
PHYSIOLOGICAL AND MECHANICAL RESPONSE TO SOCCER-SPECIFIC INTERMITTENT ACTIVITY AND STEADY-STATE ACTIVITY

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The aim of this study was to quantify response to a soccer-specific intermittent (INT) treadmill protocol based on notational analysis of match-play. Ten male semiprofessional football players (age 24.7 ± 4.4 yr, body mass 77.1 ± 8.3 kg, VO_{2max} 63.0 ± 4.8 ml·kg·min⁻¹) completed the 90 minute INT protocol and a steady-state (SS) protocol eliciting the same distance covered. Physiological (heart rate [HR], ratings of perceived exertion [RPE], blood lactate concentration, salivary cortisol concentration) and mechanical (electromyography [EMG] of biceps femoris and rectus femoris) responses were obtained at 15 minute intervals throughout each protocol. The physiological and mechanical responses were typically greater during the INT protocol than during the SS protocol, tending to increase as a function of exercise duration. The INT activity profile induces cumulative mechanical load on the musculoskeletal system. The increased incidence of injury toward the latter stages of match-play is attributed to compromised movement mechanics, rather than physiological strain.

Keywords: football, running, treadmill, protocol

INTRODUCTION

Professional football is associated with a high injury risk. A prospective epidemiological study of injuries in professional football clubs quantified

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that footballers suffered 710 injuries per 100,000 hours of training and match-play (Hawkins and Fuller 1999), 1000 times higher than for high-risk industrial occupations. Drawer and Fuller (2002) stated that the injury risk in professional football is unacceptable when evaluated against work-based criteria used by the Health and Safety Executive.

In working toward a process of injury prevention the Football Association of England and Wales conducted an epidemiological study of the injuries sustained in professional football over two seasons (Hawkins et al. 2001). In addition to quantifying injury incidence, the audit also provided data regarding primary injury types and sites, and the temporal distribution of injury incidence over both the course of a season and the duration of a competitive game. Hawkins et al. (2001) reported a significant main effect for time in injury risk during match-play, more injuries being sustained in the latter stages of each half, attributed in part to fatigue.

Football is a self-paced activity, characterised by an irregular and intermittent activity profile. Reilly (1990) suggested that the lack of experimental control, and subsequently the lack of experimental models, had discouraged sport and exercise scientists from examining football match-play. More recently both field-based (Nicholas, Nuttall, and Williams, 2000) and laboratory-based (Drust, Reilly, and Cable 2000) protocols have been developed in an attempt to replicate the activity profile of football match-play, but such protocols typically are based on the evaluation of physiological load. Whilst these studies make reference to the intermittent nature of game-play, arguably they fail to replicate this INT profile. In the present study it is suggested that a simulation of the activity profile of match-play should be subjected to the same guidelines used in biomechanical simulation modelling, that is, that input parameters are based on real data, in addition to evaluating output parameters against real data (Yeadon and Challis 1994). Arguably the primary limitation of simulation is the difficulty of validating the model (Vaughan 1984). Whilst the output (physiological response) from previous studies has been evaluated against data obtained during actual match-play, the findings lack validity unless the structure of the model is also subjected to evaluation against real data. Notational analyses of football match-play (Bangsbo 1994; Reilly and Thomas 1976) provide the data with which to develop a model of the activity profile of match-play.

The physical work load of football match-play is not great; total distance covered is often reported as around 8–12 km over a 90 min match (Reilly 1996). Thus the notion that physiological fatigue is sufficiently cumulative as to cause injury during the latter stages seems surprising. Also injuries are not confined to the latter stages, such that temporary fatigue may occur during the game (Mohr, Krstrup, and Bangsbo 2005). The actual event of injury is arguably more likely to be of biomechanical rather than physiological nature. Thus rather than physiological fatigue

causing injury per sé, it is conceivable that fatigue does produce a mechanical alteration in the technical performance of activities inherent in match-play. Mechanical fatigue therefore may be a greater problem than physiological fatigue. This is supported by the observation that running has been identified as the most common noncontact mechanism of injury (Woods et al. 2004), and that muscular strains to the thigh have been identified as a primary injury type (Hawkins et al. 2001).

The intermittent nature of the activity profile, with a change in speed on average every 6 seconds (Bangsbo 1994), places great emphasis on the acceleration and deceleration phases of the running cycle. Cumulative mechanical stress induced particularly during the intermittent high-intensity bouts of exercise may influence temporal pattern of injury incidence. To investigate the mechanical stressors associated with match-play the activity profile must be validly replicated.

The aim of the present study was to devise a football-specific treadmill-based intermittent protocol replicating the activity profile of match-play. The protocol was to be used to investigate the time history of both the mechanical and physiological responses to the activity profile. The protocol also was used to compare those same responses to SS exercise at the same average running speed. Comparison of the two activity patterns is justified as both intermittent and continuous exercise stimuli are used in football training. Also, whilst it has been stated that the physiological response to intermittent exercise is specific to the activity pattern (Christensen, Hedman, and Saltin 1960), this specificity has not been established for mechanical response.

METHODOLOGY

Subjects

Ten male semiprofessional football players (Mean \pm SD; age 24.7 ± 4.4 yr, body mass 77.1 ± 8.3 kg, $VO_{2\max}$ 63.0 ± 4.8 ml·kg·min⁻¹) were recruited from two Unibond First Division clubs. All players were regular first team members, with previous experience of professional football, and free from injury over the previous season. All players completed, on average, two squad training sessions and two matches per week. All participants provided written informed consent in accordance with the departmental and university ethical procedures.

Experimental Design

Each participant performed all exercise periods at the same time of day to account for the effects of circadian variation (Reilly and Brooks 1986)

with participants being tested between 1500 and 1800 h. Participants attended the laboratory in a 3-hour postabsorptive state, having performed no vigorous exercise or consumed any alcohol or caffeine in the 24 h prior to testing, and with diet standardised for 48 h preceding each test. Players were required to consume 500 ml of fluid 2 h prior to testing to ensure euhydration.

The participants attended the laboratory on two separate occasions within a 3-week period immediately following the end of the competitive playing season. During this period the training load of the playing season was replicated by conducting squad training sessions in addition to the testing sessions that replicated match-play.

