Muscle Damage, Endocrine, and Immune Marker Response to a Soccer Match

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Abstract

Thorpe, R and Sunderland, C. Muscle damage, endocrine, and immune marker response to a soccer match. *J Strength Cond Res* 26(10): 2783–2790, 2012—This study represents the first time that muscle damage, endocrine, and immune markers have been measured, together with activity profile, during a competitive soccer match. Seven semiprofessional soccer players participated in a competitive league match. Blood and saliva samples were obtained 1 hour before kick off and immediately postmatch. Global positioning system equipment was used to measure heart rate and activity profile data throughout the match. Percentage increase in creatine kinase (CK) and myoglobin (MYO) concentrations was correlated with the number of sprints performed during the match ($r = 0.88$, $p = 0.019$; $r = 0.75$, $p = 0.047$, respectively). Creatine kinase increased by 84% ($p = 0.17$) from prematch to postmatch, whereas MYO increased by 238% ($p = 0.05$). Players performed 39 ± 18 sprints during the course of the match. Cortisol increased by 78% ($p = 0.103$), whereas testosterone increased significantly by 44% ($p = 0.004$). No differences were seen from prematch to postmatch in the testosterone to cortisol ratio, immunoglobulin (Ig) A, IgM, or IgG. Sprinting is correlated with changes in CK and MYO and may therefore be associated with muscle damage in semiprofessional soccer players.

Key Words creatine kinase, immunoglobulin, football, testosterone, cortisol, global positioning system

Introduction

Modern day elite soccer involves a high number of competitive matches over the course of a 9-month season; moreover, with the introduction of cup competitions, it is not unusual for a team to play 2 matches in the space of a week. The physiological demands placed upon players are substantial. During a typical match, elite soccer players have been known to reach mean and peak heart rates of 85 and 98% of their maximal values, respectively, covering 2.4 km at high intensity of which 0.6 km was covered by sprinting (34). The game's high-intensity demands, and potential perturbations to the muscular, endocrine, and immune systems, mean that recovery strategies postmatch and training are essential in preparation for the next match (40).

Soccer is considered to be a high-intensity intermittent sport consisting of a large number of eccentric muscle contractions (23). Researchers have investigated the effects of this exercise on the musculature by monitoring creatine kinase (CK) and myoglobin (MYO), reliable markers for muscle damage (3), but have not measured the physical demand or activity profile of the players. Creatine kinase and MYO have been used extensively in high-impact sports such as rugby and American football to assess the degree of muscle damage (18,21,43), and been found to increase significantly from prematch/training to postmatch/training. The reasons for this increase have always been attributed to the effect of impact blows; however, the cause of muscle damage in soccer is unclear.

Although the metabolic and physical demands of soccer are well documented, there are mixed results for the effects of a soccer match on the endocrine and immune systems. Cortisol and testosterone are endocrine markers, the first of which has been known to react either preceding or during stressful situations as a homeostatic response of the body (11). Together, these hormones identify anabolic and catabolic relations, the testosterone to cortisol ratio. A decreased ratio has been correlated with tiredness, lethargy, exhaustion, and even negative performance (1). Researchers who have investigated how the levels of these hormones vary over the course of a soccer season have found that they are generally affected by intensity and duration of exercise: preseason and the final 9 weeks of the season (16,19,26). A single soccer match shows anticipatory rises before performance and then increases in cortisol during match play, with equivocal findings for testosterone; however, researchers have not calculated testosterone to cortisol ratio over the course of a match (7,14,20,23,29,36,37). Similar intermittent sports such as American football, rugby, and Australian rules football have shown comparable results (10,15,21,28,32).

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Table 1. Mean and ± *SD* percentage of match time spent and distance covered in the zones.

