Manuscript Title Lower-limb muscle excitation, peak torque and external load responses to a 120-minute treadmill-based soccer-specific simulation

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Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical approval

Institutional review board approval was granted for this study.

Acknowledgements

The authors gratefully appreciate the efforts of the participants.

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Abstract

Purpose The aim of this study was to investigate thigh musculature excitation and torque generation in response to soccer-specific exercise incorporating an extra-time (ET) period. **Methods** Twelve semi-professional soccer players performed 120-min treadmill-based soccer-specific exercise. Surface electromyography (EMG) signals for the rectus femoris (EMG_{RF}) and biceps femoris (EMG_{BF}) were measured as the mean response across a pre-determined 10-second sprint bout during each 15-min block of exercise. Peak eccentric torque of the knee flexors (eccKF) and concentric torque of the knee extensors (conKE) were recorded across angular velocities of 60, 180 and 270 deg·s^{-1} immediately pre- and post-exercise. Tri-axial PlayerLoad™ (PL-T) was monitored throughout exercise and defined across vertical (PL-V), anterior-posterior (PL-AP) and medial-lateral (PL-ML) planes of motion. **Results** A reduction in normalised EMG_{RF} amplitude was evident at 105–120 min, versus 0–15 min (−12.5%; p=0.037), 15–30 min (−12.5%; p=0.047) and 45–60 min (−14%; p=0.030). Peak torque of the eccKF was significantly reduced from pre- to post-exercise at 60 (−7.7%; p=0.018), 180 (−10.5%; p=0.042) and 270 deg·s^{-1} (−7.5%; p=0.034). A main effect for time was identified for PL-T (p<0.010), PL-V (p=0.033) and PL-AP (p<0.010). **Conclusions** These findings suggest that muscle excitation of the rectus femoris is reduced during ET, accompanied with a deficit in the torque generation of the knee flexors following 120 min of soccer-specific activity. Practitioners should adequately condition players for the additional ET period by incorporating exercises into training schedules that develop fatigue-resistant eccentric hamstring strength to minimise injury risk.

Keywords EMG · Strength · Efficiency of movement

Introduction

Soccer is characterised by an intermittent activity profile, involving rapid changes of direction, accelerations, decelerations and sprints. While soccer is traditionally competed over 90 min, several tournaments (e.g., FIFA World Cup, UEFA Champions League and English FA Cup) proceed to an additional 30 min period known as extra-time (ET) when scores are tied. Notably, during the previous four FIFA World Cup competitions 33% of knockout phase matches have proceeded to ET, with 50% requiring 120 min of match-play at the 2014 FIFA World Cup competition (Harper, Fothergill, West, Stevenson, & Russell, 2016). Match-play observations suggest that external workload (i.e., total and high-speed distance covered, as well as number of sprints, accelerations and decelerations) is reduced relatively ($m \cdot min^{-1}$) during ET compared to the initial 90 min (Peñas, Dellal, Owen, & Gómez-Ruano, 2015; Russell, Sparkes, Northeast, & Kilduff, 2015). It is likely that these performance decrements are exercise induced as a result of peripheral and central fatigue-related processes (Brownstein et al., 2017; Thomas et al., 2017).

Peripheral fatigue corresponds to a reduced muscle contractility occurring at the neuromuscular junction/within muscle, whereas central fatigue originates at the central nervous system; reducing activation of motor neurons, which can manifest as diminished muscular activation (Brownstein et al., 2017; Thomas et al., 2017). Previous research suggests that simulated and actual match-play elicits a deficit in knee extensor force production (peripheral fatigue), alongside a reduction in neural drive from the central nervous system (central fatigue) (Brownstein et al., 2017; Thomas et al., 2017). It has also been observed that 120 min of simulated soccer causes peripheral and central patterns of fatigue, with additional reductions in voluntary muscle activation identified during ET, indicating fatigue during this additional period may be primarily of central origin (Goodall et al., 2017). However, the exact within-match fatigue source has yet to be investigated across 120 min of soccer-specific exercise. This may be because measuring the precise origin of fatigue is difficult due to the complex interaction of the biological and psychological processes, and as such several indirect methods of assessment are often used such as subjective scales (Harper et al., 2017; Field et al., 2020), physical performance indices

(Goodall et al., 2017; Thomas et al., 2017) and muscle activation measurements (Rahnama et al., 2006; Page et al., 2019).