Experimental Protocols

The INT and steady-state exercise protocols were performed in randomised order to account for accommodation effects. Prior to testing a minimum of four 30 min habituation sessions were performed on the treadmill, players completing INT exercise protocols similar to that used for data collection. All familiarisation and testing sessions were performed on a programmable motorised treadmill (LOKO S55, Woodway GmbH; Steinackerstraße, Germany). Prior to completing both the intermittent and SS exercise protocols each participant completed a standardised warm-up of 30 mins. This warm-up was devised to replicate the activities performed prior to match-play. The researcher had worked directly with both teams over the course of the previous season and had implemented standardised warm-up routines for both competition and training. The competition warm-up was replicated for the present study, consisting of ball familiarisation drills, progressive intensity running, and passive and dynamic stretching. The small-sided game employed on match-day was replicated in the laboratory by a 10 min INT treadmill protocol.

The soccer-specific INT protocol comprised the varying exercise intensities inherent to match-play (e.g., stationary, walking, jogging, cruising, and sprinting). Specifically the activity profile was based on the notational analysis of Bangsbo (1994), which categorised eight modes of activity, based on the movement speed over a 90 min match play period. Table 1 shows the number of activities performed and the average duration of each activity during 90 minutes of match-play (Bangsbo, 1994). To provide a 15 min activity profile the frequency of each mode of exercise is divided by six. As the treadmill cannot cater for utility movements, the backward running mode has been combined with the low speed mode, both categorised at $12 \text{ km}\cdot\text{h}^{-1}$. The data set upon which the soccer-specific INT treadmill protocol is based is shown in Table 2. This

Table 1. The Number of Activities Performed and the Average Duration of Each Activity During Match-play

| Activity | Number of activities | Mean duration (s) |
|---|----------------------|-------------------|
| Standing (0 km·h ⁻¹) | 122 | 7.8 |
| Walking (4 km·h ⁻¹) | 329 | 6.7 |
| Jogging (8 km·h ⁻¹) | 253 | 3.5 |
| Low speed (12 km·h ⁻¹) | 251 | 3.5 |
| Backward running (12 km·h ⁻¹) | 26 | 3.6 |
| Moderate speed (16 km·h ⁻¹) | 120 | 2.5 |
| High speed (21 km·h ⁻¹) | 57 | 2.1 |
| Sprint (25 km·h ⁻¹) | 19 | 2.0 |
| Total | 1179 | 4.5 |

Table 2. The Data Set Upon Which the Soccer-specific Intermittent Treadmill Protocol Is Based

| Activity | Number of activities | Mean duration (s) |
|---|----------------------|-------------------|
| Standing (0 km·h ⁻¹) | 20 | 7.8 |
| Walking (4 km·h ⁻¹) | 55 | 6.7 |
| Jogging (8 km·h ⁻¹) | 42 | 3.5 |
| Low speed (12 km·h ⁻¹) | 46 | 3.5 |
| Moderate speed (16 km·h ⁻¹) | 20 | 2.5 |
| High speed (21 km·h ⁻¹) | 56 | 2.1 |
| Sprint (25 km·h ⁻¹) | 3 | 2.0 |

data set was arbitrarily distributed to provide a 15 min activity profile replicating the activity pattern of professional football match-play (Figure 1). The treadmill model used for all testing has a maximum acceleration of 2 m·s⁻². The relatively short duration of the high speed movements excluded consecutive stationary and sprint modes of activity. This maximum acceleration was applied for transition to and from all modes of exercise with the exception of the transition from walk to stationary (or vice versa) where an acceleration of 1 m·s⁻² was used. The activity profile was repeated six times in total, with a 15 min half-time interval, during which the participant remained seated and stationary. The 15 min activity profile resulted in a distance covered of 1.62 km, giving a total distance covered of 9.72 km. The SS exercise protocol was completed at a constant speed of 6.5 km·h⁻¹, to provide an equivalent total distance covered. The half-time activity was standardised between the protocols. Both exercise protocols were conducted with a constant treadmill incline of 2% to reflect the energetic cost of outdoor running (Davies 1980; Jones and Doust 1996).

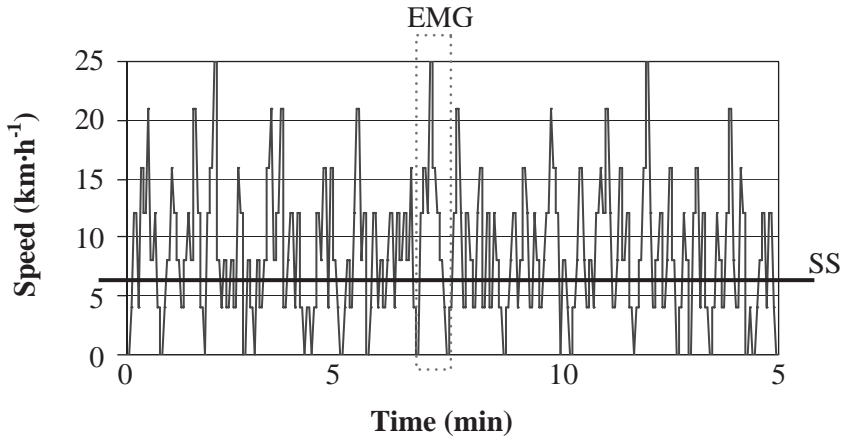


Figure 1. Schematic representation of the soccer-specific intermittent protocol.

Nude Body Mass

Dry nude body mass was measured before and after exercise.

Perceived Exertion

Subjective ratings of perceived exertion were recorded using a 6–20 point scale (Borg 1970) at the midpoint of each 15 min period.

Heart Rate

Heart rate was monitored continuously at 5-s intervals using short-range radio telemetry (Polar Team System, Polar Electro; Kempele, Finland). Mean heart rate was calculated for each 15 min exercise period during both protocols and during each 15 min half-time interval.

Blood Lactate Measurement

Blood lactate concentration was determined at rest and subsequently at the end of each 15 min exercise period, including at the end of the passive half-time interval. The Lactate Pro blood lactate test meter (ARKRAY; Japan) was first calibrated according to the manufacturer's guidelines. The puncture site on the fingertip was cleaned using an alcohol pad and dried with a sterile pad, and subsequently a sample of

free-flowing blood was collected using a Lactate Pro test strip as directed by the manufacturer.

Salivary Cortisol Measurement

Salivary cortisol concentrations were determined at 15 min intervals throughout the duration of each trial. After a resting sample, salivary cortisol also was obtained during the final 45 sec of each 15 min exercise period, and the final 45 sec of the passive half-time interval. To obtain the saliva sample participants were required to chew on a plain (non-citric acid) cotton salivette for 45 sec. During the INT protocol the salivette was administered during a stationary phase. All saliva samples were subsequently labelled according to subject, trial, and time and frozen at -80°C prior to assay.

