle damage is probably essential for normal muscle function and adaptation [27]. Insulin-like growth factor I (IGF-I) has been suggested to play an important role in formation, maintenance, and regeneration of skeletal muscles [15] as well as energy balance [3]. The possible source for increase in circulating IGF-I in response to exercise may be release from the liver due to growth hormone secretion as well as release from the exercising muscle [4, 7]. Eccentric contractions have been shown to induce production of IGF-I within the skeletal muscle [2, 44]. IGF-I is involved in tissue regeneration after exercise-induced muscle damage [2]. However, in the literature, there are conflicting reports regarding the acute effects of exercise on the circulating IGF levels, indicating an increase [9, 21], decrease [20], or no change [28, 35]. The possible reason for discrepancies in these findings may be the differences in exercise protocols (type, duration, and intensity) as well as fitness level of the subjects [21, 32].

Exercise is a particular form of metabolic and physical stress that is shown to be a potent activator of the

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hypothalamo-pituitary-adrenal axis (HPA), providing an elevation of cortisol level [6, 26]. The response of the HPA axis to exercise is different depending on the duration and intensity of the exercise and the fitness level of the subject [23, 26]. Blood cortisol levels have been shown to increase after both short-term maximal exercise [6, 12] and prolonged exercise by at least 60% of the maximal oxygen uptake ($\dot{V}O_{2max}$) [26]. Analysis of serum cortisol levels can provide valuable information on the physiological stress response and adaptation to single and repeated exercise sessions.

The use of repeated sprint exercise for the training and testing of athletes has become more popular in recent years. It is important to evaluate the anabolic and catabolic effects of the training in order to enhance the performance of the athlete. It has been documented that a 30-m repeated sprint exercise protocol replicating the activity profile of multiple-sprint sports results in muscle damage [17, 19]. However, to our knowledge, the circulating IGF-I response to repeated sprint exercise has not been previously reported. The purpose of this study was to examine the effects of a 30-m repeated sprint exercise protocol on muscle damage indicators, serum IGF-I and cortisol levels.

Material and methods

Subjects and experimental design

Nine healthy male subjects (mean ± SD; age 23.3 ± 3.6 years, height 177.1 ± 6.4 cm, mass 73 ± 9 kg) volunteered to participate in the present study. All the subjects were university-level athletes from various team sport backgrounds competing (at least 4 years) and training at least three times a week. The study was explained to all participants in detail, and written informed consent forms were acquired. Measurements were performed following the approval of the Cukurova University Medical Faculty Ethics Committee and carried out in accordance with the Declaration of Helsinki. Subjects were asked to refrain from ingesting alcohol, performance-enhancing supplements, and/or anti-inflammatory drugs for the duration of the study. Subjects' 30-m sprint performance was measured before and 24 hours after exercise. Isometric strength and flexibility were measured before, immediately after and 24 hours after exercise. Blood samples were taken before, immediately after (within 5 min) and 24 hours after exercise to determine serum CK, LDH and cortisol. Serum IGF-I values were taken before and immediately after exercise (Fig. 1). All testing took place at the Cukurova University Sports Physiology Laboratory. Blood samples were studied at the central laboratory of Cukurova University Medical Faculty Hospital.

Repeated sprint exercise protocol

The repeated sprint exercise protocol consisted of two sets of ten repetitions of 30-m all-out sprints with 30 s of active recovery between sprints and 5 min of passive recovery between sets. Sprint times were measured for each 30-m sprint with two ports of light sensors (New test Oy, Oulu, Finland) placed at the starting line and the finishing line. The recovery time was controlled by a hand-held stopwatch. The repeated sprint exercise test was performed after a standard warm-up procedure that included 5 min of self-paced jogging, 5 min of stretching for the upper and lower extremities and 5 min of specific running drills at increasing speed. After the warm-up, each subject performed three preliminary 30-m maximal sprints separated by a 2 min recovery period. The fastest sprint time was used as the criterion score for the repeated sprint exercise test. After these trials, subjects rested for 5 min before the start of the repeated sprint exercise test. If the performance in the first sprint of the repeated sprint exercise test was worse than the criterion score (i.e., an increase in time greater than 2.5%), the test was immediately terminated and subjects were required to repeat the exercise test with maximum effort after a 5 min rest.

