Physical response to fixture congestion in soccer

MANUSCRIPT TITLE: The physical response to a simulated period of soccer-specific fixture congestion
ABSTRACT

The aim of this study was to assess the physiological, perceptual, and mechanical measures associated with the completion of a simulated period of short-term soccer-specific fixture congestion. Ten male semi-professional soccer players completed three trials of a treadmill-based match simulation, with 48 hours intervening each trial. A repeated measures general linear model identified significantly (P= 0.02) lower knee flexor peak torque (PT) recorded at 300 degs∙s⁻¹ in the second (141.27 ± 28.51 Nm) and third trials (139.12 ± 26.23 Nm) when compared to the first (154.17 ± 35.25 Nm). Similarly, muscle soreness (MS) and PT data recorded at 60 degs∙s⁻¹ were significantly (P< 0.05) different in the third trial (MS= 42 ± 25 a.u; PT60= 131.10 ± 35.38 Nm) when compared to the first (MS= 29 ± 29 a.u; PT60= 145.61 ± 42.86 Nm). Significant (P= 0.003) differences were also observed for mean Bicep Femoris electromyography (EMG_{\text{mean}}) between the third trial (T_{0-15}= 126.36 ± 15.57 \mu V; T_{75-90}= 52.18 ± 17.19 \mu V) and corresponding time points in the first trial (T_{0-15}= 98.20 ± 23.49 \mu V; T_{75-90}= 99.97 ± 39.81 \mu V). Cumulative increases in perceived exertion, heart rate, oxygen consumption, blood lactate concentrations, EMG_{\text{mean}}, and PlayerLoad™ were recorded across each trial. MS and PT were also significantly different post-trial. There were however no significant main effects or interactions for the salivary Immunoglobulin A, and the medial-lateral PlayerLoad™ metrics. These data suggest a biomechanical and muscular emphasis with residual fatigue, with implications for injury risk and the development of recovery strategies.

Key Words: Biomechanics, Physiology, Isokinetic, electromyography, PlayerLoad™, Recovery
INTRODUCTION

Injury incidence continues to pose a contemporary issue associated with soccer, with fatigue being identified as a key risk factor for injury in intermittent team sports (14). Fatigue in soccer can occur temporarily following a period of high intensity (HI) activity (28), cumulatively towards the end of a match (23), and residually between successive bouts of match-play (5). It is for this reason that insufficient recovery periods between successive matches, and the occurrence of congested fixture periods (a minimum of two successive bouts of match-play, with an inter-match recovery period of ≤96 hours [5-8, 12, 13, 16, 24, 31, 34, 39]) have been previously identified as two of the top five extrinsic risk factors associated with soccer injuries (26). As such, it is important to be able to assess markers of fatigue and injury risk both during and between successive matches, especially during periods of fixture congestion.

Contemporary soccer players can be exposed to periods of congested fixture schedules, with implications for both performance (6, 31, 39) and injury risk (12, 13, 14, 30). It is common for soccer players to compete in 50-80 matches during a ~40 week competitive season. Soccer players are therefore regularly required to compete in two matches per week, with some teams completing three matches in a weekly microcycle (5, 7, 12, 13, 31, 39). Although the rotation of squads may prevent some players from competing in congested schedules, ~40% of players are required to complete all matches during a two or three game microcycle (7). Three matches completed in a 5 day period is typically the worst case scenario for fixture congestion (7, 31), with previous literature suggesting that a 2 day period is not sufficient to allow for full recovery (21). Impaired physical recovery can in turn lead to residual fatigue, impaired performances, and increased risk of injury.

The most prevalent injuries during intense fixture schedules appear to be thigh based strains, ruptures, tears, and cramps (13), with the knee flexor musculature (most specifically the bicep
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femoris) being most commonly injured (14). These data therefore further reiterate the need to assess the mechanisms associated with the observed increase in muscular injuries associated with periods of fixture congestion. When considering the aetiological risk factors commonly reported for knee flexor injuries, Isokinetic dynamometer based strength assessments have previously been utilised to infer increased injury risk as a result of fatigue induced changes in strength following the completion of single (18, 21, 25, 36, 37) and repeated bouts of soccer-specific activity (10). In addition to quantifying maximum strength, previous literature has also utilised electromyography (EMG) to quantify fatigue induced changes in muscle activity (17, 25). To the author’s knowledge, the EMG response to successive bouts of soccer-specific activity has not been previously assessed.

It has also previously been suggested that athletes undergo increased stress when exposed to repeated bouts of activity, thus resulting in impaired mucosal immunity (27, 29, 40). Impaired immune function, as typically assessed via the measurement of Salivary Immunoglobulin A (SIgA), can result in time loss from training and match-play, with obvious implications for performance (29, 40). These data reiterate the need to further assess the mechanisms associated with these increased rates of muscular injuries and impaired immune function during periods of fixture congestion.