Saliva samples were subsequently thawed completely, vortexed, and centrifuged at 3000 rpm for 15 mins. Clear 25 μL samples were pipetted in duplicate into prelabelled wells to form the assay plate. The plate also comprised duplicate standards (25 μL) of known concentrations from the kit manufacturer (Salimetrics; USA), which were used to plot the standard curve for each plate. The percent bound for each standard and unknown was calculated and plotted against the log of the concentration for each standard. Log-linear regression was used to draw a line of best fit, and subsequently the concentration of each sample was determined by interpolation.

EMG Measurement

Telemetric electromyographical (EMG) activity (Noraxon; Arizona, USA) was obtained for both the rectus femoris and biceps femoris of the dominant (kicking) leg. Pairs of disposable bipolar silver-silver chloride passive surface electrodes (Medicotest; Denmark) were placed on the belly of each muscle. Electrode location was sited and marked as the visual midpoint of the contracted muscle belly obtained during an isometric maximal voluntary contraction. The orientation of the electrode pair was made parallel to the direction of the muscle fibre alignment, with a separation of 8 mm between the electrodes. Electrode skin site was prepared by first shaving an area sufficiently large as to enable unobstructed placement of the electrode pair, then cleansed with an alcohol swab. A sterile lancet was then used to score three lines along the length of the shaved area, in the direction of alignment of the electrode pairs, whilst ensuring minimal skin damage. A fifth reference electrode was placed on the (inactive and bony) lateral epicondyle of the patella.

The preamplified electrode leads were connected to an 8-channel transmitter unit (Noraxon Telemetry 2400T) placed at the base of the spine to

ensure minimal disruption to movement, and fastened securely within an adjustable belt. The active EMG leads have a preamplifier (gain 500) and 10–1000 Hz band-pass. A three snap lead connected to the rectus femoris electrode pair and the reference electrode, with a two snap to the biceps femoris electrode pair.

A sampling frequency of 1500 Hz was used to collect the EMG signal from both the rectus femoris and biceps femoris. Data collection was constrained to a standardised 30 sec exercise period at the midpoint of the 15 min intermittent activity profile (Figure 1). The stationary period immediately preceding the exercise period over which EMG was to be analysed was used to determine a threshold value to quantify muscle inactivity. Participants were reminded to remain still during this period. This offset value was accounted for in all subsequent analysis of the EMG data. During the steady-state protocol a 30 sec sample was obtained at the midpoint of the 15 min exercise period.

Data processing of the EMG signal was conducted using Noraxon software (MyoResearch XP Master). Raw data were low-pass (300 Hz) and high-pass (10 Hz) filtered. The processed EMG signal was further analysed to determine the peak (EMG_{pk}) and the integrated (iEMG) EMG activity over the 30 sec sample for both the rectus femoris and biceps femoris. This process was repeated for each 15 min exercise period during both the intermittent and SS protocols.

Statistical Analysis

A two-way (treatment \times time) analysis of variance (ANOVA) was performed to compare differences in each of the measures between the intermittent and steady-state exercise conditions, and over the duration of each trial. Significant differences between means were identified using a least-squares difference post-hoc test. Significance was accepted at $P < 0.05$, all results being reported as the mean \pm standard error of the mean.

RESULTS

The soccer-specific INT protocol and SS protocols were completed in laboratory conditions of $21.1 \pm 1.4^\circ\text{C}$ and $49 \pm 3\%$ relative humidity, and $19.7 \pm 1.1^\circ\text{C}$ and $52 \pm 6\%$, respectively. Reductions in body mass were observed in both trials (INT = 1.2 ± 0.3 kg; SS = 0.7 ± 0.7 kg).

Rating of Perceived Exertion

Subjective RPE were greater during the INT protocol than during the SS protocol for each of the 15 min activity periods (Figure 2). This difference

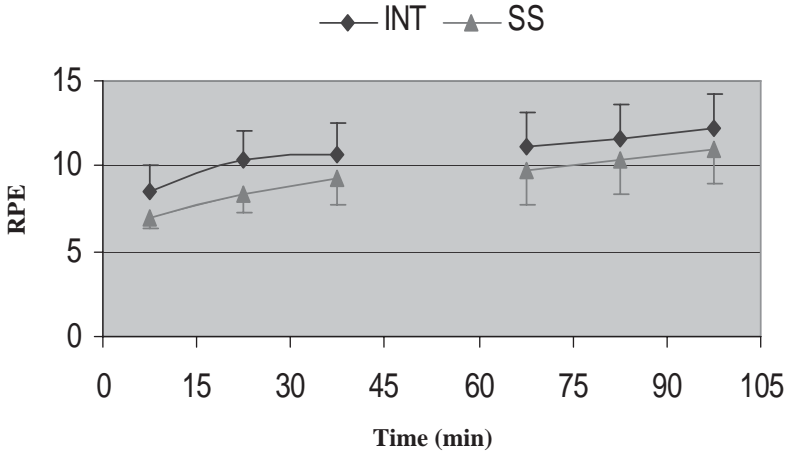


Figure 2. Mean (\pm SD) subjective rating of perceived exertion (RPE) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

was statistically significant for the first 30 min of the trial (RPE_{0-15} : INT = 9 ± 1 , SS = 7 ± 1 , $P < 0.01$; RPE_{15-30} : INT = 10 ± 2 , SS = 8 ± 1 , $P < 0.01$). Thereafter the difference between the two protocols became less great with increased duration of the test (RPE_{90-105} : INT = 12 ± 2 , SS = 11 ± 2 , $P = 0.26$).

The ANOVA revealed a significant ($P < 0.01$) main effect for time during the INT protocol, with RPE gradually increasing throughout the duration of the trial and the post-hoc analyses revealed that RPE over the first 15 min of the trial was significantly lower than at all subsequent time points. The RPE score for the final 15 min period of the INT protocol ($RPE_{90-105} = 12 \pm 2$) was significantly higher than the score observed over the first 30 min of the trial ($RPE_{0-15} = 9 \pm 1$; $RPE_{15-30} = 10 \pm 2$).

A significant ($P < 0.01$) main effect for time also was observed during the SS protocol, RPE gradually increasing as a function of trial duration as with the INT protocol. Post-hoc analyses revealed that the mean subjective RPE score for the first 15 min of the trial ($RPE_{0-15} = 7 \pm 1$) was significantly lower than the RPE for the final 15 min of the first half ($RPE_{30-45} = 9 \pm 2$) and the entire second half ($RPE_{60-75} = 10 \pm 2$; $RPE_{75-90} = 10 \pm 2$; $RPE_{90-105} = 11 \pm 2$). RPE_{15-30} remained significantly lower than RPE_{75-90} and RPE_{90-105} , and the subjective rating for the final 15 min of the first half (RPE_{30-45}) was significantly lower than for the final 15 min of the second half (RPE_{90-105}).

