Neuromuscular Fatigue and Recovery in Elite Female Soccer: Effects of Active Recovery

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ABSTRACT

ANDERSSON, H., T. RAASTAD, J. NILSSON, G. PAULSEN, I. GARTHE, and F. KADI. Neuromuscular Fatigue and Recovery in Elite Female Soccer: Effects of Active Recovery. *Med. Sci. Sports Exerc.*, Vol. 40, No. 2, pp. 372–380, 2008. **Purpose:** To investigate the time course of recovery from neuromuscular fatigue and some biochemical changes between two female soccer matches separated by an active or passive recovery regime. **Methods:** Countermovement jump (CMJ), sprint performance, maximal isokinetic knee flexion and extension, creatine kinase (CK), urea, uric acid, and perceived muscle soreness were measured in 17 elite female soccer players before, immediately after, 5, 21, 45, 51, and 69 h after a first match, and immediately after a second match. Eight players performed active recovery (submaximal cycling at 60% of HR_{peak} and low-intensity resistance training at < 50% 1RM) 22 and 46 h after the first match. **Results:** In response to the first match, a significant decrease in sprint performance (−3.0 ± 0.5%), CMJ (−4.4 ± 0.8%), peak torque in knee extension (−7.1 ± 1.9%) and flexion (−9.4 ± 1.8%), and an increase in CK (+152 ± 28%), urea (15 ± 2), uric acid (+ 11 ± 2%), and muscle soreness occurred. Sprint ability was first to return to baseline (5 h) followed by urea and uric acid (21 h), isokinetic knee extension (27 h) and flexion (51 h), CK, and muscle soreness (69 h), whereas CMJ was still reduced at the beginning of the second match. There were no significant differences in the recovery pattern between the active and passive recovery groups. The magnitude of the neuromuscular and biochemical changes after the second match was similar to that observed after the first match. **Conclusion:** The present study reveals differences in the recovery pattern of the various neuromuscular and biochemical parameters in response to a female soccer match. The active recovery had no effects on the recovery pattern of the four neuromuscular and three biochemical parameters. **Key Words:** CREATINE KINASE, UREA, COUNTERMOVEMENT JUMP, RECOVERY PATTERN

For elite female soccer players the number of competitive matches per year, including domestic and international matches, has markedly increased in the last decade. Likewise, the total distance covered during the matches have increased from 8.5 km in the early 1990s to 10.3 km in 2005 (6,17). It is known that the amount of high-intensity running has increased in male soccer during the last decade (sprinting distance increased by 37% in 2003 compared with 1991) (21). The same trend is seen for female players, even though it is difficult to compare throughout the years because there is a lack of data. In 2005, a mean of 1.3 km of high-intensity running was reported in an elite female soccer match (17). Altogether, increased match intensity and frequency highlight the importance of proper recovery between matches to perform optimally. Effective recovery is especially important in competitive tournaments, where the recovery period between two international matches is limited to 2 d. Consequently, it is important to understand the time course of the physiological changes in response to a soccer match in females and to develop effective strategies to accelerate the recovery process.

Soccer is a complex sport that involves many activities (tackles, jumps, and direction and speed changes) that put a great strain on several neuromuscular and metabolic parameters (3). In this respect, sprint performance (22,26), jump performance (26), and isokinetic knee extension and flexion (26) have been shown to be reduced immediately after a male soccer match. In parallel with the changes in the neuromuscular parameters, it has been shown that biochemical parameters such as uric acid and urea concentrations are increased immediately after a male soccer match (2). In other football codes, several other biochemical markers have been investigated (12). No significant increases were found in urea, uric acid, alanine aminotransferase, or creatine kinase (CK) immediately after a football game. However, myoglobin and aspartate aminotransferase increased immediately after the game for starting players (12). In female soccer, very little information is available on neuromuscular and biochemical changes in response to a match (11), and the time course of

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physiological changes for a period of time ranging from minutes to several hours after a match has not been fully examined.

The load of training and competition may impair the player's performance. Inappropriate recovery may thus predispose some players to overload injuries and reduced performance (5). Therefore, full recovery is important to enable the players to perform maximally. It is a common belief that active recovery enhances the recovery process by accelerating the return to homeostatis after a soccer match. This involves enhancing the rate of blood lactate removal, reducing the severity and duration of exercise induced muscle injury and delayed muscle soreness, restoration of energy levels in skeletal muscle, and quicker normalization of performance parameters (e.g., jump, sprint, and strength performance) (5). In practice, several recovery strategies are used: low-intensity training, active warm-down, massage, stretching, resistance training, deep-water running, and contrast-temperature water immersion (5). These strategies are designed to minimize the stress induced by matches and accelerate the recovery (5). The theoretical overall advantage of active recovery would be to allow the players to tolerate higher training loads (intensity, volume, and frequency) and to ultimately enhance performance (5,27). Active recovery training may include submaximal cycling and low-intensity resistance training.

To our knowledge, there is only one scientific report evaluating the effects of an active recovery program after a soccer match (25). The study was on male players who performed acute warm-down for 12 min immediately after the match. The active warm-down had a positive effect on the recovery of jump and sprint performance and perceived muscle soreness (28). The effectiveness of an active recovery program performed between two consecutive matches has not previously been evaluated in female soccer players.