The majority of studies associated with physical fatigue response associated with soccer match-play have typically focussed on the response associated with a single bout of soccer-specific activity. Where studies have attempted to assess the physical response associated with the completion of successive bouts of soccer-specific activity, they have typically done so via the assessment of actual match-play (5, 6, 8, 12, 13, 16, 27, 31). Previous fixture congestion literature has also typically focussed on match schedules characterised by inter-match recovery periods of greater than 72 hours (6, 8, 12, 13, 16), with only a couple of studies considering
shorter inter-match recovery durations with adult players (5, 31). Literature associated with more congested fixture scheduling has therefore recently been advocated. Likewise, although soccer match-play offers high ecological validity, there are restrictions on data collection (39, 43) and matches are susceptible to contextual factors (39), such as, but not limited to, formation, tactics, time spent in possession of the ball, total playing time, match result, and playing environment (5). The use of valid soccer-specific exercise protocols (SSEPs) have therefore recently been advocated as a potential method of assessing the physical response to a simulated period of fixture congestion (5).

The physical response associated with the completion of three SSEPs completed over a 5 day period has previously been assessed in relation to N-Acetylcysteine supplementation (10); however, the SSEP used in this study was not a valid representation of the velocity profile or distance covered during soccer match-play. A more contemporary study (34) assessed the physical response associated with the completion of two bouts of a valid and contemporary SSEP interspersed by either 48 or 72 hours. It was identified that 48h recovery appeared sufficient to recover all physical measures. These data cannot however be generalised beyond the measures used, thus advocating a need for future research to measure additional physical measures associated with aetiological risk factors for injury.

Given the potentially detrimental effects associated with periods of short-term fixture congestion, the aim of the current study was to assess the temporary, cumulative, and residual physical fatigue response during a simulated microcycle comprising 3 SSEPs completed over a 5 day period.
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METHOD

Experimental approach to the problem

This experimental study comprised a repeated measures design to identify the temporary, cumulative, and residual physical fatigue response to a simulated period of short-term soccer-specific fixture congestion. The participants were required to complete three experimental trials with 48 hours interspersing each trial. Considering the epidemiology of lower limb injuries in male soccer (14) especially during periods of fixture congestion (13), a number of the current experimental measures were chosen to assess aetiological markers of injury risk. The experimental trials comprised the completion of a contemporary and valid treadmill-based SSEP (33). The SSEP was utilised to ensure mechanistic rigour by standardising both the locomotion and speed profile performed by the participants. By ensuring each bout of activity was standardised, any observed differences in the dependant variables were attributable to fatigue induced changes, and not due to differences in speed and/or activity profiles performed across the trials. It was identified that although free-running SSEPs may offer increased ecological validity when compared to treadmill-based protocols, free-running SSEPs (42) do not typically standardise the running speeds performed by the participants and, consequently, this makes it more difficult to mechanistically interpret the differences in the physical fatigue response.

Subjects

Ten male semi-professional soccer players (age: 25.60 ± 3.78 years, height: 179.0 ± 7.8 cm, mass: 79.01 ± 6.78 kg, \( \text{VO}_{2\text{max}} \): 56.16 ± 5.82 ml·kg\(^{-1}\)·min\(^{-1}\); maximum heart rate: 193 ± 8 beats·min\(^{-1}\); lactate threshold running velocity: 13.01 ± 0.36 km·hr\(^{-1}\)) volunteered to complete this study. Inclusion criteria specified that during the weeks prior to testing, in addition to weekly matches, all participants were regularly completing training volumes equating to > 6
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h·week\(^{-1}\). The participants were not however involved in any training and match-play during the weekly microcycle where they were required to complete the experimental trials. The participants were also required to demonstrate the capacity to complete a 30 minute familiarisation session specific to the demands of the experimental trials. All participants were injury free for a minimum of 6 months prior to testing, and were outfield players. Prior to the commencement of each trial, participants completed a comprehensive health screening procedure to further assess the participant’s eligibility and also highlight potential risks. The current study was approved by a local University Research Ethics Committee and conformed to the declaration of Helsinki. Written informed consent was obtained for all participants. All equipment was risk assessed and calibrated in accordance to the manufacturers guidelines prior to testing commencing.

**Procedures**

Participants attended the laboratory on five occasions to complete a 30 minute familiarisation trial, a combined lactate threshold and maximal aerobic capacity (\(\dot{V}O_{2\text{max}}\)) test, followed by three experimental trials. A period of 96 hours interspersed completion of the familiarisation trial, the combined lactate threshold and \(\dot{V}O_{2\text{max}}\) test, and the start of the first experimental trial. All trials were completed on a programmable motorised treadmill (H/P/Cosmos Pulsar 4.0, H/P/Cosmos Sports and Medical GmbH, Germany). The familiarisation trial comprised 2 x 15 min bouts of the SSEP (33) associated with the experimental trials, and an Isokinetic dynamometer protocol (comprising all testing speeds) completed pre- and post-trial.

The experimental trials comprised three soccer-specific simulations (33, 34) completed over a 5 day period, with 48h interspersing each trial. The soccer-specific protocol comprised 6 x 15 min bouts of standardised intermittent activity, with a 15min passive recovery period between the third and fourth bouts to represent half-time (HT).
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1) was based on notational analysis of match-play (28) and conducted with varying levels of gradient (22). The velocity profile was designed to replicate the clusters of high intensity activity interspersed with periods of low intensity passive and activity recovery as observed during match-play (42).