Heart Rate

Heart rate (HR) was greater during the INT protocol than during the SS protocol for each of the 15 min activity periods (Figure 3). Whilst these differences were not significant it was observed that the difference was reduced toward the latter stages of the trial (t_{0-15} , t_{15-30} , t_{30-45} , t_{60-75} : $P = 0.06$; t_{75-90} : $P = 0.10$; t_{90-105} : $P = 0.20$). Heart rate was also greater during the 15 min recovery half-time period for the INT protocol ($HR_{45-60} = 89 \pm 15 \text{ beats}\cdot\text{min}^{-1}$) in comparison with the SS protocol ($HR_{45-60} = 84 \pm 13 \text{ beats}\cdot\text{min}^{-1}$), although this difference was not significant ($P = 0.44$).

The ANOVA revealed a significant main effect for time during the INT protocol ($P < 0.01$). Heart rate showed a gradual increase during the INT protocol; the HR in the final 15 min ($HR_{90-105} = 135 \pm 10 \text{ beats}\cdot\text{min}^{-1}$) was significantly different from that observed for the first 15 min ($HR_{0-15} = 125 \pm 10 \text{ beats}\cdot\text{min}^{-1}$). The HR during the half-time interval ($HR_{45-60} = 89 \pm 15 \text{ beats}\cdot\text{min}^{-1}$) was significantly lower than during each of the exercise periods.

Furthermore, the ANOVA revealed no significant main effect for time during the SS protocol ($P > 0.30$), while post-hoc testing revealed that the only significant differences were between the half-time interval ($HR_{45-60} = 84 \pm 13 \text{ beats}\cdot\text{min}^{-1}$) and the exercise periods at the end of the first half ($HR_{30-45} = 122 \pm 8 \text{ beats}\cdot\text{min}^{-1}$) and toward the latter stages of the second half ($HR_{75-90} = 124 \pm 9 \text{ beats}\cdot\text{min}^{-1}$; $HR_{90-105} = 129 \pm 10 \text{ beats}\cdot\text{min}^{-1}$).

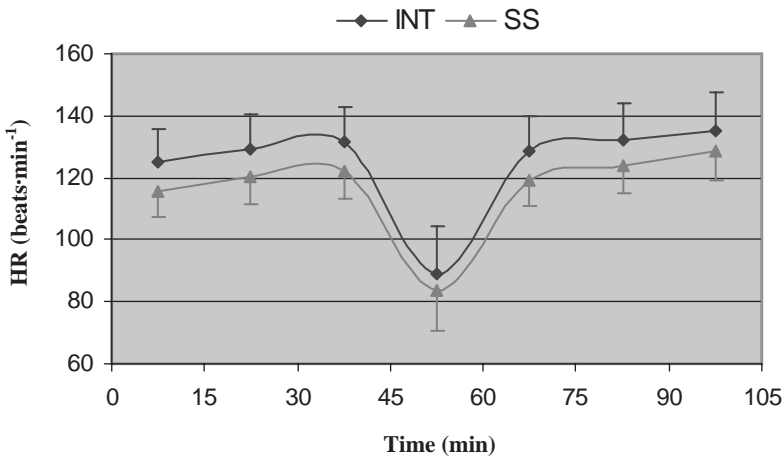


Figure 3. Mean (\pm SD) heart rate (HR) response ($\text{beats}\cdot\text{min}^{-1}$) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

Blood Lactate

Blood lactate concentration (BLa) at rest was equal for both the INT and SS trials ($INT_0 = 1.1 \pm 0.3 \text{ mM}\cdot\text{l}^{-1}$; $SS_0 = 1.1 \pm 0.2 \text{ mM}\cdot\text{l}^{-1}$). The BLa was also equivalent between the two trials at the end of the 15 min half-time interval ($INT_{60} = 0.9 \pm 0.3 \text{ mM}\cdot\text{l}^{-1}$; $SS_{60} = 1.0 \pm 0.2 \text{ mM}\cdot\text{l}^{-1}$). At each stage during the 45 min exercise periods BLa was greater during the INT protocol than during the SS protocol, this difference increasing as a function of simulated match-play duration (Figure 4). Whilst there was a trend toward significance as a function of exercise duration, however, at no time point was the BLa significantly ($P < 0.05$) greater during the INT protocol.

The ANOVA revealed no significant main effect for time during the SS protocol. Similarly no main effect for time was observed during the INT protocol, this despite the progressive increase during each 45 min activity period. Post-hoc testing revealed the only significant difference to be between the BLa concentration at the end of the simulated match ($BLa_{105} = 1.4 \pm 0.5 \text{ mM}\cdot\text{l}^{-1}$) and the value obtained at the end of the stationary half-time interval ($BLa_{60} = 0.9 \pm 0.3 \text{ mM}\cdot\text{l}^{-1}$).

Salivary Cortisol

A correlation coefficient of $r^2 \geq 0.98$ was obtained for each standard curve used to determine salivary cortisol concentrations. Salivary cortisol

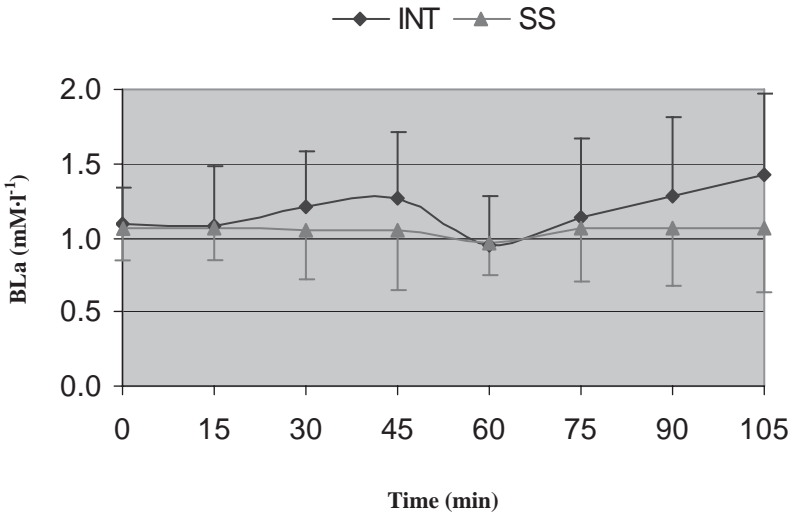


Figure 4. Mean (\pm SD) blood lactate (BLa) concentration ($\text{mM}\cdot\text{l}^{-1}$) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