The reduction in lower-limb force production following prolonged intermittent activity has previously been linked with modified twitch contractility properties that can impair torque production and changes in muscle excitation measured through an electromyography (EMG) signal (Gibson, Lambert & Noakes, 2001; Rahanama et al., 2003; Rahanama, Lees & Reilly, 2006). It has been demonstrated that 90-min treadmill-based soccer activity elicits muscle contractile deficits in knee extensors and flexors (Rahanama et al., 2003), and changes in muscle excitation of the lower limbs, measured via surface EMG (Rahanama, Lees & Reilly, 2006). A reduction in excitation of the lower-limb muscles appears to be further exacerbated by repeated bouts of a soccer simulation with minimal recovery, mimicking fixture congestion (Page et al., 2019). Thereby, while there appears to be a detrimental effect of 90 min of soccer-specific exercise and simulated fixture congestion on muscle excitation, no previous studies have assessed the potentially deleterious impact of an ET period on muscle excitation measured using surface EMG.

PlayerLoad™ (PL), a software-derived metric (Catapult Innovations, Australia), is an applied and sensitive measure that accounts for the demanding speed changes (i.e., accelerations and decelerations) accrued across three-planes of motion (Barret et al., 2016; Nicolella, Torres-Ronda, Saylor, & Schelling, 2018). Previous studies incorporated standardised bouts of activity throughout simulated match-play and identified an increased PL during the latter stages of 90 min (Page et al., 2015), which has shown to persist into the ET period (Field et al., 2020). An increase in PL values across standardised bouts of motorised treadmill-based exercise is indicative of impaired movement efficiency (Field et al., 2020). This has previously been linked with an increased energy cost to perform a fixed bout of activity, causing players to become biomechanically less efficient whilst running (Wilk, Nau & Valero, 2009). Although, such adjustments in running gait are performed subconsciously to conserve energy, this can result in a reduction in lower extremity stiffness and an increased injury susceptibility of soft tissues (Hughes & Watkins, 2008). However, microsensor technology placed at the upper trunk has yet to be validated in relation to quantifying injury-risk and sport-specific movements (Chambers, Gabbett, Cole, & Beard, 2015). Similarly, it is contentious as to whether the within-match changes in PL and discrete planar contributions are reflective of running patterns in relation to specific lower-limb musculature (Cormack, Mooney, Morgan, & McGuigan, 2013; Verheul et al., 2019). Additionally, the devices that are used to measure PL in an applied soccer setting, typically involves placement in a vest worn by players. Therefore, PL has more application in a sporting context due to the ease of measurement compared with less feasible alternatives such as EMG and isokinetic peak torque measures. Therefore, assessing PL metrics in conjunction with excitation and torque responses of major lower-limb muscles appears warranted to provide practically compatible alternatives. This will in turn facilitate our understanding of whether PL changes correspond with local fatigue patterns of thigh musculature during prolonged intermittent activity.

Due to various contextual factors and the changing demands of soccer match-play, it is often difficult to identify mechanisms associated with reductions in physical performance and injury-risk profiles. Consequently, soccer simulations are used to control the external influences associated with soccer matches (Castellano, Blanco-Villaseñor & Alvarez, 2011). To overcome the self-pacing elements associated with soccer match-play, treadmill-based simulations have been used to standardise the activity profile throughout exercise, such that the responses are a result of a reduced physical capacity as opposed to alterations in the activity profile (Grieg et al., 2008; Page et al., 2016). Accordingly, treadmill-based soccer simulations using fixed bouts of activity might provide a better tool than free-running simulations to investigate the change in physical capacity (Page, Marrin, Brogden, & Greig, 2015). In support of treadmill-based running, when comparing a free running simulation that incorporates changes of direction with treadmill running; they appear to be largely comparable in relation to kinematic, kinetic, spatiotemporal, musculotendinous and muscle activation outcomes (Azidin, Sankey, Robinson, & Vanrenterghem, 2013; Van Hooren et al., 2019).

In light of the above, the aim of the present study was to assess thigh musculature excitation and peak torque production, as well as changes in PL metrics in response to 120-min of treadmill-based soccer-specific exercise. It was hypothesised that ET would reduce the degree of excitation and torque production of the thigh musculature, and that this additional 30 min period would elicit increases in PL values.