** Insert figure 1 about here **

In an attempt to control for circadian variation (35), all trials were completed in accordance with the participant’s typical match times (15:00-18:00 hours). All trials were conducted in an ambient controlled environmental chamber with temperature and humidity maintained at 20 ± 0.5 °C and 40 ± 1.5 % respectively. Participants attended the laboratory on each occasion in a 3hr post-absorptive state following a 48hr period of abstinence from vigorous exercise, alcohol, and the use of recovery strategies. Participants were also instructed to consume standardised meals between trials, wear similar apparel, and the same running shoes for each trial.

Prior to each experimental trial, a portable refractometer (Osmocheck, Vitech Scientific, West Sussex, UK) was used to ensure participants were euhydrated (urine osmolality of <700mOsm./kgH₂O). Participants were provided water at the beginning of the first trial, and were instructed to consume this ad libitum during the HT period of the protocol. Their total fluid consumption was then recorded and replicated for the subsequent experimental trials. In an attempt to minimise the influence of both the gender (44) and the number (38) of observers, only one male researcher and the participant were present during the completion of the experimental trials.

Prior to each trial, participants were also required to complete a standardised treadmill based intermittent warm-up followed by a period of self-directed stretching. The warm-up comprised the progressive completion of the running speeds associated with the SSEP and was designed
to replicate the intensities, durations, and distributions of speed changes associated with a pre-match warm up routine (17).

**Experimental Measures**

Participants were fitted with a heart rate monitor (Polar Team system, Polar Electro Oy, Kempele, Finland) and a breath by breath portable metabolic analyser (Cosmed K4b2, Cosmed, Rome, Italy) for the duration of each experimental trial. The heart rate monitor was synchronised with the metabolic analyser. Values for average heart rate (HR\textsubscript{average}), peak heart rate (HR\textsubscript{peak}), average oxygen consumption (VO\textsubscript{2}), and peak oxygen consumption (VO\textsubscript{2peak}) were quantified at rest (following a 20min period of supine rest during the application of the electromyography equipment) and for each 15 minute bout of the experimental trials. Ratings of perceived exertion ([RPE] 2), and finger-tip capillary blood lactate (BLa) concentrations (Lactate Pro, LT-1710, Arkray, Kyoto, Japan) were also measured at rest and following the completion of each 15min bout of the experimental trials. Salivary immunoglobulin A (SIgA) concentrations were recorded at rest and immediately following the completion of each experimental trial using a lateral flow device reader (IPRO lateral flow device, IPRO Interactive, Wallingford, England). All measures were completed in duplicates with the average intra-assay coefficients of variation calculated as 7.4%.

Participants were also required to provide their perceptions of lower limb muscle soreness (MS) pre- and post-trial using a visual analogue scale (VAS). The VAS scale consisted of a 10-cm continuous line with “not sore at all” on the left end of the line (0-cm) and “very, very sore” on the right end of the line (10-cm) (9). With knees shoulder-width apart, participants were asked to squat to a knee joint angle of ~90° (9). Participants were then asked to mark their MS onto the continuum. A new VAS was used for each measurement to avoid bias from the previous measurement. A score of 0-100 was quantified by measuring the distance to the nearest 0.1cm from the left end of the line to the participants reported MS.
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Global positioning system ([GPS] MinimaxX S4, Catapult Innovations, Scoresby, Australia) based accelerometry (Kionix KX94, Kionix, Ithaca, New York, USA) was continuously recorded at 100Hz to quantify tri-axial PlayerLoad™ (PL_{Total}). Planar PlayerLoad™ in the medial-lateral (PL_{ML}), anterior-posterior (PL_{AP}) and vertical (PL_{V}) movement planes were also quantified, along with their relative contributions to PL_{Total} (PL_{ML\%}, PL_{AP\%} and PL_{V\%}). All measures were calculated for each 15 minute bout of the SSEP using the Catapult Sprint software (Version 5.0.9.2; Firmware 6.75). To reduce movement artefact, the GPS device was housed in a standardised neoprene vest (Catapult Innovations, Scoresby, Australia) at the cervical region of the spine. The PL_{Total} metric has previously been shown to possess good convergent validity with measures of exercise intensity (1), good test-retest reliability (1), and good within- and between-device reliability (3).

Electromyograms (EMG) were also recorded from the dominant (defined as preferred kicking leg) bicep femoris (BF) muscle using a pair of disposable bipolar silver-silver chloride (Ag/AgCl) passive wet gel surface electrodes (inter-electrode distance 2cm) (Noraxon USA inc, Arizona, USA). Site preparation and electrode placement was in accordance with previous guidelines (20). To ensure optimal transfer of the EMG signal via Wi-Fi, the EMG system initially high pass filtered the EMG data at 10Hz and then low pass filtered the data at 500Hz. The recorded EMG signals were analysed using the manufacturer’s software (MyoResearch 3.4, Noraxon, USA). Initially, the raw EMG data was filtered to remove movement artefact using a high pass filter of 40Hz, and was then smoothed using a root mean square (RMS) smoothing factor of a 50ms time constant (19). As identified in figure 1, values for mean EMG (EMG_{mean}) were quantified every 15 minutes for a pre-determined bout of activity (10 second period encompassing the acceleration and deceleration period) at a running velocity of 25 km·hr⁻¹. The aforementioned running velocity was chosen for analysis due to the typical injury aetiology of the bicep femoris during soccer-specific activity (14). In order to ensure
repeatability of marker placement on subsequent visits; electrode positioning was marked on the skin using a permanent marker. Participants were asked to reapply the mark during the interspersing recovery periods to ensure that it remained visible.