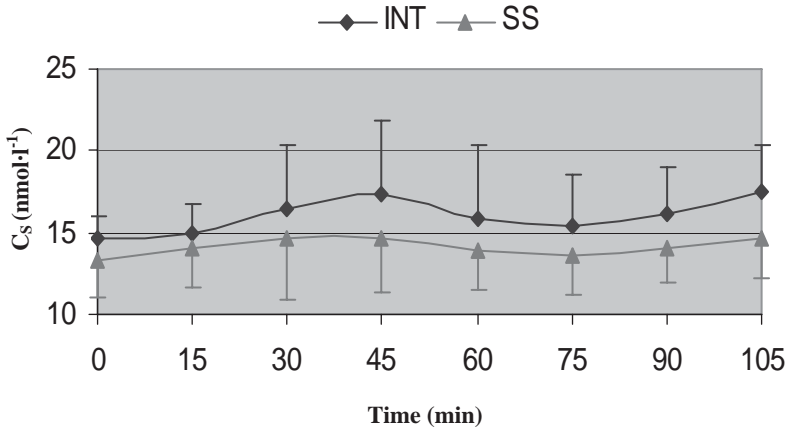


Figure 5. Mean (\pm SD) salivary cortisol (C_S) concentration ($\text{nmol}\cdot\text{l}^{-1}$) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

concentration (C_S) was greater during the INT protocol than during the SS protocol at each time point (Figure 5). The salivary cortisol remained constant throughout the SS protocol whilst concentration increased progressively during each of the 45 min activity periods of the INT protocol. The gradual increase in C_S during the INT protocol was such that P tended toward a significant difference between the two protocols; however, even at the final time-point significance was not achieved at the set level ($P > 0.07$).

No significant main effect for time was observed for either the SS or INT protocol. Whilst C_S increased progressively during both the first ($C_{S0} = 14.6 \pm 1.4 \text{ nmol}\cdot\text{l}^{-1}$; $C_{S45} = 17.4 \pm 4.4 \text{ nmol}\cdot\text{l}^{-1}$) and second ($C_{S60} = 15.8 \pm 4.6 \text{ nmol}\cdot\text{l}^{-1}$; $C_{S105} = 17.5 \pm 2.9 \text{ nmol}\cdot\text{l}^{-1}$) 45 min activity periods during the INT protocol, no significance was observed. A similar but less marked pattern was observed during the SS trial.

Electromyography: Biceps Femoris, Total EMG

The total EMG output (iEMG) from the biceps femoris over the 30s data collection period was greater during the INT protocol than during the SS protocol throughout the trial (Figure 6). Only during the first 15 min activity period did the difference fail to reach the accepted significance level ($\text{INT}_{0-15} = 744 \pm 203 \mu\text{V}$, $\text{SS}_{0-15} = 569 \pm 155 \mu\text{V}$, $P = 0.07$). Thereafter iEMG was significantly greater during the INT protocol than during the corresponding period of the SS protocol. This difference tended to increase as a function of time during each of the 45 min exercise periods, this pattern even more marked during the second half, such that the difference

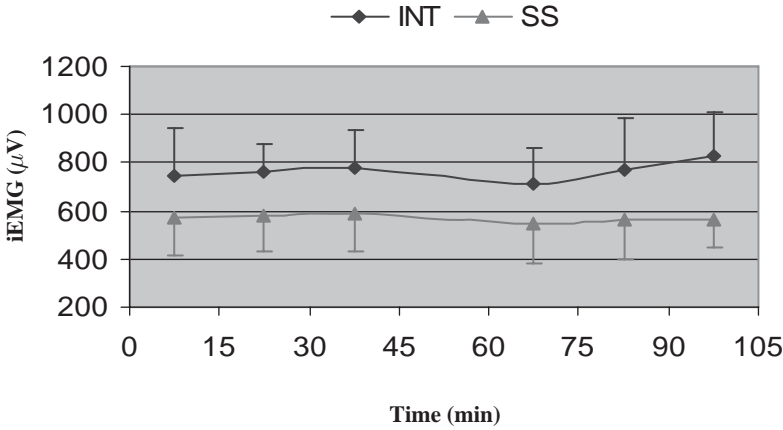


Figure 6. Mean (\pm SD) total biceps femoris EMG output (iEMG, μ V) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

between the two protocols was greatest for the final 15 min period ($INT_{90-105} = 829 \pm 180 \mu$ V, $SS_{90-105} = 564 \pm 118 \mu$ V, $P < 0.01$).

The ANOVA revealed no significant main effect for time during either protocol. Figure 5 shows that the biceps femoris iEMG tended to increase as a function of exercise duration during each trial, and more markedly so during the INT protocol; however, statistical significance ($P < 0.05$) was not attained.

Electromyography: Biceps Femoris, Peak EMG

The peak EMG output (EMG_{pk}) from the biceps femoris during the 30s data sample was greater during the INT protocol than during the SS protocol throughout (Figure 7). This difference was significantly greater at the start of each half ($INT_{0-15} = 617 \pm 72 \mu$ V, $SS_{0-15} = 468 \pm 142 \mu$ V, $P = 0.05$; $INT_{60-75} = 679 \pm 172 \mu$ V, $SS_{60-75} = 510 \pm 174 \mu$ V, $P = 0.03$) and at the end of the second half ($INT_{90-105} = 733 \pm 263 \mu$ V, $SS_{90-105} = 518 \pm 165 \mu$ V, $P = 0.05$).

Peak EMG activity at the biceps femoris increased as a function of exercise time during both the INT and SS protocols. No significant ($P < 0.05$) main effect for time was observed, however, for either protocol.