The subjects stood 30 cm behind the start line to avoid premature triggering of the timing system and completed 2 sets of 10 × 30-m sprints. During the 30 s active recovery period between sprints, the subjects decelerated within the 10-m distance after passing the finish line and jogged back to the starting line. Subjects were instructed to complete
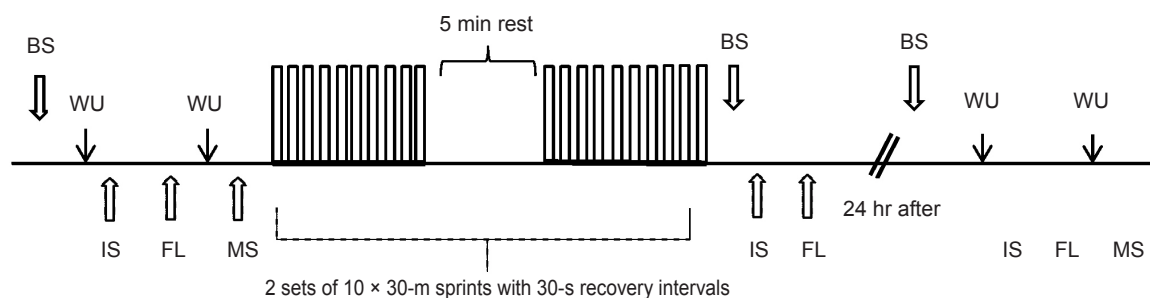

Fig. 1. A schematic representation of the study protocol. BS = Blood sampling, WU= Warming up, IS= Isometric strength, FL = Flexibility, MS= 30 m maximal sprint

all sprints as fast as possible, and strong verbal encouragement was provided to each subject during all sprints. During the active recovery, continuous verbal feedback was provided to ensure the subjects had returned to the start position in time to begin the next sprint.

From the repeated sprint protocol data (for each sets), the best sprint time (the fastest 30-m sprint time), mean sprint time (mean time to complete 10 sprints) and the fatigue index (or the sprint decrement score) expressed as a percentage were selected for the analysis. The fatigue index (FI) was used as an indication of fatigue and was calculated according to Fitzsimmons et al. [13]:

$$FI = [100 \times (\text{total sprint time} \div \text{ideal sprint time})] - 100,$$

where total sprint time = sum of sprint times from all sprints, and ideal sprint time = number of sprints × best sprint time.

30-m sprint test

30-m sprint measurements were repeated 24 hours after repeated sprint exercise. Before the 30-m sprint test, subjects completed a standardized 15 min warm-up period including self-paced jogging, stretching and specific running drills. The subjects stood 30 cm behind the start line to avoid premature triggering of the timing system and completed three maximal sprints which were separated by 2 min of recovery. The fastest 30-m sprint time was recorded for data analysis.

Maximal isometric strength

Performance measurements began with an isometric strength test. Each subject was required to complete a 10 min warm-up period involving self-paced jogging and stretching. Maximal isometric knee extension strength from the dominant leg was measured with an isokinetic dynamometer (Cybex, Norm 6000). The dynamometer was set up according to the manufacturer's instructions; the isometric contractions were performed at 60° knee angle and gravity compensation was employed to allow for inter-subject variations in moment acting upon limb weight. All participants completed two isometric strength tests with 5 s duration and separated by a 60 s recovery period. Standardized verbal instructions and encouragement were provided throughout the protocol. The best peak isometric strength value was used in the data analysis.

Sit-and reach test

A standard sit-and-reach box was used to measure the subjects' flexibility. Subjects were seated on the floor with their bare feet touching the sit-and-reach box. The subjects then slowly reached forward towards their toes while keeping their legs straight and their hands together pushing the sliding ruler that was centered on the top of the box to obtain the sit-and reach test scores. The test was performed three times and the best score was recorded.

Blood analyses

Blood samples (10 ml) were collected from the antecubital vein for serum IGF-I, cortisol, CK and LDH measurements. Whole blood was allowed to clot at room temperature. Subsequently, blood was centrifuged at 3500 rpm for 5 min and was separated into serum. Serum CK and LDH were measured with the colorimetric assay procedure and cortisol was measured with electrochemiluminescence immunoassay (Elecsys Modular Analytics E170; Roche Diagnostics, Mannheim, Germany). Serum IGF-I (KAPB2010; Biosource Europe S.A., Nivelles, Belgium) levels were determined using enzyme-linked immunosorbent assay (ELISA) (MedispecESR 200).

Statistical analysis

Data are reported as mean ± standard deviation (SD). Statistical significance was accepted at $p < 0.05$. The normality distribution of the data was checked with the Shapiro-Wilk test. All data met the assumption of normal distribution with the exception of the fatigue index. 30-m maximal sprint time, best sprint time, mean sprint time and IGF-I values were compared using the paired t-test. Changes in fatigue index for the first set and second set were analyzed using the Wilcoxon signed rank test. Changes in isometric strength, flexibility, CK, LDH and cortisol were analyzed using a repeated measures analysis of variance (ANOVA). Bonferroni correction was used as a post hoc test when main effects were found to be significant. To allow a better interpretation of the results, effect sizes were also calculated using Cohen's d [36]. Effect sizes were interpreted as negligible ($d < 0.2$), small ($0.2 \leq d < 0.5$), medium ($0.5 \leq d < 0.8$) or large ($0.8 \leq d$). SPSS version 16 was used for all analyses (SPSS Inc., Chicago, IL).