Pre- and post-exercise eccentric knee flexor peak torque was quantified using an isokinetic dynamometer (System 3, Biodex Medical Systems, Shirley, New York, USA) at speeds of 300 and 60 deg·s\(^{-1}\) (5.25 and 1.05 rad·s\(^{-1}\)). The order of the speeds was chosen to reduce the potential fatigue effect induced by the slower isokinetic speed (17). For each testing speed, participants were instructed to perform 5 (dominant leg) maximal contractions through a range of 95-15º of knee flexion. Passive knee flexion at 60 deg·s\(^{-1}\) separated each repetition, and a rest period of 30s interspersed each set. No performance feedback was provided during any of the experimental trials. The dynamometer setup was adjusted specifically for each participant in line with the manufacturer’s guidelines. Prior to the commencement of data collection, the passive flexion of the participant’s leg was recorded to correct for the effects of gravity. The isokinetic phase (a cut-off of 1% was used to identify the isokinetic phase of the movement) of each repetition was identified, with the repetition with the highest peak torque (PT) being chosen for further analysis.

**Statistical Analyses**

The assumptions associated with a repeated measures general linear model (GLM) were assessed to ensure model adequacy, with none of the current measures violating the assumptions of the test. Where significant main effects or interactions were observed, post hoc pairwise comparisons with a Bonferroni correction factor were applied. Partial eta squared (\(\eta^2\)) values were calculated to estimate effect sizes for all significant main effects and interactions. Partial eta squared was classified as small (0.01 to 0.059), moderate (0.06 to 0.137), and large (>0.138). All statistical analysis was completed using PASW Statistics Editor 22.0 for windows.
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(SPSS Inc, Chicago, USA). Statistical significance was set at P ≤ 0.05. All data is reported as mean ± SD unless otherwise stated.

RESULTS

Physiological and Perceptual Measures

As identified in table 1, the GLM identified significant (P< 0.001) main effects for time associated with Bla (η² = 0.42), VO₂ (η² = 0.99), VO₂peak (η² = 0.98), HRaverage (η² = 0.97), HRpeak (η² = 0.98), and RPE (η² = 0.61). However, there were no significant (P> 0.05) main effects for trial, and no significant (P> 0.05) trial*time interactions associated with the aforementioned variables.

As identified in figure 2, significantly (P< .001, η² = 0.85) higher perceptions of muscle soreness post-trial (Tₚₒsₜ = 51 ± 24 a.u) when compared to pre-trial (Tₚ𝕣ₑₚ = 20 ± 19 a.u). The GLM also identified a significant (P= 0.001, η² = 0.42) main effect for trial, with significantly higher MS being recorded in the third trial (42 ± 25 a.u) when compared to the first (29 ± 29 a.u). There was however no significant (P= 0.72) trial*time interaction.

No significant main effects for time (P= 0.477) or trial (P= 0.913), and no significant interactions (P= 0.652) were identified for the SIgA data (Pre-trial 1= 107.54 ± 46.75 µg·ml⁻¹; Post-trial 1= 130.52 ± 71.61 µg·ml⁻¹; Pre-trial 2= 115.47 ± 35.85 µg·ml⁻¹; Post-trial 2= 112.35 ± 47.09 µg·ml⁻¹; Pre-trial 3= 106.85 ± 45.32 µg·ml⁻¹; Post-trial 3= 121.35 ± 65.71 µg·ml⁻¹).
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Mechanical Measures

As identified in table 1, significant main effects for time were identified for the PL<sub>total</sub> (P< 0.001, η<sup>2</sup> = 0.43), PL<sub>AP</sub> (P< 0.001, η<sup>2</sup> = 0.67), PL<sub>V</sub> (P= 0.03, η<sup>2</sup> = 0.24), PL<sub>AP%</sub> (P< 0.001, η<sup>2</sup> = 0.70), and PL<sub>V%</sub> (P< 0.001, η<sup>2</sup> = 0.56) data. There were however no significant main effects for time were identified for PL<sub>ML</sub> (P= 0.78) and PL<sub>ML%</sub> (P= 0.13). Irrespective of time and trial, mean values for PL<sub>ML</sub> and PL<sub>ML%</sub> were 51.29 ± 7.95 a.u and 23.97 ± 2.05 % respectively. No significant (P> 0.05) main effects for trial, and no significant (P> 0.05) time*trial interactions were identified for any of the PlayerLoad<sup>TM</sup> metrics.