Electromyography: Rectus Femoris, Total EMG

In contrast to the pattern observed for the biceps femoris, the iEMG from the rectus femoris was not consistently greater during the INT protocol than during the SS protocol (Figure 8). Muscular output during the 30s

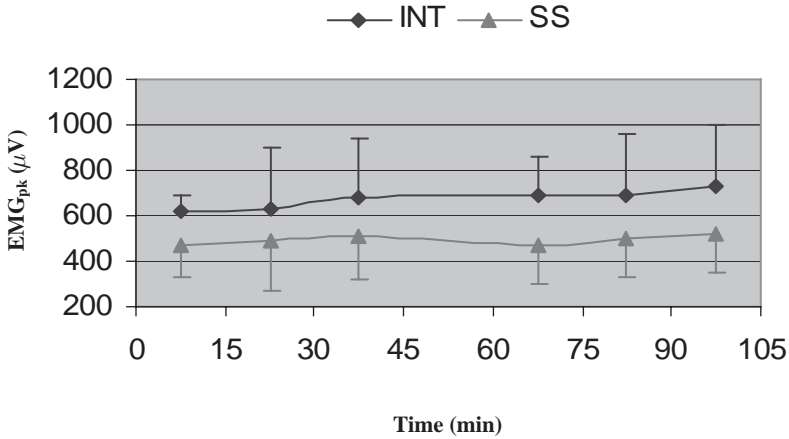


Figure 7. Mean (\pm SD) peak biceps femoris EMG output (EMG_{pk} , μV) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

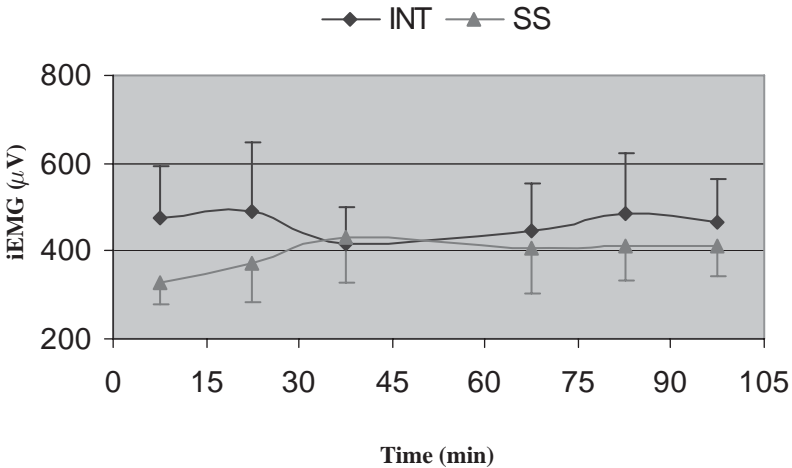


Figure 8. Mean (\pm SD) total rectus femoris EMG output (iEMG, μV) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

sample within the first 15 min activity was significantly greater during the INT protocol ($INT_{0-15} = 474 \pm 117 \mu V$, $SS_{0-15} = 328 \pm 47 \mu V$, $P < 0.01$). For the final 15 min period of the first half, however, the rectus femoris output actually was slightly greater during the SS protocol ($INT_{30-45} = 414 \pm 86 \mu V$, $SS_{30-45} = 431 \pm 103 \mu V$, $P = 0.71$). Throughout the second 45 min exercise period the sampled iEMG was greater during the INT

protocol than during the corresponding period of the SS protocol. This difference failed to increase as a function of time (as observed with the biceps femoris), however, such that no significant difference was observed ($INT_{90-105} = 464 \pm 100 \mu V$, $SS_{90-105} = 410 \pm 68 \mu V$, $P = 0.41$).

The ANOVA revealed no significant main effect for time during either protocol. Figure 8 shows that the rectus femoris iEMG tended to decrease during the final 15 min of each half of the INT protocol. During the SS protocol the rectus femoris EMG_S increased progressively during each half, although this pattern was less marked during the second 45 min period.

Electromyography: Rectus Femoris, Peak EMG

As observed with peak biceps femoris activity, the peak EMG output (EMG_{pk}) from the rectus femoris was greater during the INT protocol than during the SS protocol throughout (Figure 9). This difference was significant ($P < 0.05$) at each time point.

ANOVA revealed a significant main effect for time in peak rectus femoris activity. For the SS protocol the peak activity toward the end of the trial ($SS_{75-90} = 283 \pm 98 \mu V$, $SS_{90-105} = 282 \pm 86 \mu V$) was significantly ($P < 0.05$) higher than that recorded at the start of the trial ($SS_{0-15} = 196 \pm 56 \mu V$). For the INT protocol the peak activity recorded over the final 15 min of the second half ($INT_{90-105} = 495 \pm 186 \mu V$) was significantly ($P = 0.03$) greater than that recorded over the first 30 min of the first half ($INT_{0-15} = 341 \pm 43 \mu V$; $INT_{15-30} = 350 \pm 104 \mu V$).

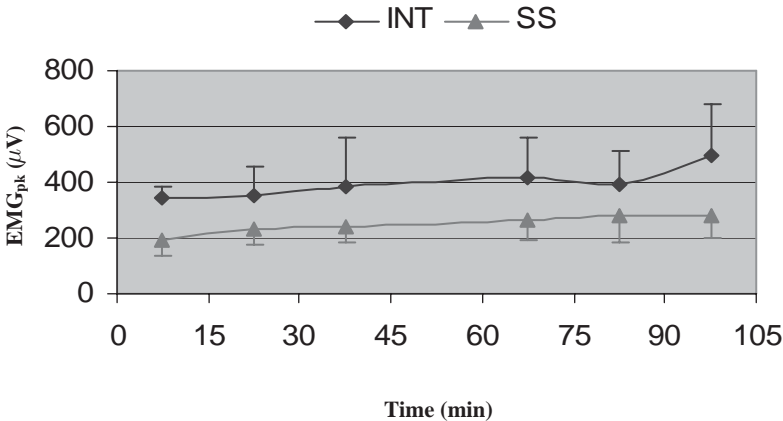


Figure 9. Mean ($\pm SD$) peak rectus femoris EMG output (EMG_{pk} , μV) during the soccer-specific intermittent (INT) and steady-state (SS) protocols.

DISCUSSION

The primary aim of the present study was to investigate the time history of physiological and mechanical responses to a soccer-specific INT treadmill protocol. Specificity to soccer match-play was validated by using an activity profile based on notational analyses of match-play (Bangsbo 1994). A secondary aim was to compare the same physiological and mechanical responses to a constant velocity protocol that produced the same total distance covered (9.7 km).

As expected the physiological responses (RPE, HR, BLa, salivary cortisol concentration) were greater during the intermittent protocol than during the SS protocol. The magnitude of the physiological response tended to increase as a function of exercise duration, suggesting a cumulative affect of physiological strain. This increase as a function of exercise duration was more marked during the INT protocol. These findings are, as expected, that intermittent exercise is acknowledged as eliciting a greater physiological strain than exercise of a continuous and steady rate nature (Bangsbo 1994). To evaluate the INT protocol, however, the physiological and mechanical responses to the protocol must be compared with data obtained from match-play wherever possible.

Heart rate during the INT trial increased from 125 ± 10 beats·min⁻¹ during the first 15 minutes of simulated match-play to 135 ± 10 beats·min⁻¹ during the final 15 minutes. In contrast, HR values of 171 beats·min⁻¹ have been observed during competitive match-play (Bangsbo 1994). Blood lactate concentration peaked at the end of the simulated game at 1.4 ± 0.5 mM·l⁻¹. Blood lactate concentrations of approximately 4.4 mM·l⁻¹ have been observed at the end of competitive matches (Bangsbo, Norregaard, and Thorso 1991; Rohde and Espersen 1988).