Given the above discussion, the overall aim of this investigation was to study the time course of changes of four neuromuscular and three biochemical parameters in response to two female soccer matches separated by either an active or passive recovery. More specifically, we investigated 1) changes in jump and sprint performance, maximal isokinetic knee flexion and extension, blood CK, urea and uric acid concentrations, and perceived muscle soreness before, immediately after, and 5, 21, 45, 51, and 69 h after the first match; 2) changes in the neuromuscular and biochemical parameters immediately after a second match played 72 h after the first match; and 3) the effects of an active recovery regime performed at 22 and 46 h after the first match. On the basis of the theory that light muscle activity may accelerate the return of homeostasis in exercised muscle, we hypothesized that the neuromuscular function would recovery quicker for the active recovery group compared with the passive recovery group.

METHODS

Subjects. Twenty-two elite female soccer players (two goalkeepers, eight defenders, eight midfielders, and four forwards) from two teams of the highest division in Sweden and Norway played two international 90-min friendly matches separated by 72 h. Two defenders and one midfielder were not available on the recovery days between the matches and were therefore excluded from the data analysis. Because the physical loading of goalkeepers differs from that of field players, they were not included in the analysis. The physical and anthropometrical characteristics of the players are shown in Table 1. The players were fully informed of the experimental procedures and possible discomforts associated with the study before given their written informed consent to participate. The study was conducted according to the Declaration of Helsinki and approved by the regional ethics committee of Uppsala, Sweden.

Experimental design. The two matches were played during a period of 4 d and were separated by 2 d of either active or passive recovery. The same players in both teams participated in both games and occupied the same field position. After the first match, the players in both teams were randomly assigned to the active recovery group ($N = 8$) and the passive recovery group ($N = 9$). The players were matched for age, height, weight, maximal oxygen consumption, and field playing position. The active recovery consisted of a low-intensity training program (submaximal cycling at 60% HR_{peak} and approximately 45% $\dot{V}O_{2peak}$) and low-intensity resistance training (< 50% 1RM) performed at 22 and 46 h after the first match. On the match day, baseline values for neuromuscular parameters were obtained 3 h before the match. Subsequent tests were performed immediately after (within 15 min), 5, 21, 27, 45, 51, and 69 h after the first match, and immediately after the second match (Fig. 1). The following measurements were performed: 20-m sprint, countermovement jump (CMJ), maximal isokinetic knee extension and flexion, and perceived muscle soreness. Blood samples were taken 3 h before, immediately after, 21, 45, and 69 h after the first match, and immediately after the second match. The following biochemical parameters were analyzed: CK, urea,

TABLE 1. Physical characteristics of the players participating in the study.

Group	*N*	Age (yr ± SD)	Weight (kg ± SD)	Height (cm ± SD)	HR_{peak} (bpm ± SD)	$\dot{V}O_{2peak}$ ($mL \cdot min^{-1} \cdot kg^{-1}$ ± SD)
Active	8	22.6 ± 4.2	63.3 ± 7.1	167.1 ± 5.7	198 ± 6	55.4 ± 3.6
Passive	9	21.6 ± 2.6	65.0 ± 4.6	167.2 ± 4.7	199 ± 6	53.8 ± 2.3

Values are means ± SD.

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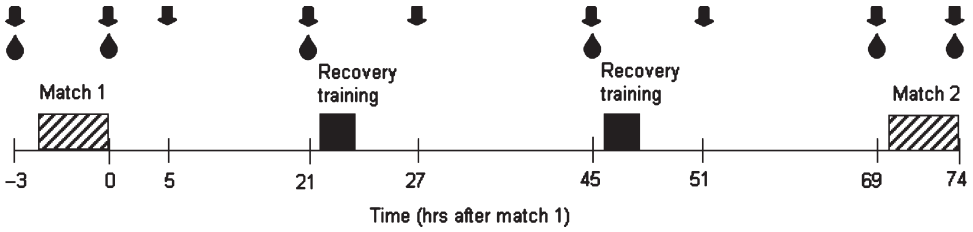

FIGURE 1—Test schedule for the whole test period. ***Downward arrows*** **denote the time points when sprint, jump, isokinetic strength, and perceived muscle soreness were measured, and** ***drop marks*** **denote the time points for blood sampling.**

and uric acid concentrations. Two days before the commencement of the study, all subjects performed a maximal oxygen consumption test running on a treadmill, and 1 d before the start of the study, the players were familiarized with all testing procedures.

Evaluation of work intensity and fluid loss during the matches. To compare the work intensity of the matches in our study with those previously reported in elite female soccer (17), heart rate was recorded (Polar Team System, Polar Electro OY, Kempele, Finland), and periods of high-intensity running were registered during the matches. The players wore a heart rate monitor around their chests, and data were continuously collected every 5 s. Heart rate was collected from all players, and amount of high-intensity running was collected from a total of 10 players who played in different field positions (four defenders, three midfielders, and three forwards). These 10 players gave a representation of the movement pattern during the matches. Each of these 10 players was filmed close up by a digital camera (Canon DM-MV 600) during the entire first and second matches. The cameras were positioned at the side of the field, at the level of the midfield line, at a height of about 15 m above the field and at a distance of 30–40 m from the side line. The videotapes were later replayed on a monitor for computerized coding of the activity pattern. The locomotor speed ranges for high-intensity running were chosen according to Krustrup et al. (17) for moderate-speed running (15 $km \cdot h^{-1}$), high-speed running (18 $km \cdot h^{-1}$), and sprinting (25 $km \cdot h^{-1}$). The distance covered for each activity within each interval was determined as the product of the total time and mean speed for that activity. All match recordings included in the present study were analyzed by the same experienced observer with a coefficient of variation for test–retest analysis of < 5% for distance covered in high-intensity running.