	Walking	Jogging	Low speed	Moderate speed	Fast speed	Sprinting
% Total time	68.89 ± 14.7	8.16 ± 3.1	15.66 ± 2.9	7.15 ± 1.5	3.22 ± 1.1	2.69 ± 1.0
Distance (m)	2,978 ± 1,075	718 ± 456	1,787 ± 647	1,064 ± 369	566 ± 234	617 ± 330

This study will be the first to measure testosterone and the testosterone to cortisol ratio alongside the activity pattern of the players to ascertain the activities that may alter these endocrine responses.

The secretory immune system of the mucosal tissues, such as the upper respiratory tract, is known to be the first barrier to colonization by microorganisms. The predominant immunoglobulin in mucosal fluid, immunoglobulin (Ig) A, has been found to be capable of neutralizing pathogens, viruses, and toxins (44). The response of IgA to exercise has been studied extensively and has generally followed trend with the dual effect of exercise. Immunoglobulin G and IgM also provide defenses against pathogens and have rarely been studied in response to exercise and stressors (30). There is a distinct lack of research regarding soccer and immunoglobulins, although Sari-Sarraf et al. (41) found increased levels of IgA after a simulated soccer trial. No significant change in immunoglobulin concentrations was found in rugby players after a competitive match, although there was a large interindividual variation (24). In addition, IgA has even been shown to affect the levels of salivary cortisol (22). Clearly, further research is required into the response of immune markers and hormones to a soccer match.

To fully understand an athlete's condition and thus prescribe appropriate recovery, it is important to know the way in which muscle damage and immune markers respond (40), and how anabolic and catabolic relationships interact after exercise (4). Understanding the causes of muscle damage, immune responses, and anabolic and catabolic relationships is fundamental to allow coaches and sport scientists to recommend relevant and effective training and recovery strategies. To address this issue for the first time in soccer, this study has used global positioning system (GPS) technology to measure variables such as total distance covered, high-intensity running and sprint distance, sprint number, and heart rate response. The potential relationships between match movements and muscle damage, endocrine, and immune responses may elucidate the causes of these responses and allow changes to be made to both training and recovery to augment performance.

This study involved a semiprofessional competitive league match. Moreover, for the first time at a competitive level, GPS equipment was used to collect match intensity data. The study investigated the association of muscle damage, endocrine, and immune responses with match intensity. We hypothesize that a soccer match will result in skeletal muscle damage demonstrated by increases in CK and MYO, an increase in cortisol, and no change in immunoglobulins.

Methods

Experimental Approach to the Problem

Before and after a competitive football match, plasma and saliva samples were collected for the assessment of muscle damage, immune function, and endocrine response. The dependent variables plasma CK activity and MYO changes were monitored to reflect muscle damage and IgA, IgG, and IgM to assess acute changes in immune function. The dependent variables salivary cortisol and testosterone were measured to assess the catabolic and anabolic responses. The activity profile of the players, the independent variable, was monitored using a GPS. Correlations between changes in the dependent variables CK, MYO, IgA, IgG, IgM, cortisol, and testosterone and the activity profile, which includes distances and percentage time in specific-speed zones, sprint number, and mean sprint distance, may elucidate potential mechanisms for these

Table 2. Mean ± *SD* (range) creatine kinase, myoglobin, and immunoglobulin (Ig) A, IgG, and IgM prematch and postmatch.

	Creatine kinase ($U \cdot L^{-1}$)	Myoglobin ($ng \cdot ml^{-1}$)	IgA ($g \cdot L^{-1}$)	IgG ($g \cdot L^{-1}$)	IgM ($g \cdot L^{-1}$)
Prematch	345 ± 360	57.8 ± 59.5	2.5 ± 0.7	12.5 ± 2.1	1.3 ± 1.0
(range)	(110–1,067)	(29.3–192.4)	(1.4–3.5)	(9.2–15.5)	(0.5–3.2)
Postmatch	668 ± 878	202.9 ± 219.6*	2.3 ± 0.6	12.8 ± 2.0	1.3 ± 1.0
(range)	(186–2,624)	(70.2–691.3)	(1.3–3.3)	(10.2–15.8)	(0.5–3.3)

*$p = 0.05$ from prematch.