Material and Methods

Participants

Institutional ethical approval was granted, and the study adhered with the most recent version of the Declaration of Helsinki. Twelve semi-professional soccer players (mass: 74 ± 8 kg; height: 179 ± 3 cm; age: 22 ± 3 years; maximal oxygen uptake [$\dot{V}O2_{max}$]: 59 ± 7 ml·kg·min^{-1}) provided written informed consent. An *a priori* power calculation was undertaken (GPower v3.1; Germany) which deemed a sample size of 11 sufficient based on 95% power ($1-\beta$), an alpha (α) of 0.05, and a large effect size (Cohen's d = 1.1) to detect significant differences for EMG based on previous data (Page et al., 2019). Participants were recruited on the basis they were male with > 5 years of soccer experience and had no medical contraindications to exercise (e.g., musculoskeletal injury). Participants visited the laboratory on three separate occasions and were to avoid strenuous exercise external to the study throughout this testing period. Participants refrained from caffeine for 12 h and alcohol 24 h prior to testing. Mean participant energy and macronutrient intake was recorded across the 24 h period prior to testing through use of weighed food diaries (energy: 1998 ± 490 Kcal, carbohydrates: 218 ± 66 g, protein: 111 ± 45 g, fat: 75 ± 19 g).

Preliminary visits and study design

The preliminary visit involved taking anthropometric measures of height (SECA 213 portable stadiometer, SECA, Germany) and mass (SECA 875 electronic flat scale, SECA, Germany), and the completion of a $\dot{V}O2_{max}$ test. This involved a graded ramp test until volitional exhaustion in order to assess participant's eligibility. A secondary visit was used for familiarisation, which included a full habituation of experimental procedures including the completion of a 120 min simulation. This was preceded by a standardised treadmill-based warm-up that consisted of 10 min of aerobic activity with multiple sporadic speed changes and a dynamic stretching sequence. One week thereafter, the third and final visit involved the main trial. This included the 120 min soccer simulation following the completion of the same warm-up as described above. During the main trial, *ad libitum* intake of a carbohydrate–electrolyte solution was permitted (Lucozade Sport, GlaxoSmithKline, Gloucestershire, UK). Participants ingested a mean of 729 ± 28 ml.

Soccer simulation

The soccer simulation was performed on a treadmill (h/p/ cosmos pulsar® 3p: h/p/cosmos sports & medical GmBH, Germany) consisting of eight 15 min periods, with a HT period interspersing the 3rd and 4th and a 5 min passive rest interspersing the 6th and 7th periods. The protocol was validated alongside 90-min of match-play (Page et al., 2015). Two additional bouts were incorporated for the ET period, with the PL responses demonstrating very strong reliability over 120 min (Pearson's correlation coefficient = 0.75–0.92; Field et al., 2020). Participants completed 16.26 km during the 120-min protocol (Field et al., 2020), with the activity profile designed to replicate the velocities, durations, and frequencies of speed changes associated with match-play (Page et al., 2015). The simulation repeated the same fixed activity profile every 15 min (Table 1) and data were analysed accordingly. The activity profile was standardised to minimise the potential inertial delay associated with instantaneously reaching the desired velocities on a treadmill (Yao et al., 2019). The 120 min simulation was divided into eight epochs including: E1 (00:00–14:59 min), E2 (15:00–29:59 min), E3 (30:00–44:59 min), E4 (45:00–59:59 min), E5 (60:00–74:59 min), E6 (75:00–89:59 min), E7 (90:00–104:59 min), E8 (105:00–119:59 min).

INSERT TABLE 1

Surface electromyography

The EMG signal of the rectus femoris (EMG_{RF}) and biceps femoris (EMG_{BF}) of the dominant leg (defined as the preferred kicking leg) were recorded using wireless surface EMG sensors (Inter-electrode distance 10mm; Trigno™, Delsys, USA). In accordance with recommendations for surface

EMG sensor placement procedures (Stegeman, Blok, Hermens & Roeleveld, 2000), the skin was shaved and cleaned prior to electrode attachment to reduce impedance. To ensure that movement artefacts were minimal, the electrodes were carefully taped to the skin using surgical tape. The EMG activity was recorded at 2000Hz and processed using Delsys software. In accordance with the methods of Page et al., (2019), the EMG signal was recorded over a single 10-second action within each 15-min bout of the soccer simulation to capture the myoelectric activity for the entire acceleration and deceleration phase (running velocity of 25 $km{\cdot}h^{-1}$). An example of the EMG_{RF} and EMG_{BF} data obtained from a single participant across the initial (E1) and final (E8) 10-second sprint bout is provided in Figure 1. This specific action was chosen in accordance with the injury etiology of the knee flexors being mainly associated with the deceleration stage during the late swing phase of sprinting (Chumanov et al., 2012; Setuain, Lecumberri & Izquierdo, 2017).