Figure 3 depicts the time history for PT across the three trials. A significant main effect for time was identified for PT recorded at isokinetic speeds of 300 (P= 0.01, η<sup>2</sup> = 0.52) and 60 (P= 0.05, η<sup>2</sup> = 0.37) deg·s<sup>-1</sup>. Post hoc pairwise comparisons identified significantly higher pre-trial values (T<sub>300pre</sub> = 151.37 ± 31.03 Nm; T<sub>60pre</sub> = 142.33 ± 41.26 Nm) when compared to post-trial (T<sub>300post</sub> = 138.33 ± 29.51 Nm; T<sub>60post</sub> = 134.23 ± 28.87 Nm). Significant main effects for trial were also observed for PT values recorded at 300 (P= 0.02 η<sup>2</sup> = 0.35) and 60 deg·s<sup>-1</sup> (P= 0.02 η<sup>2</sup> = 0.35), with significantly higher values in the first trial (PT<sub>300</sub> = 154.17 ± 35.25 Nm; PT<sub>60</sub> = 145.61 ± 42.86 Nm) when compared to the third trial (PT<sub>300</sub> = 139.12 ± 26.23 Nm; PT<sub>60</sub> = 131.10 ± 35.38 Nm). The GLM also identified significantly lower PT values recorded at 300 deg·s<sup>-1</sup> in the second trial (141.27 ± 28.51 Nm) when compared to the first trial. There was however no significant trial*time interactions for the PT data recorded at 300 (P= 0.49) or 60 (P= 0.44) deg·s<sup>-1</sup>.

** Insert figure 3 about here **

Figure 4 depicts the time history of changes in EMG<sub>mean</sub> data recorded across the three experimental trials. Significant (P< 0.001; η<sup>2</sup> = 0.61) main effects for time were identified for the EMG<sub>mean</sub> data with significantly higher values recorded in the first half (T<sub>0-15</sub> = 108.28 ±
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22.94 µV; T₁₅₋₃₀ = 95.67 ± 28.68 µV; T₃₀₋₄₅ = 99.92 ± 30.02 µV) when compared to 60-75 minutes (77.8 ± 32.83 µV). Significantly higher values were also recorded at 0-15 and 30-45 minutes when compared to the first 15 minutes of the second half (79.3 ± 26.36 µV), and during the first 15 minute period when compared to the final 15 minute period (76.1 ± 36.04 µV). No significant main effect for trial (P= 0.59) was identified for the EMG<sub>mean</sub> data; however, there was a significant (P= 0.003, η² = 0.36) trial*time interaction. Post hoc pairwise comparisons identified significantly higher EMG<sub>mean</sub> values in the last 15 minutes of the first trial (99.97 ± 39.81 µV) when compared to the corresponding time point in the third trial (52.18 ± 17 µV). Similarly, significantly higher EMG<sub>mean</sub> values were recorded in the first 15 minutes (126.36 ± 15.57 µV) of the third trial when compared to the corresponding time point in the first trial (98.20 ± 23.49 µV).

**Insert figure 4 about here**

DISCUSSION

The aim of the current study was to assess the temporary, cumulative, and residual physical fatigue response associated with the completion of a simulated period of short-term fixture congestion. With the exception of an increase from resting values, the $\dot{V}O_2$peak and $\dot{V}O_2$ did not increase as a main effect for time; however, in support of the current SSEP the values were similar to those previously reported in the literature (43). A similar response was also identified for the BLa (23), and HR<sub>peak</sub> (28) data. In support of match-play observations, significant cumulative fatigue responses were observed for the RPE, and HR<sub>average</sub> (28) data recorded across each trial. These data, therefore, support the observed reductions in performance towards the end of soccer match-play, with an increase in physiological load suggesting a reduction in movement efficiency and an increased perception of effort. In support of this, the PL<sub>Total</sub> data had a similar magnitude of change (~5%) across the SSEP when compared to the HR<sub>peak</sub> data.
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It has previously been identified that a 2% change in PL\textsubscript{Total} is a meaningful change, with the current data being similar to that previously reported in the literature (11). Similar changes were also observed in the absolute and relative anterior posterior and vertical PlayerLoad\textsuperscript{TM} metrics. It has previously been identified that during actual match-play, players modify their velocity profiles, to maintain their capacity to preform HI activity (29). Due to the standardised nature of the protocol, these aforementioned modifications to the participant’s velocity profiles are not possible and, as such, any observed alterations in PlayerLoad\textsuperscript{TM} must be attributable to impairments in running technique and/or efficiency (1, 11, 33, 34). The current observed changes in the PL\textsubscript{V%} data may therefore be indicative of a technical modification of running technique in an attempt to conserve energy and enhance movement economy by adopting a flatter mass trajectory over each stride (11). The compensatory increase in the PL\textsubscript{AP%} data is indicative of a fatigue induced impairment in sagittal plane kinematics (33, 41), thus supporting changes in running technique (11), reductions in HI activity (28), and increased injury risk during soccer-specific activity (14). In support of previous literature (34), there was no significant difference in the aforementioned physiological, RPE, and PlayerLoad\textsuperscript{TM} metrics recorded across the successive trials. These data therefore raise questions in relation to the use of these measures in monitoring changes in residual fatigue during intense training and fixture schedules.