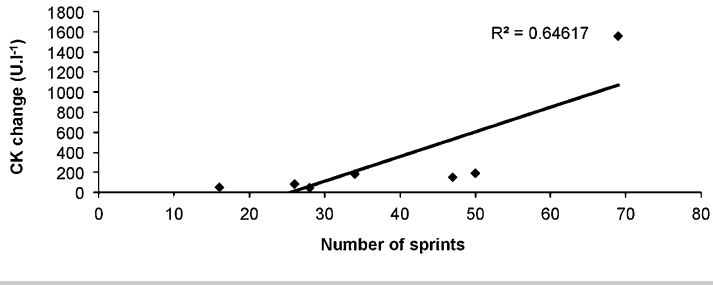

Figure 1. Correlation between number of sprints performed and change in creatine kinase ($r = 0.80$, $p = 0.029$).

responses. Providing information relating the activity profile of players to muscle damage, immunity, and endocrine responses will help with the development of appropriate player recovery strategies and prematch training.

Subjects

Seven injury-free, semiprofessional male soccer players from the same team volunteered to participate in the study. Their mean age, mass, and height were 25 ± 6 years, 75.3 ± 4.6 kg, and 179 ± 6 cm, respectively. All participants (3 defenders, 2 midfielders, and 2 forwards) competed in the entire match. Players who played <90 minutes were not included, although the data were recorded. They reported no significant oral, dental, or other symptoms of infection and were not taking any medication in the month before the match. The study was approved by Nottingham Trent University ethical committee. Participants were notified of the requirements of the research and of any risks before giving their written and verbal informed consent.

Procedures

The semiprofessional players trained 2 times a week, consisting of 4 hours per week plus a 1.5-hour match. Participants continued their training routine but did not partake in competitive match play in the 7 days before the experimental match (15:00 start, 15° C, 51% relative humidity) and refrained from strenuous exercise during the 24 hours before the match. Players were asked to maintain their normal prematch diet and refrain from alcohol for 48 hours before the match. In the 2 hours before the match, players were asked to consume water only (500 ml) to ensure adequate hydration and avoid variations on saliva secretion (38). Before warm-up, body mass (Seca 877; Seca, Birmingham, United Kingdom) was recorded wearing dry underwear. After the completion of the match, players towel-dried and recorded a postexercise body mass (wearing dry underwear), from which we calculated sweat loss and percentage body mass loss, taking into account voluntary water consumption (Cookworks, London, United Kingdom). Samples of saliva and blood were collected at 2 time points: 1 hour before the match before the warm-up (1) and the other immediately postmatch (2). During the match, heart rate and activity profile of the players were monitored via GPS.

Activity Profile

Player on-field activities were recorded by a single GPS unit per player, for the duration of the game (SPI Elite 1 Hz; GPSports, Fyshwick, Australia). The GPS unit is integrated with a 100-Hz accelerometer to improve speed and distance accuracy. The same unit was used for each player throughout and the system used has previously been validated specifically for team sport match movements at latitudes employed in the present study (mean difference ± 95% limits of agreement: 0.0 ± 0.9 km·h^{-1} and 2.5 ± 15.8 m) (8,31), including high-speed running and sprinting (mean difference ± 95% limits of agreement: 0.2 ± 1.2 km·h^{-1}) (31). Heart rate was also measured using heart rate monitors (Polar Electro, Oy, Kempele, Finland) and stored on the GPS device. The activity categories used during the match included walking (0–6 km·h^{-1}), jogging (6–8 km·h^{-1}), low-speed running (8–12 km·h^{-1}), moderate-speed running (12–15 km·h^{-1}), fast-speed running (15–18 km·h^{-1}), and sprinting (>18 km·h^{-1}). High-intensity activity was defined as >15 km·h^{-1} and thus incorporated fast-speed running and sprinting. The total number of sprints is the number of times players achieved a speed >18 km·h^{-1} during the match. Mean sprint distance was also recorded.