The players were weighed before and immediately after the match, using a digital scale (Seca 708, Seca Ltd, Birmingham, United Kingdom). The players were allowed to use a sports drink *ad libitum* (Maxim Energy, Maxim Sports Nutrition) during the match, and their fluid intake was recorded. The fluid loss was calculated by the formula: fluid loss = Δ body weight + fluid intake.

Active recovery regime. The active recovery regime consisted of a low-intensity training program lasting 1 h. The training consisted of 20 min of submaximal cycling (60% HR_{peak} and approximately 45% $\dot{V}O_{2peak}$), 30 min of low-intensity resistance training (< 50% 1RM) with exercises for both the upper and lower body, and 10 min of submaximal cycling (60% HR_{peak}). The exercise intensity during the cycling was monitored using a heart rate monitor (S610i, Polar Electro OY, Kempele, Finland). The rationale for using cycling as low aerobic training was that it increases blow flow in the leg muscles and minimizes the load on the muscles, which is proposed to be beneficial for recovery (27). Also, performing low-resistance weight training may enhance the protein metabolism in the exercised muscle, which would be beneficial for recovery. Additionally, the recovery regime was designed to mirror the recovery training used by many Nordic soccer teams. During the active recovery sessions, the subjects drank 1 L of the sports drink, giving a carbohydrate load of 60 g.

Sprint, jump, and isokinetic strength tests. Before the sprint, CMJ, and isokinetic strength tests, the players performed a standardized warm-up consisting of 5 min of low-intensity running (according to YOYO intermittent endurance test level 1) (16). After the warm-up, the players performed three maximal 20-m sprints, and the best result was used in the data analysis. The players were instructed to stand with one foot on a marker, and time started when the subjects touched a mechanical switch (placed 88 cm from the marker) in the first step. Sprint time was thereafter measured with photocells every 10 m. The photocells were placed at a height of 133 cm. CMJ was performed on a force plate (SG 9, Advanced Mechanical Technology Inc., Newton, MA) and low-pass filtered at 1050 Hz. The CMJ started from a standing position with the hands fixed to the hips. Jump height was calculated from the vertical reaction force impulse during take-off. At each test, the players performed three jumps, and the best was used in the data analysis (CV < 5%). Body mass was included in the jump-height calculation, and this was measured on the force plate before the first jump at each test. Isokinetic concentric knee extensions and flexions were performed on a Cybex 6000 dynamometer (Lumex, Ronkonfoma, NY) and Technogym REV 9000 (Gambettola, Italy) to enable testing of all subjects in the shortest possible period of time. Although controlled studies revealed that there were no differences in performance measured using the two instruments, the same

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TABLE 2. Heart rate during the matches for both groups.

		Match 1			Match 2		
Group	*N*	First Half	Second Half	Total Match	First Half	Second Half	Total Match
Active	8	165 ± 3	162 ± 3	163 ± 3	173 ± 3*	169 ± 3*	171 ± 3*
Passive	9	162 ± 2	162 ± 2	161 ± 2	172 ± 3*	165 ± 2*	168 ± 2*

Values are heart rate in beats per minute (means ± SE). * Significantly higher ($P < 0.05$) compared with match 1.

subject was always tested in the same machine. The range of motion was set from a knee angle of 90 to 20° from full extension, and the angular velocity of contraction of the isokinetic dynamometer rotating lever arm was set at $60°\cdot s^{-1}$. The subjects performed four warm-up contractions followed by three maximal contractions. Peak torque was analyzed (CV < 5%). Only one leg was tested (dominant leg). Because of the tight test time schedule, isokinetic maximal strength at 45 h and sprint performance at 51 h after the first match were not measured.

Perceived muscle soreness. Perceived muscle soreness was assessed using a seven-point Likert scale (23) designed to measure the level of muscle soreness in the lower body. The scale consisted of the following verbal anchors: 1 = very, very good; 2 = very good; 3 = good; 4 = tender but not sore; 5 = sore; 6 = very sore; and 7 = very, very sore. Scores were recorded to the nearest 0.5.

Biochemical parameters. First in the testing procedures, blood was taken from an antecubital vein into a 9-mL vacutainer tube. The blood coagulated in room temperature for 30–45 min. Serum was pipetted off and stored in Eppendorf tubes at −80°C until analysis. CK, urea, and uric acid were analyzed with standard routine measurements in a Modular P Analyzer (Hitachi, Tokyo, Japan). CV for all three variables in these measurements were < 5%. Changes in plasma volume from baseline were calculated from changes in hemoglobin concentration and hematocrit in accordance with the Dill and Costill method (7).

Diet. The food intake was standardized for all players during the whole study period. All players were given a meal plan composed by a nutritionist. The composition of the meals was developed using a national food database (Mat på data 4.3 LKH, Norway), and carbohydrate (CHO) and protein were adjusted to each player's body weight (55/60/65/70 kg, respectively) to meet the recommendations for daily recovery in players participating in moderate training (≥ 6 g body weight CHO, ≥ 1.2 g body weight protein) (20). Twenty-five percent of the total energy intake was from fat, and the meal plan included a variation of bread, cereals, milk/yogurts, meat, pasta/rice, fruit, and vegetables to ensure adequate intake of macro- and micronutrients. In addition, the players had a sports drink available during match, providing approximately 30–60 g of CHO per hour (Maxim Energy) and a CHO intake of 1 g body weight within 30 min after match to ensure optimal recovery (banana, yogurt, sports drink) (20).