In contrast to previous literature (29), the SIgA data recorded in the current study did not identify a significant cumulative or residual fatigue response across successive bouts of soccer-specific activity. The current SIgA data does however support the findings of other literature (27, 40), thus suggesting that trained adult players possess the capacity to complete a period of short-term fixture congestion without impairing their immune function.
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The current study also measured MS, BF $\text{EMG}_{\text{mean}}$, and PT data. The current data suggests that MS was significantly lower post-trial, paralleled with a significant reduction in PT recorded at both 60 and 300 deg·s$^{-1}$. These findings can be attributed to the temporal pattern in muscular response to the exercise protocol, with the $\text{EMG}_{\text{mean}}$ data eliciting a fatigue response as a function of exercise duration. The velocity and activity profile of the exercise protocol emphasises a high frequency of eccentric contractions that have been associated with muscle damage, subsequent increases in MS (15) and changes in both PT and muscular activity (17, 18). Reductions in PT observed towards the end of match-play may be a result of reductions in central motor output in an attempt to offset the occurrence of peripheral fatigue (25). The cumulative fatigue response observed in the $\text{EMG}_{\text{mean}}$ and PT data supports previous studies that have assessed the mechanical response to soccer-specific activity (18, 36) and epidemiological observations of increased KF injury risk during the latter stages of match-play (14). Running has previously been identified as the primary mechanism of non-contact injuries in soccer and, as such, the current $\text{EMG}_{\text{mean}}$ data is specific to this. When considering the biarticular nature of the bicep femoris and the importance of this muscle for knee stability, then the current observed changes in muscular activity, could help explain the high injury incidence associated with knee ligamentous injuries in soccer (14). Moreover, the decrease in high speed PT also suggests a compromised capacity to complete ballistic activities, thus in turn, supporting previous observations of increased injury incidence of thigh based strains towards the latter stages of soccer match-play (14).

The current data also demonstrates a trend for altered BF muscle activity following the passive half time period, thus supporting observations of reduced performance (28), reduced muscular function (18) and increased injury risk (14) during the initial stages of the second half. The current data therefore advocates the use of half-time strategies that are designed to prevent the observed reductions in muscular function.
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In support of the physiological and PlayerLoad™ metrics, the fatigue response associated with the MS and EMG$_{\text{mean}}$ data was not significantly different in the second trial when compared to the first. These data support previous literature that has identified no change in activity profile in a second game performed less than 72 hours after the completion of an initial match (5, 31). In contrast, significantly lower fast PT data was recorded in the second trial when compared to the first. Reductions in a player’s capacity to generate PT at fast speeds may result in an increased susceptibility to injury during the completion of high intensity actions, such as accelerating and decelerating from sprinting (17). The current data, therefore, supports previous observations of increased injury risk during periods of fixture congestion (12, 13), and impaired functional capacity in the days following soccer match-play (21). It should be acknowledged that although the current population was semi-professional by definition, they did elicit similar pre and post fatigue PT values as those previously reported in professional soccer players (18), thus enabling comparisons. When considering the self-paced nature of match-play, the observed reductions in fast PT may support previous observations of reduced high intensity activity during periods of fixture congestion (16). These data also suggest that in comparison to the aforementioned measures, the measurement of fast speed PT may offer the best assessment of physical recovery during periods of short-term fixture congestion.

In support of previous observations of a change in activity profile performed in the third game of a congested weekly microcycle (31), significantly lower fast and slow speed PT values were recorded in the third trial when compared to the first. It is possible that the frequency of HI running, accelerations, and decelerations during the completion of the SSEPs could have resulted in muscle damage, particularly with the repeatedly recruited type II muscle fibres, which are more susceptible to muscle damage than type I fibres (15). The rates of fatigue associated with different fibre types may help to explain the current data, whereby fast speed PT is significantly reduced as early as the second trial, whereas the slow speed PT is not
significantly impaired until the final trial. The observed reductions in fast and slow speed PT may also be related to a decrease in the number of fibres that can be recruited to generate force (36). The observed fatigue response associated with the PT data supports the observed injury incidence during periods of fixture congestion (13), where thigh and lower limb ligamentous injuries have been shown to be most prevalent (12). Similar to the PT response, MS was significantly higher in the third trial when compared to the first trial. These data are in support of recent research that has identified that markers of muscle damage and inflammation are elevated after a second game of a three game congested microcycle (27). Measures of MS may therefore potentially be utilised to infer alterations in muscular strength, without the use of expensive and time consuming clinical and/or invasive measurement tools.

Significantly higher EMG\text{mean} values were observed during the first 15 minutes of the third trial when compared to the corresponding time point in the first trial. The current data therefore identifies a need for increased BF activity at the beginning of the third trial to achieve the same standardised workload, thus suggesting a reduction in movement efficiency. Parallel with the observed increase in muscle activity at the beginning of first trial, fast PT at the beginning of the third trial was shown to be 7% higher than that identified at the end of the second trial. The observed increases in both PT and EMG\text{mean} may be indicative of a protective mechanism associated with increased motor unit recruitment. In the same way that reductions in central motor output can occur to offset the occurrence of peripheral fatigue (25), central motor output could also be increased to elicit an increased neuromuscular effort in an attempt to compensate for the existence of residual physical fatigue.

Following the increase in muscle activity observed at the beginning of the third trial there is a compensatory decrease in the second half, with significantly lower EMG\text{mean} in the final 15 minutes period when compared to corresponding time point in the first trial. As supported by
the lack of observed main effects for trial associated with the EMG\textsubscript{mean} data, the current study suggests that hamstring muscles are capable of completing a finite amount of work during the course of a match. Subsequently, the reduction in BF muscle towards the latter stages of the third trial may be a direct result of the increase in BF muscle activity highlighted at the beginning of the trial. As such, although the increase in activity may facilitate performance and reduce injury during the first half, it might also result in an increased susceptibility to injury towards the latter stages of the second half. The deficit in hamstring strength and muscular activity is not only of concern in relation to hamstring injury risk (14) but may also correspond with a compromised capability for joint stabilization leading to increase muscular and ligamentous injuries (36).