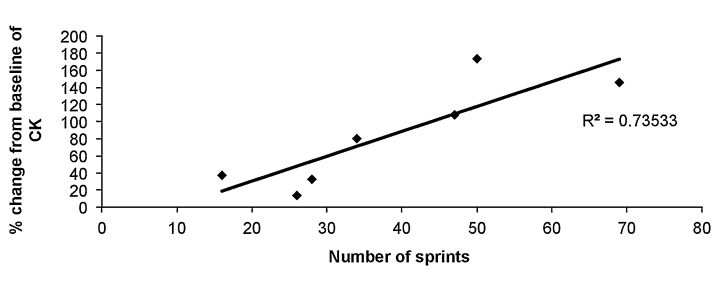

Figure 2. Correlation between number of sprints performed and percentage change in creatine kinase ($r = 0.86$, $p = 0.014$).

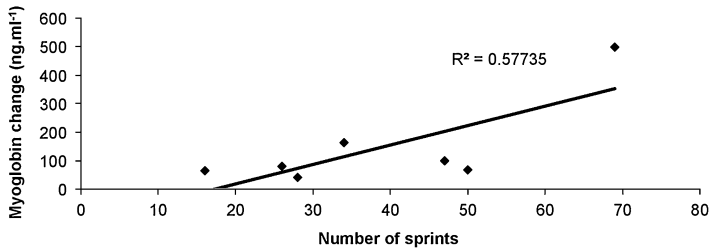

Figure 3. Correlation between change in myoglobin and number of sprints performed ($r = 0.76$, $p = 0.047$).

Saliva Collection and Analysis

Each participant was asked to rinse out his mouth with distilled water to prevent potential sample contamination that might affect sample measurement. Whole unstimulated saliva was collected by expectoration for 5 minutes into sterile preweighed plastic containers (Sarstedt, Nümbrecht, Germany). All saliva samples were put on ice before freezing at −80° C until analysis. Cortisol and testosterone concentrations were measured by enzyme-linked immunosorbent assay (salivary cortisol/testosterone EIA; Salimetrics, State College, PA, USA). The intra-assay variance was 3.7% and the sensitivity of the assay 0.0828 nmol·L^{-1} for cortisol. The intra-assay variance for testosterone was 5.6% and the sensitivity of the assay 0.0035 nmol·L^{-1}.

Blood Collection and Analysis

Samples were obtained using the standard finger prick method, guiding the blood into a microcuvette (Microvette CB 300 FH; Sarstedt). Samples were then put on ice until being centrifuged for 12 minutes at 5,000*g* (Micro Centaur; MSE, London, United Kingdom) and the supernatant was stored (Microtube 0.5 ml; Sarstedt) at −80° C before analysis. Aliquots of plasma were analyzed to determine the concentration of CK (A11A01632), MYO (A11A01904), and immunoglobulin isotypes IgA (A11A01683), IgG (A11A01684), and IgM (A11A01685; all Horiba, Montpellier, France) using an automated clinical chemistry analyzer (ABX Pentra 400; Horiba, Northampton, United Kingdom). For CK and MYO, the intra-assay variances were 1.5 and 1.6%, respectively. For IgA, IgG, and IgM, the intra-assay variances were 3.0, 2.0, and 1.9%, respectively.

Statistical Analyses

Results are reported as mean ± *SD*. Student's paired *t*-tests were used to compare responses from prematch with postmatch for the endocrine, immune, and muscle damage responses measured in saliva and plasma. Pearson correlations were performed between the dependent variables plasma CK, MYO, IgA, IgG, IgM, salivary cortisol, and testosterone and the independent variables percentage time and distance covered in intensity zones, sprint number, and mean sprint distance. Outliers were determined by producing a box-and-whisker plot and were defined as an observation whose distance from the edge of the box is >1.5 times the length of the box (5). All statistics and box-and-whisker plots were computed using SPSS Base 15.0 software (SPSS, Inc., Chicago, IL, USA) and the significance level for all tests was set at $p \leq 0.05$.