Statistical analysis. Data are presented as means and standard errors. The statistical significance of the differences between groups and time points was determined using a two-way (group × time) repeated-measures ANOVA, using absolute values. Where appropriate, Dunnett *post hoc* test was applied to the data. *P* values below 0.05 were considered statistically significant.

TABLE 3. Sprint time, jump height, peak torque knee extension and flexion, perceived muscle soreness, CK, urea, and uric acid concentration for the active recovery group and passive group at baseline, immediately after match 1, immediately before match 2 (+ 69 h), and immediately after match 2 (+ 74 h).

	N	Baseline − 3 h	Immediately after Match 1 (0 h)	Before Match 2 (+ 69 h)	Immediately after Match 2 (+ 74 h)
20-m sprint (s)					
Active recovery	8	3.18 ± 0.03	3.26 ± 0.03 *	3.17 ± 0.03	3.25 ± 0.03*
Passive	9	3.17 ± 0.03	3.28 ± 0.04*	3.15 ± 0.04	3.23 ± 0.04*
Jump height (cm)					
Active recovery	8	30.5 ± 1.2	29.1 ± 1.0*	29.2 ± 1.1*	28.6 ± 1.2*
Passive	9	29.8 ± 1.2	28.4 ± 1.0*	28.9 ± 1.2*	28.4 ± 1.3*
Peak torque flexion ($°\cdot s^{-1}$)					
Active recovery	8	102 ± 5	93 ± 6*	101 ± 5	96 ± 4*
Passive	9	104 ± 5	95 ± 5*	104 ± 5	98 ± 4*
Peak torque extension ($°\cdot s^{-1}$)					
Active recovery	8	175 ± 5	165 ± 6*	170 ± 6	166 ± 5*
Passive	9	167 ± 4	154 ± 5*	160 ± 6	154 ± 7*
Muscle soreness					
Active recovery	8	2.8 ± 0.2	4.3 ± 0.1*	3.5 ± 0.1	4.8 ± 0.2*
Passive	9	3.1 ± 0.2	3.9 ± 0.2*	3.1 ± 0.2	4.6 ± 0.2*
CK ($U\cdot L^{-1}$)					
Active recovery	8	158 ± 33	344 ± 41*	211 ± 31	414 ± 57*
Passive	9	146 ± 25	327 ± 52*	157 ± 29	363 ± 65*
UREA (mM)					
Active recovery	8	5.1 ± 0.2	5.7 ± 0.2*	5.0 ± 0.4	6.0 ± 0.3*
Passive	9	5.5 ± 0.3	6.4 ± 0.4*	5.7 ± 0.4	7.1 ± 0.5*
Uric acid (mM)					
Active recovery	8	243 ± 19	270 ± 24*	244 ± 12	276 ± 13*
Passive	9	249 ± 21	274 ± 20*	266 ± 15	304 ± 16*

Values are means ± SE. * Significantly different ($P < 0.05$) from baseline values.

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RESULTS

Work intensity and fluid loss during the matches. The amount of high-intensity running averaged 1.09 ± 0.2 km in the first match and 1.11 ± 0.1 km in the second match. Heart rate values recorded during the two matches were over 160 bpm. Altogether, the levels of high-intensity running and heart rate values indicate the relative high intensity of the matches. During match 2, there was a significantly higher mean heart rate compared with match 1 for both groups (Table 2). However, there were no significant differences in mean heart rate between the groups during match 1 (163 ± 3 bpm active, 161 ± 2 bpm passive) or match 2 (171 ± 3 bpm active and 168 ± 2 bpm passive) (Table 2). The average body weight loss was 882 ± 100 g in match 1 and 947 ± 103 g in match 2. The fluid intake was 324 ± 48 mL during match 1 and 252 ± 41 mL in match 2. The calculated fluid loss was 1139 ± 473 g (1.9 ± 0.1% of BW) in match 1 and 1132 ± 122 g (2.0 ± 0.1% of BW) in match 2. There were no significant differences between the active and the passive groups in fluid loss during the two matches. We noted similar weather conditions when the two matches were played (light rain and approximately 12°C).

Neuromuscular and biochemical changes immediately after the first match. Sprint, CMJ performance, and isokinetic strength were significantly reduced immediately after the first match. There was a significant decrease in sprint performance (−3.0 ± 0.5%), CMJ (−4.4 ± 0.8%), and peak torque in knee extension (−7.1 ± 1.9%) and flexion (−9.4 ± 1.8%). In parallel, blood CK (+152 ± 28%), urea (+14.8 ± 2%), and uric acid (+10.9 ± 2%) were significantly increased at the end of the match (Table 3). Likewise, a significant increase in perceived muscle soreness was reported (Table 3).

The effects of active recovery regime. At baseline, there were no significant differences in absolute values for sprint, CMJ, isokinetic strength, blood CK, urea and uric acid, and perceived muscle soreness between the active and passive recovery groups (Table 3). At all time points, we found no significant differences between the two groups in the recovery patterns of any of the four neuromuscular and three biochemical parameters.