Alterations in MS, PT and EMG\textsubscript{mean} in the third trial both support (13) and contrast (6) previous literature in relation to an increased injury risk during periods of fixture congestion. It has previously been identified that thigh and knee based injuries are significantly more prevalent when successive games are completed with $\leq$ 96 hours recovery (13) and, as such, the current data appears to mechanistically support these observations. In further support of the current data, the most prevalent injury types suffered during short-term fixture congestion are also those which are observed in non-congested fixtures (14). It could therefore be inferred that the mechanisms associated with these injuries are similar. Where previous literature has not identified any differences in injury incidence during periods of fixture congestion (6, 12), they have typically looked at prolonged periods of fixture congestion (six to eight games performed over eighteen to twenty six days). It could therefore be suggested that injury risk is more pronounced during periods of short-term fixture congestion. The aforementioned studies (6, 12) have also identified that during more prolonged periods of fixture congestion the injuries that are suffered are typically characterised by shorter lay off times than those suffered during non-congested schedules. These data therefore suggest that not only is injury incidence lower
during more prolonged periods of fixture congestion, but injury severity is also reduced. These observations may be associated with increased squad rotation practices during more prolonged fixture congestion scenarios (7).

In relation to performance, it has previously been observed that team sport players will alter their activity profiles to preserve their high intensity running capacity, and that these alterations may be more pronounced during periods of short-term fixture congestion (16). However, it is difficult to identify if these reductions in performance are indicative of the self-paced nature of match-play, or if they are a result of decreased physical capacity. The current study identified altered physical capacity across successive trials, thus supporting previous observations of reduced physical performance during periods of fixture congestion (6, 31, 39).

Although the need to standardise the activity profile within and between trials was fundamental to the current study, the use of a treadmill protocol negates the inclusion of soccer-specific utility movements which can further induce muscle damage and influence the rate of recovery post-exercise (41). As such, the current data may be considered to provide a conservative mechanical response when compared to soccer match-play, thus further reiterating the implications of the current data. Future research should attempt to investigate potential methods to either reduce the mechanical load elicited from soccer-match play, or attempt to artificially speed up the time course of mechanical recovery between successive bouts of match-play.

In summary, the current study identified that playing three games in five days had no residual effect on the physiological, immunological, and PlayerLoad™ response. The mechanical response was characterised by insufficient recovery of PT recorded at 300 deg·s⁻¹ with 48h recovery, with the third trial eliciting further detriments in MS, BF muscular activity, and PT recorded at both 60 and 300 deg·s⁻¹. The interaction of muscular response to soccer-specific
Physical response to fixture congestion in soccer

activity, the subsequent reduction in muscular strength, and the perception of muscular soreness suggests a biomechanical and muscular emphasis with residual fatigue. The current data therefore supports previous observations of reduced physical performance (6, 31, 39) and increased injury risk (13) during periods of fixture congestion.

PRACTICAL APPLICATIONS

The current data has implications for the practitioner in relation to the micro management, monitoring, and design of training and competition schedules. Consideration of squad rotation strategies (6) during congested schedules is advocated. The current study suggests that players possess the capacity to complete two games with < 72 hours recovery, but that the risk of injury is increased if a 3rd game is completed at this frequency. Where < 72 hours precedes the 3rd (and all subsequent) game in a congested period, the implications for performance and injury risk should be considered.

Squad rotation is an option where the playing roster is sufficiently large, but a more considered approach to substitutions also has merit. Rather than excluding a player from a game to facilitate recovery, the substitution strategy may more proactively consider the physical demands. The temporal responses presented in the current study suggest that substitutions made no later than the 60th minute of the match may have a beneficial effect on reducing the fatigue response associated with match-play.

Whilst substitutions are regulated by governance of the sport, the three permitted in soccer are relatively lower than other intermittent team sports and ‘football’ codes. An increased frequency of player inter-change would further facilitate the opportunity to respond proactively to the physical demand associated with match-play, and particularly during periods of fixture congestion. It has previously been identified that regular interchanges reduce the incidence of KF strain injuries in intermittent team sport athletes, with this being attributed to the reduction
in total match volume and subsequent cumulative and residual fatigue (32). Over the past few years the International football association board (IFAB) has identified the potential merit of the aforementioned strategies by approving the use of additional substitutions during extra-time of English domestic cup matches, and the use of an unlimited “return sub” ruling at grass root soccer.

Aside from strategically resting players, either by exclusion or strategic substitution, the current study also highlights the need to develop functional capacity of the KF musculature and resistance to fatigue. It is common practice in soccer for muscular screening to take place in a non-fatigued state—thus negating the association between fatigue and injury risk. The current data suggests that there should be an increased emphasis placed on screening athletes during congested fixture and/or training schedules. Identifying individual players who are most susceptible to periods of high frequency competition (or training), would help to refine an individualised approach to strength and conditioning regimes.