Results

Activity Profile

Table 1 presents the distance covered and the percentage time spent in each of the zones. Total distance covered in the match was 9,742 ± 1,025 m and players performed 6 ± 2% of the game time at high intensity. Players performed 39 ± 18 sprints during the 90 minutes covering a mean distance of 15.7 ± 2.3 m per sprint. The peak number of sprints (69) was performed by a left midfield player. The mean heart rate during the match play was 165 ± 10 b·min^{-1} and the peak heart rate 192 ± 8 b·min^{-1}.

Plasma and Saliva

Table 2 illustrates the change in the blood parameters after the match. Myoglobin increased by 238 ± 79% ($p = 0.05$) and CK by 84 ± 61%. Despite CK increasing in all players, this was not significant because of the large variation in values ($p = 0.17$). Immunoglobulin A, IgG, and IgM did not change after the match (IgA: $p = 0.36$; IgG: $p = 0.37$; IgM: $p = 0.32$).

Table 3. Mean ± *SD* (range) cortisol, testosterone, and testosterone to cortisol ratio (T:C) prematch and postmatch.

	Cortisol (nmol·L^{-1})	Testosterone (nmol·L^{-1})	T:C
Prematch (range)	10.09 ± 4.04 (6.70–16.08)	0.26 ± 0.07 (0.19–0.40)	0.028 ± 0.008 (0.018–0.039)
Postmatch (range)	14.88 ± 5.98 (10.70–27.70)	0.37 ± 0.10* (0.23–0.50)	0.027 ± 0.009 (0.018–0.043)

*$p = 0.008$ from prematch.

Increase in CK concentration was correlated with sprint number ($r = 0.80$, $p = 0.029$; Figure 1) and sprint distance ($r = 0.78$, $p = 0.039$). Similarly, percentage increase in CK was correlated with sprint number ($r = 0.86$, $p = 0.014$; Figure 2), sprint distance ($r = 0.89$, $p = 0.007$), and high-intensity distance covered ($r = 0.92$, $p = 0.004$). One of the players had high baseline CK concentrations (1,067 $U \cdot L^{-1}$); thus, correlations were also run with this outlier removed. Increase in CK concentration remained significantly correlated with sprint number ($r = 0.82$, $p = 0.045$), and percentage increase in CK was correlated with sprint number ($r = 0.88$, $p = 0.20$), sprint distance ($r = 0.92$, $p = 0.009$), and high-intensity distance covered ($r = 0.93$, $p = 0.008$).

Figure 3 shows that increase in MYO concentration was correlated with number of sprints performed ($r = 0.76$, $p = 0.047$), although sprint distance was not significantly correlated ($r = 0.71$, $p = 0.07$). The same player, who was an outlier, was removed and the correlation between number of sprints and change in MYO was no longer evident ($r = 0.21$, $p = 0.69$). Percentage change in MYO was not correlated with number of sprints ($r = 0.17$, $p = 0.71$) and no other correlations were evident for blood and saliva parameters with match activity.

Table 3 presents the salivary cortisol and testosterone concentrations and the testosterone to cortisol ratio. Testosterone increased by 44% from prematch to postmatch ($p = 0.008$); there was no change in either cortisol ($p = 0.20$) or testosterone to cortisol ratio ($p = 0.83$).

Body Mass Loss and Fluid Intake

Mean body mass did not differ from prematch (75.3 ± 4.6 kg) to postmatch (74.9 ± 4.6 kg, $p = 0.132$), with a decrease of 0.4 ± 0.6%. Fluid intake was 0.48 ± 0.25 L during the match, with a sweat loss of 0.8 ± 0.3 L.