Time course of changes in neuromuscular and biochemical parameters after the first match. Times for the 20-m sprint returned to baseline values 5 h after the first match (Fig. 2A). CMJ was significantly lower than baseline values at all tests performed after the match (Fig. 2B). Peak torque in knee extension was significantly reduced at 5 and 21 h and returned to baseline at 27 h (Fig. 2C) after the first match. Peak torque in knee flexion was significantly lower at 5, 21, and 27 h and returned to baseline values at 51 h (Fig. 2D) after the first match. Peak CK was reached 21 h after the first match (Fig. 3B). CK returned to baseline values at 69 h. Urea and uric acid reached peak values immediately after the first match (+15 ± 2% and +11 ± 2%, respectively) and returned to

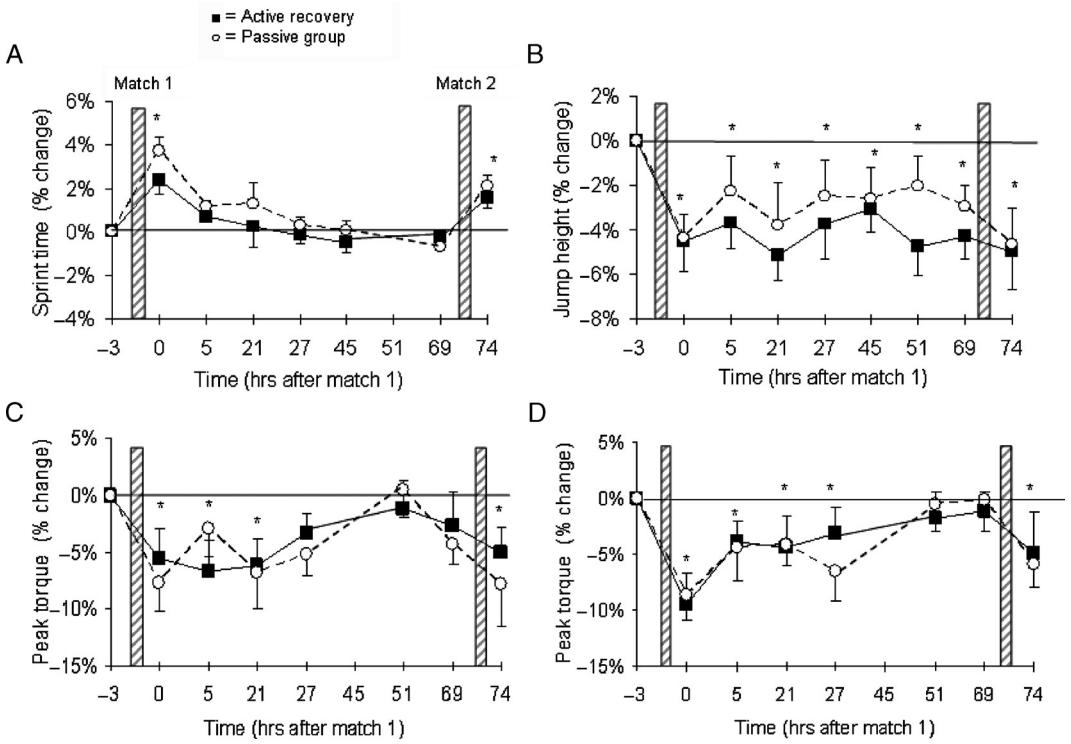

FIGURE 2—Sprint performance (*A*), jump performance (*B*), and peak torque isokinetic knee extension (*C*) and flexion (*D*) during 74 h for the active recovery group (*filled squares*) and passive group (*open circles*). Data are percent changes from baseline values. Data are means ± SE. No significant differences were observed between groups. * Significantly different from baseline ($P < 0.05$).

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baseline at 21 h (Fig. 3C and D). Perceived muscle soreness peaked at 27 h and returned to baseline at 69 h after the first match (Fig. 3A).

Neuromuscular and biochemical changes immediately after the second match. For all parameters, except for jump performance, there were no significant differences between the magnitude of the changes observed immediately after match 2 (played 72 h after the first match) compared with that observed immediately after match 1 (Table 3). The reduction in CMJ performance after the second match was less than after the first match (Table 3).

DISCUSSION

This is the first study documenting the time course of changes in neuromuscular and biochemical parameters in response to an elite female soccer match followed by 72 h of recovery and by a second match. The main findings were a significant reduction in sprint, CMJ, and isokinetic strength, accompanied by a significant rise in CK, urea and uric acid, and perceived muscle soreness scores immediately after the first match. Thereafter, the different variables recovered gradually, with a fast normalization of sprint performance, a slower normalization of peak torque knee extension and flexion, and jump performance that was not fully recovered at the start of the second match. Furthermore, the active recovery regime had no effect on the recovery pattern of the neuromuscular and biochemical parameters. In general, the magnitude of the changes in neuromuscular and biochemical parameters after the second match did not significantly differ from that observed immediately after the first match.

The mean maximal oxygen uptake, heart rate, and distance covered in high-intensity running during the first and second matches correspond well with what is reported in elite female soccer (6,17). Consequently, the results from this study can be regarded as representative for elite female soccer players. The mean heart rate differed between the first and second matches. Such small intermatch variability (~4%) could be expected between matches. Because of a number of factors, it is unlikely that the same player will exhibit an identical heart rate in two different matches. More importantly, the amount of high-intensity running, which is an important factor in soccer performance (4), did not significantly differ between the two matches. Thus, our results suggest that the players' performance during the game, expressed as the mean heart rate value and amount of high-intensity running, is not impaired when two matches are interspersed by 2 d of recovery.