INSERT FIGURE 1

To process the EMG data, the raw EMG signals were low pass filtered at 500Hz and high pass filtered at 10Hz to preclude movement artefacts, using a Butterworth fourth order filter. The signal was then rectified and smoothed using a root mean square (RMS) smoothing factor with a 50-ms time constant (Hader et al., 2014). The mean RMS value was obtained for each 10-second recording to quantify the mean EMG (the degree of muscle excitation) for each bout of the soccer simulation (Rahnama et al., 2006; Page et al., 2019). The amplitude mean was analysed as opposed to the single peak data point because it is a more stable reference value, and less sensitive to duration differences across intervals (Konrad, 2005). The EMG signal recording for the pre-determined sprint during E1 was used as the reference value for normalisation of E2–E8. Similar to previous methods (Pincivero, Aldworth, Dickerson, Petry & Shultz, 2000), a decision was taken to normalise against the first sprint because participants were likely in a less fatigued state during the initial bout of activity and sprint measures are more functionally relevant than an isolated maximal voluntary contraction. The normalised EMG data were expressed as a percentage of the mean value obtained during E1. This method was undertaken in accordance with recommendations for the normalisation of EMG amplitudes (Besomi et al., 2020).

Isokinetic testing

Peak eccentric torque of the knee flexors (eccKF) and peak concentric torque of the knee extensors (conKE) were measured immediately post warm-up/pre-exercise and post-exercise using the Cybex HUMAC Norm isokinetic dynamometer with HUMAC2009 software version 0.8.4 (CSMI, USA). The agonist-antagonist relationship of the eccKF/conKE are often used to detect imbalances with a low ratio being associated with an aetiological risk factor for hamstring injury (Rahnama, Lees & Bambaecichi, 2005). Knee flexor strength deficits as a result of fatigue are commonly observed during the latter stages of simulated and actual match-play (Greig, 2008; Small, McNaughton, Greig & Lovell, 2010). During the eccentric phase of contraction, injury risk is heightened as fatigued muscles are more likely to suffer stretch injuries due to an impaired capacity to resist over lengthening (Croisier et al., 2008; Opar, Williams & Shield, 2012). The preferred kicking leg was tested at three respective angular velocities of 180, 270 and 60 deg·s^{-1}. This specific order was used to reduce potential fatigue induced by slower velocities (Greig, 2008). One set was performed for each speed (i.e., three sets) which included five repetitions completed through a range of 0–90° (0° equal to full extension) and were interspersed by a 30-second passive rest period. Once seated, participants were secured, and the contralateral limb was isolated as per manufacturer guidelines. To account for the influence of the participants limb weight to subsequent torque generation, the HUMAC2009 software automatically performs a gravity correction procedure. This involves the participant's passive limb being weighed at anatomical zero (defined as full knee extension). Due to several factors, the participants are not always able to achieve a limb position which is exactly horizontal to the ground. As such, the software corrects for angular error from the horizontal using the following equations:

Limb weight = Torque measured/ Sine(angle)

During the test, the limb weight contribution is then calculated as:

Torque correction value = Limb weight calculated above * Sine(angle)

Note. The (angle) refers to the angle from horizontal at which the limb weight measure was initially performed.

The limb is working against gravity (i.e., eccentric knee flexor and concentric knee extensor work involves an upward motion) and, as such, the limb weight contribution value for each participant is subsequently added as a constant value to their torque curves.

PlayerLoad™

A portable accelerometer (Kionix KXPA4, Kionix Inc., Ithaca, NY, USA), integrated within a GPS-unit (OptimEye S5, Catapult Innovations, Scoresby, Australia), continuously recorded PL data at 100Hz. PlayerLoad™ is a vector magnitude calculated as the square root of the instantaneous rate of change in acceleration across individual planes of motion and divided by a scaling factor (Graham, Zois, Aughey & Duthie, 2019). The same device was used between simulations as intra-device test re-test has demonstrated good reliability as evidenced by low coefficient of variations (CV: 0.01–3.0%) and intra-class-correlations (ICC: 0.77–1.0; Nicolella, Torres-Ronda, Saylor & Schelling (2018)). This device was placed directly inferior to the 7th cervical vertebrae inside the pouch of a tightly fitted garment to reduce excessive movement. Tri-axial data were recorded across the vertical (PL-V), anterior-posterior (PL-AP) and medial-lateral (PL-ML) vectors. The subsequent summation of the planar contributions provided a combined value for PL total (PL-T). These metrics were defined as the accumulated mean value across each 15 min block of exercise and expressed as arbitrary units.