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REFERENCES


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FIGURE AND TABLE LEGENDS

Figure 1. Schematic representation of a single 15min bout of the SSEP and the data upon which it is based. The dashed line indicates the EMG data collection period.

Figure 2. Perception of muscle soreness data recorded across the three experimental trials. a Denotes a significant main effect for trial with the first trial.

Figure 3 Eccentric knee flexor peak torque (60 [□] and 300 deg·s⁻¹[■]) recorded across the three experimental trials. a Denotes a significant main effect for trial with the corresponding data in the second and third trial. b Denotes a significant main effect for trial with the corresponding data in the first trial.

Figure 4. Mean electromyography recorded at 25km·h⁻¹ across each of the three experimental trials. a Denotes a significant trial*time interaction with the corresponding time point in the first trial

Table 1. Time history of changes in a number of physical measures irrespective of trial.
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<table>
<thead>
<tr>
<th>Time (mins)</th>
<th>Rest</th>
<th>0-15</th>
<th>15-30</th>
<th>30-45</th>
<th>45-60</th>
<th>60-75</th>
<th>75-90</th>
</tr>
</thead>
<tbody>
<tr>
<td>BLa (mmol·L⁻¹)</td>
<td>1.3 ± 0.3</td>
<td>2.4 ± 1.0 a</td>
<td>2.9 ± 1.2 a</td>
<td>3.3 ± 1.9</td>
<td>3.2 ± 1.8</td>
<td>3.2 ± 1.6 a</td>
<td>3.6 ± 2.4 a</td>
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<tr>
<td>HRpeak (beats·min⁻¹)</td>
<td>75 ± 12</td>
<td>157 ± 15 a</td>
<td>157 ± 12 a</td>
<td>159 ± 12 a</td>
<td>158 ± 15 a</td>
<td>159 ± 12 a</td>
<td>164 ± 14 a</td>
</tr>
<tr>
<td>HRaverage (beats·min⁻¹)</td>
<td>67 ± 12</td>
<td>136 ± 13 a</td>
<td>141 ± 12 ab</td>
<td>144 ± 13 abc</td>
<td>139 ± 13 a</td>
<td>144 ± 13 abde</td>
<td>148 ± 13 abc</td>
</tr>
<tr>
<td>V̇O₂peak (mL·kg⁻¹·min⁻¹)</td>
<td>10.07 ± 1.85</td>
<td>40.50 ± 5.38 a</td>
<td>40.12 ± 5.50 a</td>
<td>40.49 ± 6.33 a</td>
<td>41.04 ± 5.71 a</td>
<td>39.58 ± 4.64 a</td>
<td>41.15 ± 5.38 a</td>
</tr>
<tr>
<td>V̇O₂ (mL·kg⁻¹·min⁻¹)</td>
<td>6.72 ± 1.44</td>
<td>29.03 ± 3.94 a</td>
<td>28.84 ± 3.71 a</td>
<td>28.92 ± 3.91 a</td>
<td>28.95 ± 3.85 a</td>
<td>28.71 ± 3.89 a</td>
<td>29.10 ± 3.96 a</td>
</tr>
<tr>
<td>RPE (a.u)</td>
<td>11 ± 2</td>
<td>12 ± 2</td>
<td>12 ± 2 bc</td>
<td>12 ± 2 b</td>
<td>13 ± 2 bc</td>
<td>14 ± 2 bc</td>
<td></td>
</tr>
<tr>
<td>PLtotal (a.u)</td>
<td>207.14 ± 19.64</td>
<td>209.33 ± 17.30</td>
<td>213.21 ± 21.64</td>
<td>216.22 ± 19.56 bc</td>
<td>215.29 ± 21.56 b</td>
<td>218.02 ± 20.35</td>
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</tr>
<tr>
<td>PLAP (a.u)</td>
<td>51.32 ± 9.29</td>
<td>52.66 ± 9.06</td>
<td>54.41 ± 9.95</td>
<td>55.95 ± 10.74 b</td>
<td>57.34 ± 11.05 bc</td>
<td>58.78 ± 10.72 bc</td>
<td></td>
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<tr>
<td>PLV (a.u)</td>
<td>105.00 ± 10.16</td>
<td>105.55 ± 9.76</td>
<td>107.36 ± 11.80</td>
<td>108.53 ± 10.57 b</td>
<td>106.94 ± 12.88</td>
<td>107.27 ± 11.43</td>
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</tr>
<tr>
<td>PLAP% (%)</td>
<td>24.74 ± 3.63</td>
<td>25.25 ± 3.48</td>
<td>25.48 ± 3.60</td>
<td>25.82 ± 3.90</td>
<td>26.61 ± 3.99 bcde</td>
<td>26.91 ± 3.83 bcde</td>
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</tr>
<tr>
<td>PLV% (%)</td>
<td>51.00 ± 4.39</td>
<td>50.55 ± 4.28</td>
<td>50.51 ± 4.52</td>
<td>50.31 ± 4.21</td>
<td>49.74 ± 4.42 bde</td>
<td>49.30 ± 3.98 bc</td>
<td></td>
</tr>
</tbody>
</table>

a-b-c-d-e-f denote significant differences with Rest, 0-15, 15-30, 30-45, 45-60, and 60-75 respectively.
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