Discussion

The main finding of this study is the significant correlation between CK, a marker of muscle damage, and the number of sprints performed during a soccer match. Furthermore, percentage increase in CK was also correlated with sprint and high-intensity distance covered. No correlations were found between either CK or MYO and distance covered throughout the match. This suggests that sprinting and high-intensity running may be associated with muscle damage in semiprofessional soccer players. Creatine kinase concentration increased in all players (mean increase 74%) from prematch to postmatch, whereas MYO increased by 238%. These large increases from prematch to postmatch are in agreement with other studies observing muscle damage changes over the course of a soccer match (3,23); however, these researchers did not gather match performance data to indicate the reason for the muscle damage observed. Salivary testosterone increased from prematch to postmatch, but there were no changes in salivary cortisol or plasma IgA, IgG, or IgM levels.

This study showed an increase in CK and MYO concentrations, a finding that is reported in a number of high-contact sports such as American football and rugby. These studies have demonstrated CK and MYO concentration ranging from 200 to 450 $U \cdot L^{-1}$ and 50 to 300 $ng \cdot ml^{-1}$, respectively (21,28,33,42,43), values similar to those recorded in the semiprofessional soccer players of the present study. However, Gill et al. (18) found increased levels of CK up to a sizable 1,023 $U \cdot L^{-1}$ after a rugby match and suggested that contact blows could be the limiting factor in muscle damage. The large-scale increase seen in their research could be because of a number of factors; for example, they measured interstitial CK, which may exhibit more elevated levels than plasma CK. As a result of muscle damage, CK leaks from the damaged cells through the muscle membrane into the interstitial fluid before entering the circulation via the lymphatic system. It is possible that CK in plasma is at a lower concentration than that of interstitial fluid because of processes (metabolic and otherwise) occurring between the interstitial fluid and the circulation (6). Furthermore, Gill et al. (18) only reported peak CK activity, which only highlights the response of a single player to match participation (33). Soccer consists of high-intensity intermittent exercise involving rapid acceleration and deceleration movements. During the landing phase of the action of running, hamstrings are activated eccentrically to slow up hip flexion and knee extension, consequently decelerating the body. Eccentric activation produces higher tension per cross-sectional area of active muscle mass compared with concentric actions (2), which can result in significant structural muscle damage (disruption of structural proteins such as Z-lines, troponin, and tropomyosin) (9), which is a key contributor to increased CK after exercise (6). This is evidenced in the current study by the strong correlation between sprinting and high-intensity running and CK and MYO change during a match. Therefore, players who complete more high-intensity running and sprinting may require longer recovery time and should complete more match-specific training incorporating high-speed running with directional changes, accelerations, and decelerations to limit this muscle damage.

In this study, cortisol increased by 78% from prematch to postmatch in semiprofessional footballers, although this was not significant ($p = 0.1$) because of large interindividual and intraindividual variation (36). The finding that cortisol increased after the match is in agreement with many other soccer studies (7,14,20,23,29,36,37), the explanation of which is likely to be multifaceted. The increase in cortisol level could be explained by the extensive muscle damage because of cortisol's anti-inflammatory properties (17) or the combination of physical and psychological factors could have produced the observed elevations in salivary cortisol. Cortisol increases after exercise greater than 60% maximal power and longer than 30 minutes in duration (47), the response being proportional to the intensity and duration of the exercise

(45). The adrenal response is also greater for intermittent versus continuous exercise (46). A beneficial effect of the intermittent exercise-related increase in cortisol relates to the permissive role of this hormone in maintaining blood glucose and blood pressure (14). However, not only physical stress causes an increase in cortisol, indeed, Haneishi et al. (20). Found larger increases in cortisol after a competitive match compared with a practice session for female collegiate soccer players and Doan et al. (13) found increases after 36 holes of competitive golf. In a study attempting to eliminate psychological influences, Sari-Sarraf et al. (41) observed no difference between precortisol and postcortisol levels during laboratory-simulated intermittent soccer-specific exercise. It is therefore proposed that the increased cortisol response in the present study relates to the psychological aspects of competitive match play, prolonged duration of exercise, and intermittent nature of soccer. Cortisol, as the main catabolic hormone, should be monitored on a player-by-player basis because of the large individual variability shown in the present study and previous research (38).