Statistical Analysis

Eight EMG data points were absent across six participants due to technical difficulties with the wireless recording, though all other data were presented for analyses. Linear mixed modelling (LMM) is appropriate for repeated measures designs that involve random and fixed level factors with missing data; assuming data are missing at random (Di Salvo, Gregson, Atkinson, Tordoff & Drust, 2009). As such, LMM analysis was employed for the current study. Initially, the normality of residuals was checked through visually examining q-q plots, boxplots and histograms, and residuals > 3.0 SD from the mean were removed. Within-subject LMM with both fixed (i.e., *time* [E1–E8]) and random (i.e., *participant*) factors were assessed. The model fit was determined using Akaike's information criterion (AIC) with the most suitable for all variables deemed the first order auto-regressive (AR-1) repeated covariance structure for the repeated measures. Main effects for time were identified *post hoc* using Fisher's LSD with 95% confidence intervals (CI) for the difference reported where significance was detected. Unless otherwise specified, data are expressed as mean ± SE and were analysed using SPSS version 26.0 (SPSS Inc., Chicago, IL, USA). Alpha was accepted as $p \leq 0.05$ prior to analyses.

Results

Significant reductions were identified for normalised EMG_{RF} between E8 (87.5 ± 4.3%; 95% CI = 78.9 to 96.2) versus E1 (−13%; 100 ± 4%; 95% CI = 92 to 102%; 95% CI for diff = −24 to −1; $p = 0.037$), E2 (−12%; 99 ± 4%; 95% CI = 91 to 108%; 95% CI for diff = −23 to −2; $p = 0.047$) and E4 (−13%; 100 ± 4%; 95% CI = 92 to 108%; 95% CI for diff = −23 to −1; $p = 0.030$). No significant time effects were observed for EMG_{BF} ($p = 0.73$).

As illustrated in Figure 2A, a significant reduction in peak torque of 10.5% was observed for $eccKF_{180}$ from pre- (162.3 ± 9.0 Nm; 95% CI = 143.2 to 181.3 Nm) to post-exercise (145.2 ± 9.0 Nm; 95% CI = 126.1 to 164.3 Nm; 95% CI for diff = −33.9 to −0.8; Nm; $p = 0.042$). Peak torque was significantly reduced by 7.5% for $eccKF_{270}$ from pre- (159.0 ± 9.0 Nm; 95% CI = 139.4 to 178.6 Nm) to post-exercise (147.2 ± 9.0 Nm; 95% CI =127.6 to 166.7 Nm; 95% CI for diff = −22.4 to −1.3 Nm; $p = 0.034$), and by 7.7% for $eccKF_{60}$ from pre- (159.5 ± 7.9 Nm; 95% CI = 142.4 to 176.6 Nm) to post-exercise (147.2 ± 7.9 Nm; 95% CI = 142.4 to 176.6 Nm; 95% CI for diff = −21.7 to −3.0 Nm; $p = 0.018$; Figure 2B and 3C). No differences were observed for the conKE data recorded pre- (180 $deg \cdot s^{-1}$ = 150.8 ± 7.7 Nm; 270 $deg \cdot s^{-1}$ = 116.8 ± 5.6 Nm; 60 $deg \cdot s^{-1}$ = 193.7 ± 11.0 Nm) when compared to post-exercise (180 $deg \cdot s^{-1}$ = 153.0 ± 7.8 Nm; $p = 0.441$; 270 $deg \cdot s^{-1}$ = 114.8 ± 5.6 Nm; $p = 0.493$; 60 $deg \cdot s^{-1}$ = 183.3 ± 11.0 Nm; $p = 0.062$).

INSERT FIGURE 2

As outlined in Table 2, significant main effects for time were identified for PL-T ($p < 0.010$), PL-V ($p = 0.038$) and PL-AP ($p < 0.010$), though no timepoint differences were detected for PL-ML ($p = 0.094$).

Mean (± SD) percentage contributions of PL-T were 53 ± 3 % (PL-V), 24 ± 3 % (PL-AP) and 23 ± 2 % (PL-ML) throughout the 120 min simulation.

INSERT TABLE 2

Discussion

The main purpose of this study was to assess thigh musculature excitation and peak torque production in response to 120 min of soccer-specific exercise. In line with the study hypotheses, an additional ET period elicited a reduction in rectus femoris muscle excitation and decreased eccentric knee flexor peak torque from pre-to-post 120 min of soccer-specific exercise. Increments in PL values were identified as a function of exercise duration which was further increased during ET. However, no changes in indices of isokinetic knee extensor peak torque were observed, nor did the degree of muscle excitation change throughout the exercise simulation in the biceps femoris.