All neuromuscular parameters measured in this study were significantly altered after the first match. The 3% decline in sprint and 4% decline in CMJ are of similar magnitude to what have been previously reported in male players (3,18,28). In females, Hoffman et al. (11) have reported a significant decline in CMJ power performance

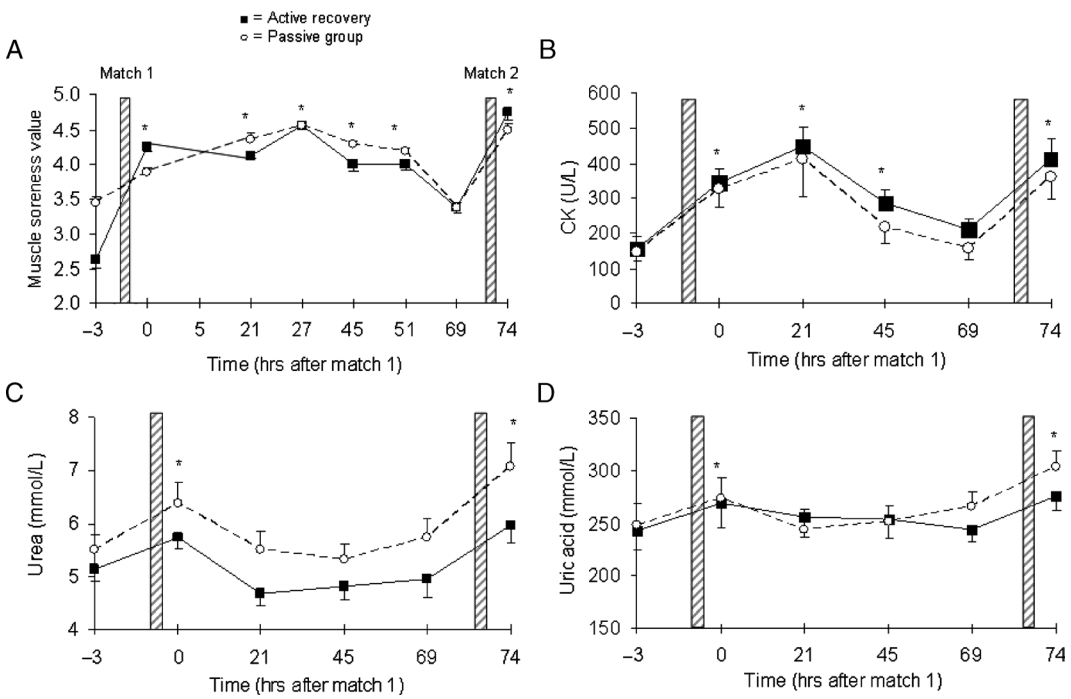

FIGURE 3—Perceived muscle soreness (*A*), creatine kinase (*B*), urea (*C*), and uric acid (*D*) during 74 h for the active recovery group (*filled squares*) and passive group (*open circles*). Data are presented as absolute values. Perceived muscle soreness was assessed using a seven-point Likert scale and contained the following anchors: 1 = very, very good; 2 = very good; 3 = good; 4 = tender but not sore; 5 = sore; 6 = very sore; and 7 = very, very sore. Data are means ± SE. No significant differences were observed between groups. * Significantly different from baseline ($P < 0.05$).

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occurring within 24 h after the match. According to Hoffman et al. (11), the decline in CMJ may have resulted from muscle damage. Additionally, the 7–9% reduction in peak torque knee extension and flexion observed immediately after the first match is in accordance with the results observed in male players immediately after a soccer match (26). Thus, the acute reductions in CMJ and sprint performance, together with the decline in isokinetic strength, indicate that different aspects of the force-generating capacity are compromised in response to a female soccer match. It can also be concluded that sprint and CMJ performance and isokinetic strength are similarly affected in male and female players.

The decline in sprint, jump, and isokinetic strength can be caused by several factors. High-force muscle actions, such as those performed during soccer, can lead to structural disruptions within myofibers, which can explain the alteration of the force-generation capacity immediately after the match (9,25). In this study, the twofold increase in blood CK observed immediately after the match indicates muscle cell damage during the match. We also observed a significant increase (~10%) in urea and uric acid immediately after the match. Similar elevations in uric acid have been shown after a male soccer match (2). Uric acid and urea are markers of enhanced nucleotide cycle turnover and the breakdown of amino acids (30). Our results suggest that the soccer match resulted in an increase in AMP levels, with a subsequent increase in the formation of ammonia (NH_3) and the enhancement of protein breakdown. In addition to changes in CK, urea, and uric acid, there was a parallel increase in perceived muscle soreness. This suggests that fatigue in active muscles after a soccer match reflects structural alterations within the muscles, enhanced nucleotide cycle turnover, and breakdown of amino acids, as well as possible alterations in the central nervous system (24).