Even accounting for the methodological issues in collecting such data, the values obtained in the present study are consistently lower than observed in previous research considering competitive match-play. This may be explained, at least in part, by situational factors. Interestingly, Reilly (1986) recorded HR of 157 beats·min⁻¹ during friendly games. Whilst the total distance covered was reported to be within 2% of competitive match-play data, the HR response was reduced. Just as a friendly match might place reduced emotional stress on the player, completing the activity profile in the laboratory environment might reduce the emotional stress inherent within the competitive environment (Whitehead et al. 1996). Theoretically, such additional stressors may arise from interactions with other players, the match situation, and importance of the game.

In addition to the psychological stressors that would be evident in match-play, which may serve to increase the physiological response, the nature of the treadmill protocol is such that many activities inherent in

match-play are not included. Utility movements such as dribbling with the ball have been shown to increase the physiological load (Kirkendall 2000; Reilly and Ball 1984). Furthermore, the irregular, INT activity profile of match-play is further complicated by its multidirectional nature. There is an increased physiological cost of “unorthodox” modes of running such as backward or lateral, this load increasing disproportionately as a function of movement speed (Reilly and Bowen 1984; Williford et al. 1998).

Based on such limitations it should be argued that a laboratory-based replication of purely the activity profile of competition should not elicit the same physiological response as match-play.

The finding of reduced physiological load with respect to match-play data is supported by the subjective rating of load during the INT protocol. The rating of perceived exertion was consistently greater during the INT protocol than during the SS protocol, and increased gradually as a function of exercise duration. The mean RPE at the end of the INT protocol, however, was 12, equivalent to “fairly light,” and the highest recorded value was 15, or “hard” (mean HR 135 ± 12 beats·min⁻¹, max. 153 beats·min⁻¹). On the 20-point scale used in the present study this gives an indication that the players were not overly stressed by completion of the protocol.

In evaluating the INT exercise protocol as a “model” of match-play, it is also relevant to compare the output and input or structure of the model, with other models developed to “simulate” the demands of soccer match-play. Previous models (Drust, Reilly, and Cable 2000; Nicholas, Nuttall, and Williams 2000) have been declared successful in replicating the physiological demands of soccer match-play. The structure of such models, however, questions the validity of the findings: A model must be based on realistic input. The free-running protocol developed by Nicholas, Nuttall, and Williams (2000), has intuitive appeal as a fitness test, but it should not be considered a valid model of match-play. The activity profile of this model (Nicholas, Nuttall, and Williams 2000) does not accurately represent the intermittent nature of soccer match-play, comprising a 60m bout of walking, for example. Notational studies of match-play would suggest that the average distance covered walking is approximately 7m (Bangsbo, 1994; Reilly and Thomas, 1976). At walking pace this discrete bout of exercise would last for approximately 7s. The treadmill protocol of Drust, Reilly, and Cable (2000) also fails to replicate the frequency of activity change evident in match-play. The duration of the walking bout in this model is 35.3s. Drust, Reilly, and Cable (2000) also used an SS protocol at a constant running speed of 12km·h⁻¹, this being the same average speed as their INT protocol. This would give a total distance covered of 18 km, far in excess of the total distance observed during match-play.

Thus whether free-running or treadmill-based, previous protocols have failed to replicate the frequency of change in activity observed during match-play. It is this failure that has resulted in producing physiological responses of equal magnitude to match-play, despite the additional stressors associated with the situational context of competition. Just as the lower intensity bouts of exercise are too long in such models, so too are the high intensity bouts. Nicholas, Nuttall, and Williams (2000) have a 60m phase at 95% of VO_2max . Drust, Reilly, and Cable (2000) maintain a cruising speed of $15 \text{ km}\cdot\text{h}^{-1}$ for 51.4s, and a sprinting speed of $21 \text{ km}\cdot\text{h}^{-1}$ for 10.5s. These high intensity bouts are much longer than observed in match-play, and with regard to the aims of such research, are sufficiently long as to elicit elevated HRA and BLa concentrations.

It was the intention of the present protocol to more closely replicate the frequency of velocity change observed during match-play. This protocol is not merely intermittent in nature, but as intermittent as match-play. This is likely to make a great difference to the physiological (and mechanical) response. The INT protocol simulates the work-rate pattern of match-play, and hence reflects the 7:1 ratio of low intensity work to high intensity work. The predominance of low to moderate intensity exercise facilitates active recovery in accelerating the removal of metabolic waste products such as lactate from the blood. It appears that the periods of low intensity are sufficiently long, and the high intensity bouts sufficiently short, as to dissipate the cumulative nature of physiological stress.

Whilst the frequency of velocity change is sufficient to reduce the potentially limiting accumulation of physiological stress, however it is likely that the mechanical stress will be increased. More frequent periods of acceleration and deceleration are likely to impose greater mechanical stress on the musculoskeletal system. This is supported by the finding of greater muscular activity during the INT protocol than during the constant velocity protocol.

Fatigue has been described as being indicated by reduced maximal force or power (Reilly 1996), this reduction in muscular force attributed to reduced fibre recruitment (Bangsbo 1994). Thus a fatigue affect has been quantified by comparing maximal voluntary force before and after exercise (Rahnama *et al.* 2003), although the functionality of such isometric tasks lacks specificity to the incidence of injury during soccer activities. It is more difficult to examine the time history of muscular fatigue during match-play. Subsequently, it is difficult to evaluate the EMG time histories obtained in the present study with data from actual match-play.

Due to such methodological constraints, most studies investigating the influence of soccer-specific fatigue on mechanical responses have used a postexercise evaluation of muscular function. Whilst fatigue may be cumulative, and might be expected to peak at the end of a game, it should

be noted that muscular strain injuries occur at all stages of match-play (Hawkins et al. 2001) as does fatigue (Mohr, Krstrup, and Bangsbo 2005).