Normalised EMG_{RF} amplitudes were reduced during ET, though excitation deficits were not associated with significant impairments in ConKE following the 120-min simulation. These results are consistent with previous findings demonstrating that patterns of potentiated knee-extensor twitch force/voluntary activation do not reflect deficits in maximal knee extensor force capacity following ET (Goodall et al., 2017). Therefore, it is considered likely that the knee extensor fatigue experienced during ET occurs centrally along the pathway and could be linked to a reduced neural drive and/or excitation contraction coupling as opposed to within-muscle contractile failure and/or substrate depletion. Duration dependent central fatigue development has also been observed in response to varying exercise modalities (Place, Maffiuletti, Martin & Lepers, 2007; Thomas et al., 2015). However, the reduction in muscle excitation during high velocity sprinting in response to an additional ET period is a novel finding and implies that central fatigue (indicated through reduced surface EMG signals) may progressively become a more prominent limiting factor in response to an increased exercise duration.

The reduction in EMG_{RF} amplitude during a fixed work rate soccer-specific treadmill simulation is in line with the findings of Rahnama et al., (2006). However, the lack of change in EMG_{BF} amplitude across 120 min in the present study was an unexpected finding and contradicts the work of Rahnama et al. (2006) who reported a reduction in EMG_{BF} following a soccer-specific treadmill simulation. The conflicting results might be partly attributed to differences in the soccer simulation protocol and EMG measurements. Rahnama et al., (2006) used a soccer simulation which involves a different activity profile (i.e., a disproportionate amount of high-speed running) to the present study. They also measured the mean RMS value of EMG amplitudes in a separate protocol at three time points (pre, half-time and post simulation) as opposed to during the protocol itself, potentially allowing aspects of central fatigue development to dissipate. The lack of EMG amplitude inhibition for EMG_{BF}, despite the reduction in EMG_{RF} in the current investigation, implies that neural fatigue may occur sooner in the quadriceps than in the hamstrings during soccer-specific exercise. This is perhaps due to the repetitive braking forces incurred during rapid decelerations, for which the quadriceps are a primary muscle group (Hewit, Cronin, Button & Hume, 2011). As a result, it is possible that if the hamstrings were incapable of performing these functions, injury risk may have been increased during rapid deceleration movements.

The lack of change for EMG_{BF}, despite eccKF strength reductions following ET implies the strength capacity of the hamstrings were impaired, but still maintained a similar level of muscular activation from E1 (100 ± 3%) to E8 (103 ± 4%). Conversely, it has previously been purported that impairments in contractile properties of the muscle require an elevated neural drive to maintain a constant running velocity, which are reflected with larger EMG amplitudes within working muscles (Pincivero et al., 2000). However, the torque reductions observed are possibly explained by a protective mechanism which acts to regulate extracellular damage by enforcing a temporary energy restriction to limit the recruitment of muscle fibres. This may be imposed because muscle fibres are unable to disregard cerebral and neural commands which control the recruitment of motor units and subsequent muscle contraction (Baird, Graham, Baker & Bickerstaff, 2012). Another plausible explanation may be that musculature that play a role in flexing the knee were measured as a unit, and as such, the maintenance of activation in the bicep femoris may suggest that the observed changes in peak torque may be due to impairments in other such muscles — semitendinosus, semimembranosus, sartorius, gracilis, popliteus and gastrocnemius — that contribute to knee flexion. It is also possible that as the biceps femoris causes external rotation of the knee joint (Opar, Williams & Shield, 2012), the lack of change of direction movements involved with treadmill running, may have resulted in a conservative response when compared to match-play in the sense that the biceps femoris may be stressed to a greater extent from changes of direction tasks.

A reduction in eccKF torque production was observed in the present study, irrespective of the isokinetic testing speed. However, conKE appear to maintain their torque capacity from pre- to post-120 min, despite a reduction in neural drive. Considering the standardised nature of the protocol, other musculature must compensate for the reduced neural drive of the quadriceps in order to maintain the propulsion needed to perform the exercise protocol. Therefore, it is possible that the reductions in knee flexor peak torque are due to an inhibited quadriceps activation and subsequent changes to how other musculature operate. It is also likely that differences in muscle composition can explain the disparity in peak torque maintenance between muscles. For instance, while the precise anatomical properties that predispose hamstrings to peripherally derived measures of reduced strength are unclear, it has been purported that fibre type distribution and muscle architecture are factors that may contribute (Opar, Williams & Shield, 2012). It should also be considered that symptoms of muscle damage and soreness are exacerbated in response to eccentric versus concentric modes of exercise (Mirzayev, 2017). Additionally, hamstring strains typically occur when the lengthening demands of the muscle exceeds the tissue strength limits, with fatigued muscles able to absorb less energy before failure versus unfatigued muscles (Opar, Williams & Shield, 2012; Coratella et al., 2012). Eccentric strength exercises reportedly shift the optimum length-tension curve (i.e., towards greater lengths/joint angles), thus reducing hamstring strain injuries at extended joint positions (Brughelli & Cronin, 2008). A 12-week Nordic hamstring protocol delivered bi-weekly before training, resulted in a greater degree of improvement at extended muscle lengths versus post-training (Lovell et al., 2018). As such, implementing such preventative exercises within training programmes appears warranted. Practitioners should develop the eccentric component of the hamstrings to increase player resistance to fatigue-induced torque deficits and increase the muscle length at which eccentric hamstring torque development is attained in order to reduce hamstring injury susceptibility during the ET period.