Results

The repeated sprint protocol best sprint time, mean sprint time and fatigue index were 4.14 ± 0.14 s, 4.32 ± 0.14 s, 4.3 ± 2.4% for the first set and 4.32 ± 0.18 s, 4.57 ± 0.2 s, 5.9 ± 4.7% for the second set, respectively. The best sprint time ($d = 1.18$, $p = 0.008$) and mean sprint time ($d = 1.54$, $p = 0.002$) were significantly lower in the first set compared to the second set. The fatigue index was not significantly different between the two sets ($p > 0.05$).

30-m maximal sprint, isometric strength and flexibility values are presented in Table 1. 30-m sprint performance measured 24 hours after exercise was significantly lower than the pre-exercise value ($d = 0.6$, $p = 0.01$). The subjects' flexibility decreased significantly immediately after

Table 1. Changes in the 30-m maximal sprint, isometric strength and flexibility of subjects after exercise

	Isometric Strength [N]	Flexibility [cm]	30-m Sprint [s]
Pre-exercise	291.8 ± 50.4	28.6 ± 5.1	4.1 ± 0.15
Post-exercise	240.3 ± 32.9*	26.9 ± 6.5*	
24 hr after exercise	256.6 ± 43*	26 ± 5.6*	4.2 ± 0.2*

Values are mean ± SD. * – Significantly different from pre-exercise (p < 0.05).

(d = 0.31, p = 0.03) and 24 hours after (d = 0.51, p = 0.001) exercise compared to the pre-exercise value. A significant decrease in the isometric strength was observed immediately after (d = 1.28, p = 0.004) and 24 hours (d = 0.8, p = 0.02) after exercise.

Serum IGF-I, cortisol, CK and LDH values are presented in Table 2. Serum CK level increased significantly immediately after (d = 1.03, p = 0.001) and 24 hours after exercise (d = 1.83, p = 0.01) compared to pre-training values. A significant increase in serum LDH level was observed immediately after exercise (d = 1.76, p = 0.008), whereas it remained unchanged 24 hours after exercise (p > 0.05). A significant increase in serum IGF-I level was found after exercise (d = 0.42, p = 0.03). Serum cortisol level increased significantly immediately after exercise (d = 2.41, p = 0.008), whereas it remained unchanged 24 hours after exercise compared to pre-exercise values (p > 0.05).

Discussion

Repeated sprint exercise is one of the most commonly used training methods in multiple-sprint sports [34]. This investigation was performed to evaluate the effects of a repeated sprint exercise protocol on anabolic and catabolic indicators. The decrease in isometric strength, flexibility and sprint performance together with the increase in serum CK and LDH levels immediately after and 24 hours after exercise may indicate the presence of muscle tissue damage caused by repeated sprinting. The acute increase in serum IGF-I in response to the repeated sprint exercise protocol may suggest that the anabolic process is triggered [25]. In addition, significantly elevated cortisol levels

following the exercise suggest that the repeated sprint exercise was intensive enough to cause physiological stress.

Repeated sprinting together with 10-m rapid deceleration at the end of each sprint has been reported to have the potential to cause muscle damage [17, 19, 37]. In this study, the increases in serum CK and LDH levels following repeated sprint exercise provide evidence of muscle damage [5, 30]. Consistent with our results, previous studies have reported that the repeated sprint protocol consisting of fifteen 30-m sprints interspersed by 60 s of rest resulted in muscle damage [17, 19]. Eccentric muscle contractions during the landing phase of the sprinting may cause frequent abnormalities of the contractile material and the cytoplasmic organelles at the ultrastructural level [14]. These structural abnormalities may explain the reduction of athletic performance and changes in CK and LDH levels as reported in the literature [14, 17, 37]. Changes in CK levels in our study were similar to those previously reported in trained male athletes using the repeated sprint protocol [17].

The loss of maximal isometric strength for several days is considered to be an accurate and reliable indirect marker of muscle damage [41]. The decrease in isometric strength of the knee extensors immediately after and 24 hours after the repeated sprint exercise in the present study is consistent with the findings of previous studies [17]. Our findings also showed that 30-m sprint performance was significantly reduced 24 hours after repeated sprint exercise. Similarly, previous studies reported that 30-m and 10-m sprint performance decreased 24 hours after a muscle-damaging exercise protocol consisting of plyometric jumps or repeated sprints [16, 19, 38].