Many of the studies investigating the mechanical response to soccer-specific exercise are limited further by the same validity issues as discussed previously in replicating the demands of match-play, particularly the biomechanical demands. There are parallels to be drawn, however, with the mechanical responses observed in the present study. Rahnama et al. (2003) used the protocol of Drust, Reilly, and Cable (2000) to demonstrate a progressive reduction in peak isokinetic strength of both the knee flexors and extensors. Mercer, Gleeson, and Wren (2003) also demonstrated reduced knee flexor strength following an acute and prolonged high intensity task designed to simulate the energetics of match-play. It should be noted that the task was performed as a single leg pedalling task on a modified cycle ergometer. Gleeson et al. (1998) used a free-running protocol similar to that of Nicholas, Nuttall, and Williams (2000) to demonstrate impaired knee flexor and extensor strength, electromechanical delay, and anterior tibio-femoral displacement. Such studies, despite variation in methodology, consistently imply an increased risk of injury due to compromised mechanics.

The present study aimed to quantify the fatigue affect on the EMG of functional movements performed during the soccer-specific activity profile. Movement inherent in match-play does not necessitate maximal force production, and thus the aim was to analyse alteration in the movement inherent in performance. Rahnama, Lees, and Reilly (2005) conducted a similar study, analysing EMG activity of the lower extremity pre-, mid-, and post-completion of the INT protocol developed by Drust, Reilly, and Cable (2000). As discussed previously this protocol is more demanding than match-play with respect to the nature of the high intensity bouts. An additional limitation of the application of the findings of Rahnama, Lees, and Reilly is attributed to the methodology used. Rather than collecting EMG data during the protocol, the authors collected EMG over a 3 m period within a separate protocol. Muscular activity was analysed over 5s of a 15s bout of continuous exercise at four different speeds. As expected, EMG activity was observed to increase as a function of running speed. In contrast to the findings of the present study, however, Rahnama, Lees, and Reilly found that EMG activity decreased as a function of exercise duration. This finding also contradicts the findings of other studies that have shown either no change (Miller et al. 2000) or an increase in EMG amplitude during repetitive exercise (Ober 1995; Kellis and Baltzopoulos 1999).

These contradictory results can be attributed, at least in part, to methodological variation with respect to the activity protocol. Rahnama et al.

(2005) collected EMG data during a 15s exercise bout, whereas in the present study no single bout of exercise is beyond 8s, in accord with notational data of match-play (Bangsbo 1994). In both the present study and the study of Rahnama, Lees, and Reilly (2005) the activity protocol during which EMG is collected is repeated, such that the locomotor requirement is maintained irrespective of the level of fatigue. Rahnama, Lees, and Reilly acknowledged that the expected result would be increased muscular activation with fatigue, and they attributed their findings to compensations in the locomotor pattern to enable the player to accommodate the same workload. This compensation is much more plausible over a 15s continuous bout of exercise than during the much higher frequency of speed change required in the INT protocol presented in this chapter. Thus it may be the specifics of the activity profile that create the contradictory findings. The notion of technical compensation in the fatigued state, and a change in locomotion, might be achieved by greater recruitment of other muscles.

In the present study the biceps femoris was required to produce greater muscular output to achieve the same standardised workload. Both the integrated EMG over a 30s intermittent bout of exercise and the peak EMG during this bout increased as a function of exercise duration. During sprinting activities, the hamstring muscle complex is working harder to produce the same running speed and to accommodate changes in speed. This will predispose the hamstring muscle complex to injury during the latter stages of each half, supporting the prospective epidemiological injury incidence data of Hawkins et al. (2001). This increased hamstring activity may in turn create the apparent inhibition of the quadriceps group. During the last 15 m of each half of the simulated game the total muscular work performed by the rectus femoris decreased. This apparent redistribution of muscular work is likely to increase the risk of muscular strain injury and might be attributed to altered kinematics of movement in the latter stages.

Running has been identified as a primary mechanism of soccer injury (Hawkins et al. 2001). Altered kinematics as a result of fatigue highlights the mechanical implications of injury incidence. Pinniger, Steele, and Groeller (2000) observed altered kinematics and EMG activity in sprinting following hamstring fatigue tasks. Hamstring activity was extended in duration whilst rectus femoris activity was observed to switch off earlier in the swing phase of the sprint cycle. These observations support the findings of the present study, despite methodological differences in the running task. Localised muscle fatigue also has been shown to impair function in ergonomic tasks. Gorelick, Brown, and Groeller (2003) reported alteration in both magnitude and sequencing of muscle activation following localised muscular fatigue tasks. No such alteration in coordinated

performance was observed, however, following a generalised fatigue task designed to induce cardiovascular stress. Thus it may be interpreted that the variation observed in EMG measures during the INT protocol is a direct result of the load placed on the hamstrings group by the frequency of acceleration. Mechanical rather than physiological fatigue is therefore a greater consideration with respect to the incidence of injury during the latter stages of match-play.

It also must be considered that the nature of the protocol might impose kinematic constraints on movement. With the exception of the transition from stationary to walk, the protocol used an acceleration of $2 \text{ m}\cdot\text{s}^{-2}$. This was the maximum acceleration of the treadmill used. Data provided by notational analyses (Bangsbo 1994; Reilly and Thomas 1976) does not account for transition between the speed classifications used. As such a bout of sprinting in the current protocol also includes the time taken to accelerate from the previous bout. This reduces the total amount of work done during each high intensity bout. This is likely to reduce both the physiological and mechanical load. Drust, Cable, and Reilly (2000) acknowledged the problem of acceleration in selecting to use a nonmotorised treadmill.

It should also be noted that there will be both kinetic and kinematic variation between overground and treadmill running. Wank, Frick, and Schmidtbleicher (1998) observed a shorter stride length, a compensatory higher stride frequency, and lower vertical displacement of the mass centre in treadmill running. Such kinematic alterations will necessitate altered muscular recruitment strategies. It should be noted, however, that in the present study the subjects were selected as being familiar with the activity pattern and fully habituated to the treadmill protocol. Soccer players, due to the demands of the game, typically have altered kinematics to runners, making comparisons with studies on distance running difficult.

CONCLUSION

The INT protocol replicates the activity pattern of match-play. This protocol imposed greater physiological load than constant velocity running at the same average velocity. The physiological responses to the INT protocol, however, were lower than have been observed during match-play. This may be attributed to the additional physiological cost of the many activities inherent in soccer that are not reproduced in the treadmill protocol. There is also likely to be greater stress imposed in the competitive environment by the very nature of the situation. The mechanical responses to the INT protocol support epidemiological data on the increased incidence of muscular strain injuries during the latter stages of

match-play. The frequency of change in running speed induces mechanical load on the musculature, which is cumulative in nature.

A total distance covered of approximately 10 km in 90 min does not suggest a great physiological cost. The increased incidence of injury toward the latter stages of match-play, having been attributed to fatigue, is more likely to be the result of mechanical rather than physiological load. Specifically, altered kinematics and kinetics of running with cumulative fatigue may predispose the player to injury.

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