Dehydration has also been suggested to cause decline in performance. A fluid loss of 2.7% of the total body weight has been shown to reduce 5- and 10-m sprint times (19). According to Hoffman et al. (10), a small dehydration (1–2%) can contribute to an elevation in core temperature, which increases the cardiovascular strain. In our study, we observed an approximately 1.5% fluid loss, which is similar to data reported in male players (18). However, Mohr et al. (22) report no effects on core or muscle temperature with a fluid loss of 2.2% in male players, and they conclude that fatigue at the end of the game was not related to muscle or body temperature. Several other mechanisms, such as increased inorganic phosphate, alterations in the excitation–contraction process involving alteration in the Ca^{2+} release, and low muscle pH from lactic acid accumulation, have been suggested to cause a decline in force-generating capacity (31,32). However, a weak relationship between muscle lactate concentrations, lowered muscle pH, and decline in sprint performance has been found in male players (18). In that study a selective glycogen depletion occurred in some muscle fibers in response to a soccer match, which would contribute to a decline in maximal performance (18). Another possible mechanism contributing to decreased jump performance at the end of the match would be an impairment of the stretch-shortening cycle (SSC) (1).

Our study reveals, for the first time, significant differences in the time course of recovery of the different neuromuscular and biochemical parameters. Sprint performance was the first physical capacity to return to baseline after the match (5 h after) and was followed by peak torque knee extension (27 h) and flexion (51 h), whereas CMJ did not recover throughout the remaining time points of the study. Although strong correlations have been shown between maximal strength and the performance in sprint and vertical jump (33), our results suggest differences in the recovery pattern between these physical qualities. The differences in recovery pattern can be explained by the existence of differences in the amount of muscle work and in the intermuscular coordination between sprint, jump, and isokinetic knee flexion and extension (13–15). The long-lasting reductions in jump performance and isokinetic strength indicate some slow recovering processes within the muscle. Although not studied after soccer matches, myofibrillar disruptions observed after heavy strength training and downhill running (8,9) may contribute to the long-lasting reductions in force-generating capacity. In addition, the moderate CK and urea accumulation in serum indicates alterations in muscle membranes and in protein metabolism. Urea and uric acid levels returned to baseline, whereas CMJ, CK, and muscle soreness were still significantly altered. This implies that the normalization of urea and uric acid cannot be used as indicators for CMJ recovery and for the normalization of perceived muscle soreness.

The CMJ performance in this study was still impaired, even though knee extensor and flexor strength had recovered. Rodacki et al. (29) suggest, in an experimental study, that deterioration of CMJ performance was affected by fatigue of the knee extensors but not the knee flexors. The findings in our study suggest that the recovery of CMJ performance were not solely dependent on the recovery of knee extensor and flexor muscle strength. It is suggested that there are additional mechanisms that contribute to the recovery of CMJ. As discussed above, one mechanism could be an impairment of the SSC component (1).

Low-intensity training did not significantly affect the time course of recovery for the four neuromuscular or the three biochemical parameters. The training regime used in this study is widely used by teams in many countries, and it is believed to accelerate recovery after a soccer match. However, it has never been scientifically evaluated in soccer. Our results clearly show that the time course of neuromuscular and some biochemical changes were not significantly improved by the active recovery regime. Nonetheless, it is possible that the evaluation of parameters other than those used in this study might have proved to be

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more sensitive in detecting differences between groups. For example, it has been shown that CMJ peak force and peak power revealed differences between starters and nonstarters after a game (11,12). Also, it has been proposed that myoglobin is a more sensitive measure of muscle damage than CK (12). However, using the parameters evaluated in our study, we were able to detect differences over time, which indicates that these measurements seem sensitive enough to detect fatigue. It is known that restoration of energy stores is vital for proper recovery (27). Because diet in our study was standardized to meet each player's individual needs for CHO and protein, it is likely that the energy restoration was similar between the two groups. Furthermore, in our study, the anaerobic threshold was not determined. It can be speculated that the effect of the active recovery is influenced by the exercise intensity performed relative to the anaerobic threshold. Additionally, and more importantly, the work intensity and performance during the second match were not affected by an active or passive recovery strategy. However, we cannot rule out the possibility that the active recovery regime affects aspects of the performance or biochemical markers other than those measured in our study. Our results are in favor of the conclusion from a recent review by Barnett (5) suggesting a lack of evidence supporting the use of active recovery strategies. Nevertheless, it should be emphasized that low-intensity training did not have any detrimental effects on recovery. Consequently, performing low-intensity exercise between matches could be important for the physical fitness and performance of the players throughout a tournament lasting up to 3 wk.

CONCLUSION

In conclusion, the present study clearly demonstrates the existence of differences in the recovery pattern of the various neuromuscular and some biochemical parameters in response to a female soccer match. This study also shows that the time course of recovery of the neuromuscular and biochemical parameters is not affected by active recovery. Finally, the performance of players during the game, expressed as mean heart rate value and amount of high-intensity running, is not impaired when two matches are interspersed by 2 d of active or passive recovery.