The magnitude of the reduction in eccKF torque generation from pre- to post-exercise at 270 and 60 $deg \cdot s^{-1}$ were similar with that of previous work with corresponding measures at 300 and 60 $deg \cdot s^{-1}$ following 90 min of a treadmill simulation (Page et al., 2019). The deficit in eccKF torque between pre- and post-exercise suggests that soccer players are unable to attain eccentric peak torque of the knee flexor musculature following 120 min of soccer-specific activity. However, as evident in Figure 2, large inter-individual variability was present for eccKF with changes ranging from −34% to +21% (depending on the isokinetic speed) between pre- and post-exercise. This suggests that muscle contractile fatigue mechanisms are highly dependent on the individual and thus, torque deficits should be interpreted on an individual level. Likewise, considered within a practical setting, these data may have implications for squad rotation and ET substitutions, especially since a contemporary rule change has been implemented permitting a fourth substitution be made during the ET period of match-play in major tournaments (Hills et al., 2020). For players unable to be replaced during ET, carbohydrate provision is recommended where practical to attenuate reductions in physical performance.

Reductions in muscle torque generation and subsequent compensatory adjustments in gait can manifest as changes in movement patterns (Jonkers et al., 2003). Therefore, the compromised capacity of the players in this study to maintain knee flexor peak torque may partially elucidate the increase in PL-AP postural sway. Specifically, the mechanism likely involved an impaired ability for the hamstrings to maintain hip extension (i.e., an upright trunk posture), coupled with a potential antagonistic dominance of the quadriceps musculature in flexing the hip. Additionally, hamstrings are most susceptible to injury during the late swing phase of sprinting due to lengthening across both hip and knee joints (Chumanov, Schache, Heiderscheit, & Thelen, 2012), and are possibly in a comprised state following 120 min of soccer-specific exercise. Therefore, it may be pertinent for players to employ pacing strategies to reduce the impact of fatigue-induced reductions in knee flexor strength during matches that proceed to ET. However, players were unable to ‘self-pace’ during the current study (i.e., the treadmill dictated the activity profile), thus, a reduced physical capacity may have instead manifested through increases in PL metrics. Furthermore, the ~7.5–10.5% deficit in knee flexor torque production identified pre- to post-exercise supports an increased hamstring injury propensity and a compromised joint stability (Page et al., 2019), elicited by 120 min of exercise. Therefore, players not conditioned to manage the additional demands of ET, may be at an increased susceptibility to suffer an acute musculotendinous rupture during this additional 30 min period.

Similar to the reduced physical performance capacity observed in the latter stages of 120-min matches (Peñas et al., 2015; Russell et al., 2015), PL-T has demonstrated increases during ET when compared to a number of the preceding fixed bouts of treadmill-based simulated soccer exercise (Field et al., 2020). Though inferential statistics were not carried out to assess correlations, the increases in PL appears to partially correspond with reductions in excitation (i.e., EMG_{RF}) and peak torque data (i.e., eccKF). Therefore, these data provide an initial step towards providing evidence that suggests PL metrics are reflective of EMG and peak torque lower-limb thigh musculature changes. However, prospective validation of PL metrics is required that focus intently and establish the extent to which changing planar contributions reflect EMG lower-limb patterns during both treadmill and free-running soccer simulations.