Testosterone concentrations in the present study were significantly elevated (44%) after the match in semiprofessional soccer players. Although testosterone, the major anabolic marker for protein signaling, has been seen to increase during exercise and rugby league (33), the majority of researchers have not seen an increase during a soccer match (7,20,23,29,37). As an exception, Edwards et al. (14) did find increases in testosterone and cortisol in men from prematch to postmatch in collegiate soccer players. This increase in testosterone in the present study could be because of a combination of factors including hemoconcentration, a decrease in metabolic clearance rate, and an increase in secretion (14). Exercise-induced increases in testosterone are more prevalent in strength training than in prolonged aerobic endurance-type exercise (25,27). The present study has shown increases in testosterone more akin to those observed in strength exercise (25,27). In the current study, players performed up to 69 sprints in 90 minutes of intermittent-type exercise. This, linked with the substantial muscle damage, might have provided a stimulus for the testosterone increases by mechanisms more associated with strength exercise (25,27,39) than aerobic exercise. Clearly, the increase in testosterone in this study warrants further investigation.

There was no change in the testosterone to cortisol ratio from prematch to postmatch. Similarly, Edwards et al. (14) found concomitant increases in cortisol with testosterone. One reason why testosterone and cortisol increase in parallel during exercise may be because at least some of the increase in testosterone is derived from the conversion of increased levels of dehydroepiandrosterone (DHEA) and DHEA-S whose adrenal secretion is stimulated by the same adrenocorticotropic hormone that stimulates the secretion of cortisol (14). Measurements over a longer recovery period may have showed changes but the findings do suggest that during soccer, the anabolic-catabolic profile remains unchanged.

There was large interindividual variation observed in IgA, IgG, and IgM concentrations, with overall no change in the group mean response. Immunoglobulin concentration variation was also reported for top-level basketball players during a 17-day international championship. Plasma IgA demonstrated the largest range response, with some players showing increases of up to 28% above prelevels and others displaying decreases of up to 64% (35). Thus, there appears to be no team acute change to immune function after a soccer match, but the variation between players does suggest that individual player monitoring may be useful for some players to assess changes in immune status.

In the current study, prematch to postmatch muscle damage/endocrine responses were comparable with those of previous research that studied soccer and other intermittent sports (15,23,28). The time course changes observed by these investigations would suggest that the players in the present study would still be in a state of recovery for 72 hours postmatch. Based on previous research findings, the match intensity experienced by the players in this study, in terms of high-intensity exercise, would seem slightly less than "top-class" Danish players but comparable with "moderate" professional Danish footballers and Spanish Premier League players (12,34). Therefore, the findings would seem relevant to many professional players.

In summary, significant correlations between muscle damage markers and the number of sprints performed in a competitive match was seen in semiprofessional soccer players. It appears that the extensive muscle damage in this study originates predominantly from the high number of sprints performed and the distance covered by high-intensity running and sprinting, and not from the longevity of match participation or total distance covered by the players. There is a need for future research to ascertain the exact causes of muscle damage in similar sports and physical exercise.

Practical Applications

This study is the first to relate match activity profiles with markers of muscle damage, endocrine response, and immune function during competitive soccer match play. Our findings demonstrate that increase in CK is correlated with sprint distance, sprint number, and high-intensity distance and in MYO with sprint number. There was an increase in both cortisol, though this was nonsignificant, and testosterone in response to the match. Acute immune response was unaffected by competitive match play. Recovery strategies and training should be tailored to the individual player demands, particularly high-intensity activity completed, to reduce muscle damage and recovery time required, and improve future performances. It is clear from the findings of this study and from previous research that endocrine and immune responses are highly dependent upon the individual and vary greatly between athletes within a team. Individual player monitoring would be necessary to determine the endo-immune status and

skeletal muscle damage to develop specific recovery strategies.

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