It has been shown that flexibility reduction is associated with muscle damage [1, 43]. In the present study,

Table 2. Serum IGF-I, cortisol, CK and LDH variables of subjects after exercise

	IGF-1 [ng/ml]	Cortisol [µg/dl]	CK [U/L]	LDH [U/L]
Pre-exercise	482.8 ± 60.8	14.5 ± 4.9	211.7 ± 69.2	376.1 ± 47.6
Post-exercise	509.3 ± 72.6*	25.1 ± 4.4*	286.5 ± 83.9*	456.7 ± 49.3*
24 hr after exercise		12.5 ± 2.2	665 ± 365.9*	382.5 ± 68.7

Values are mean ± SD. * – Significantly different from pre-exercise (p < 0.05).

a significant decrease in flexibility was observed immediately and 24 hours after repeated sprint exercise. We assessed flexibility by the sit-and-reach test, which is typically used to evaluate flexibility of the low back and hamstring muscles. In repeated bursts of running, the swing phase may cause hamstring damage [42] and a decrease in flexibility [1, 43]. Loss of flexibility may be associated with a rise in passive tension and swelling of the muscle together with volume changes exerting strain on perimysial and epimysial connective tissue elements [18].

IGF-I plays a central role in exercise-induced anabolic adaptations of skeletal muscle [15]. The present study demonstrates that repeated sprint exercise increases the serum IGF-I level. The increase in serum IGF-I may reflect exercise-related anabolic adaptations [25]. However, conflicting results regarding the acute effect of exercise on serum or plasma IGF-I levels have been reported in the literature. The reasons for differences in circulating IGF-I in response to exercise have not been clarified. High-intensity exercise at above the lactate threshold has been shown to stimulate larger increases in serum IGF-I than lower intensity exercise at below the lactate threshold [32]. On the other hand, Copeland et al. found that continuous exercise at 60–65% of $\dot{V}O_{2max}$ and interval exercise at 80–85% of $\dot{V}O_{2max}$ of equal duration (20 min) led to similar increases in the circulating level of IGF-I [9]. Besides the exercise intensity, the IGF-I response appears to depend on the duration of the exercise as well. For example, it has been shown that a single 90 s maximal cycle exercise resulted in an increase in serum IGF-I levels [21], whereas a single 30 s maximal cycle exercise did not lead to an increase [31]. In addition, the effect of exercise on IGF-I level may also depend on the differences in running distances in each interval. A significant increase in serum IGF-I was observed after increasing (i.e., 100–200–300–400-m) and decreasing distance (i.e., 400–300–200–100-m) of sprint interval runs at 80% of the maximal speed [29]. However, no significant increase in serum IGF-I was observed after four 250-m sprint interval runs at 80% of the maximal speed [28]. Energy deficit induced by exercise has also been shown to cause a reduction in circulating IGF-I [33]. This may be observed especially following long-term exercises such as marathon running [20]. Besides all this, the increase in circulating IGF-I in response to exercise is associated with the subjects' fitness status [11]. On the other hand, exercise-induced muscle damage may be one of the factors triggering the acute increase in serum IGF-I [2, 44]. It has been suggested that IGF-I may modulate tissue regeneration after mechanical damage [2]. Multiple sprint repetitions at maximal effort with short recovery intervals together with muscle tissue damage may explain the increased serum IGF-I in our study. To our knowledge, this is the first study that examines serum IGF-I level in response to repeated sprint exercise.

Cortisol is one of the primary catabolic hormones that increases protein degradation and reduces protein synthesis [10]. On the other hand, the acute increase in blood cortisol levels during exercise is essential for a normal metabolic response and adaptation to exercise [22]. During the recovery phase after exercise, the blood cortisol level usually decreases rapidly and, within hours, reaches the initial value again [39]. The exercise-induced cortisol increase depends on the duration and intensity of the exercise and the fitness level of the subject [23, 26]. Cortisol levels have been shown to be correlated linearly with exercise intensity and duration [31]. However, there are limited studies investigating blood cortisol response to repeated short-sprint exercise in the literature. Serum cortisol has been shown to increase after single or repeated 30 s maximal sprints [12, 40]. In addition, serum cortisol has been shown to increase following repeated sprint exercise (10 sets of 5×4 s cycle sprints, interspersed with 20 s recovery intervals) [24]. In this study, the significantly elevated serum cortisol following the repeated 30-m sprint exercise, which is in agreement with previously published data [24, 41], indicates that this type of physical activity was sufficient to cause a stressful situation for the body.

Conclusions

Sprint type of exercises is frequently used for athletic development. The results of the present study indicate that 30-m repeated sprint exercise led to significant increases in muscle damage indicators together with serum IGF-I and cortisol levels. Therefore, coaches have to keep in mind the anabolic effects as well as traumatic effects of repeated sprint exercise when planning the training period.

Conflict of interest: Authors state no conflict of interest.

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