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REFERENCES

1. Avela J, Komi PV. Interaction between muscle stiffness and stretch reflex sensitivity after long-term strecth-shortening cycle exercise. *Muscle Nerve* 1998;21:1224–7.
2. Bangsbo J. Energy demands in competitive soccer. *J. Sports Sci*. 1994;12:S5–12.
3. Bangsbo J. The physiology of soccer: with special reference to intense intermittent exercise. *Acta Physiol Scand*. 1994;15(suppl): 1–156.
4. Bangsbo J, Mohr M, Krustrup P. Physical and metabolic demands of training and match-play in the elite football player. *J Sports Sci*. 2006;24:665–74.
5. Barnett A. Using recovery modalities between training sessions in elite athletes: does it help? *Sports Med*. 2006;36:781–96.
6. Davis JA, Brewer J. Applied physiology of female soccer players. *Sports Med*. 1993;16:180–9.
7. Dill D, Costill D. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol*. 1974;37:247–8.
8. Fridén J, Sjöström M, Ekblom B. A morphological study of delayed muscle soreness. *Experientia* 1981;37:506–7.
9. Gibala MJ, MacDougall JD, Tarnopolsky MA, Stauber WT, Elorriaga A. Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. *J Appl Physiol*. 1995;78:702–8.
10. Hoffman J, Maresh CM, Armstrong LE, et al. Effects of hydration state on plasma testosterone, cortisol and concentrations before and during mild exercise at elevated temperature. *Eur J Appl Physiol Occup Physiol*. 1994;69:294–300.
11. Hoffman J, Nusse V, Kang J. The effect of an intercollegiate soccer game on maximal power performance. *Can J Appl Physiol*. 2003;28:807–17.
12. Hoffman JR, Maresh CM, Newton RU, et al. Performance, biochemical, and endocrine changes during a competitive football game. *Med Sci Sports Exerc*. 2002;34(11):1845–53.
13. Iossifidou A, Baltzopoulos V, Giakas G. Isokinetic knee extension and vertical jumping: are they related? *J Sports Sci*. 2005;23:1121–7.
14. Jacobs R, Bobbert MF, van Ingen Schenau GJ. Mechanical output from individual muscles during explosive leg extensions: the role of biarticular muscles. *J Biomech*. 1996;29:513–23.
15. Jacobs R, van Ingen Schenau GJ. Intermuscular coordination in a sprint push-off. *J Biomech*. 1992;25:953–65.
16. Krustrup P, Mohr M, Amstrup T, et al. The yo-yo intermittent recovery test: physiological responses, reliability, and validity. *Med Sci Sports Exerc*. 2003;35(4):697–705.
17. Krustrup P, Mohr M, Ellingsgaard H, Bangsbo J. Physical demands during an elite female soccer game: importance of training status. *Med Sci Sports Exerc*. 2005;37(7):1242–8.
18. Krustrup P, Mohr M, Steensberg A, Bencke J, Kjaer M, Bangsbo J. Muscle and blood metabolites during a soccer game: implications for sprint performance. *Med Sci Sports Exerc*. 2006;38(6): 1165–74.
19. Magal M, Webster MJ, Sistrunk LE, Whitehead MT, Evans RK, Boyd JC. Comparison of glycerol and water hydration regimens on tennis-related performance. *Med Sci Sports Exerc*. 2003;35(1): 150–6.
20. Maughan R, Burke L, Coyle E, editors. *Food, Nutrition and Sports Performance II. The International Olympic Committee Consensus on Sports Nutrition*. New York (NY): Routledge; 2004.
21. Mohr M, Krustrup P, Bangsbo J. Match performance of high-standard soccer players with special reference to development of fatigue. *J Sports Sci*. 2003;21:519–28.
22. Mohr M, Krustrup P, Nybo L, Nielsen JJ, Bangsbo J. Muscle temperature and sprint performance during soccer matches—beneficial effect of re-warm-up at half-time. *Scand J Med Sci Sports*. 2004;14:156–62.
23. Morgan WP, Costill DL, Flynn MG, Raglin JS, O'Connor PJ. Mood disturbance following increased training in swimmers. *Med Sci Sports Exerc*. 1988;20(4):408–14.

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24. Proske U, Allen T. Damage to skeletal muscle from eccentric exercise. *Exerc Sport Sci Rev*. 2005;33(2):98–104.
25. Raastad T, Hallén J. Recovery of skeletal muscle contractility after high- and moderate-intensity strength exercise. *Eur J Appl Physiol*. 2000;82:206–14.
26. Raastad T, Theting T, Ingjer F, Hallén J. Neuromuscular fatigue and recovery after soccer matches in elite soccer players. In: *7th Annual Congress of the European College of Sport Science*. Athens, Greece: Pashalidis Medical Publisher. Book of Abstracts. 2002;2: p. 825.
27. Reilly T, Ekblom B. The use of recovery methods post-exercise. *J Sports Sci*. 2005;23:619–27.
28. Reilly T, Rigby M. Effect of an active warm down following competitive soccer. In: Spinks T, Reilly A, editors. *Science and Football IV W*. London (UK): Routledge; 1999. p. 226–9.
29. Rodacki ALF, Fowler NE, Bennett SJ. Vertical jump coordination: fatigue effects. *Med Sci Sports Exerc*. 2002;34(1):105–16.
30. Viru A, Viru M. *Biochemical Monitoring of Sport Training*. Champaign (IL): Human Kinetics; 2001. p. 37–9.
31. Warren G, Ingalls C, Lowe D, Armstrong R. Excitation-contraction uncoupling: major role in contraction-induced muscle injury. *Exerc Sport Sci Rev*. 2001;29(2):82–7.
32. Westerblad H, David A, Lännergren J. Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News Physiol Sci*. 2002; 17:17–21.
33. Wisløff U, Castagna C, Helgerud J, Jones R, Hoff J. Strong correlation of maximal strength with sprint performance and vertical jump height in elite soccer players. *Br J Sports Med*. 2004;38: 285–8.

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