While this study offers novel insight into muscle excitation and torque production throughout 120 min of soccer exercise, there are some methodological limitations present within the current research. Firstly, the absence of additional isokinetic peak torque measures for the thigh musculature (i.e., eccentric knee extensors and concentric knee flexors) may be considered a limitation, especially given the role of the knee extensors during eccentric knee flexion upon ground contact (Paquette, Peel, Schilling, Melcher, & Bloomer, 2017). Reductions in strength of the eccentric knee extensors could lead to kicking related injuries and therefore is an important future avenue of research. While EMG was measured at 15-min time intervals throughout the simulation, peak torque was only measured pre and post the 120-min protocol. This could be deemed a limitation, however, additional measures on the isokinetic dynamometer at 15-min intervals would have invalidated the soccer-specific fatigue response and, as such, was considered inappropriate for this study. Though participants covered distances (16.26 km) and performed the number of sprints (*n* = 56) comparable with an actual 120 min match (Russell et al., 2015; Winder, Russell, Naughton, & Harper, 2018), the pre-arranged nature and lack of kicking, jumping and tackling actions involved with simulations will inevitably impact peripheral and central patterns of fatigue. Notwithstanding the considerable experimental control of our research, its lack of ecological validity yields difficulty when extrapolating the data to match-play scenarios considering the number of contextual variables that influence soccer match performance (Castellano, Blanco-Villaseñor & Alvarez, 2011).

Conclusion

To summarise, thigh musculature excitation and peak torque production, and PL responses were investigated in response to 120 min of soccer-specific activity. These novel data suggest that muscle excitation of the rectus femoris was reduced during ET, though no notable change was evident in the biceps femoris throughout 120 min of soccer activity. The torque generating capability of the knee flexors was reduced post 120 min compared with baseline assessments. Increases in PL were evident as a function of exercise, which was further increased during the additional period of ET. It is recommended that exercises are implemented within the weekly training schedule to develop resistance to fatigue-induced knee flexor torque deficits in order to limit injury risk during matches that progress to the ET period. Likewise, carefully considered interventions should be orchestrated following ET matches to promote recovery and enhance physical performance in subsequent matches.

Figure Captions

Figure 1. An example of the EMG signal recorded for an individual participant for the 10-second sprint performed during E1 and E8 within the soccer-specific simulation.

Figure 2. Individual eccKF peak torque values across angular velocities of 180 (A), 270 (B) and 60 deg·s^{-1} (C). * indicates significant difference from pre- to post-exercise. Dash lines with open circles represent mean eccKF responses.

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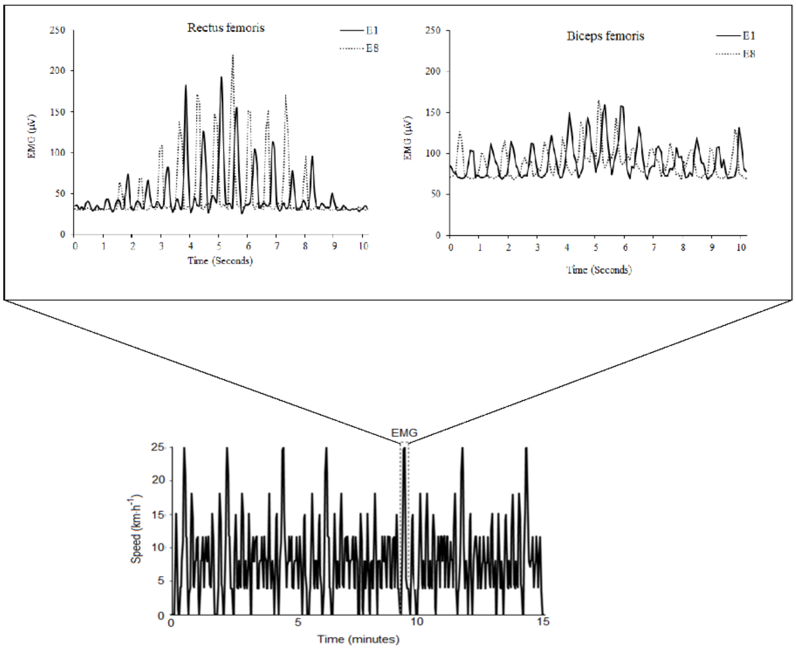
Table 1. The activity profile of an individual 15-minute bout of the soccer-specific exercise simulation			
Locomotion category (velocity)	**Number of repetitions**	**Duration (s)**	**Gradient (%)**
Standing (0 $km \cdot h^{-1}$)	29	7.0	1.0
Walking (4 $km \cdot h^{-1}$)	65	6.4	1.0
Jogging (8 $km \cdot h^{-1}$)	53	3.0	1.0
Low speed running (11.6 $km \cdot h^{-1}$)	48	2.6	1.0
Moderate speed running (15 $km \cdot h^{-1}$)	17	2.2	2.0
High speed running (18 $km \cdot h^{-1}$)	12	2.1	2.0
Sprinting (25 $km \cdot h^{-1}$)	7	2.0	2.5

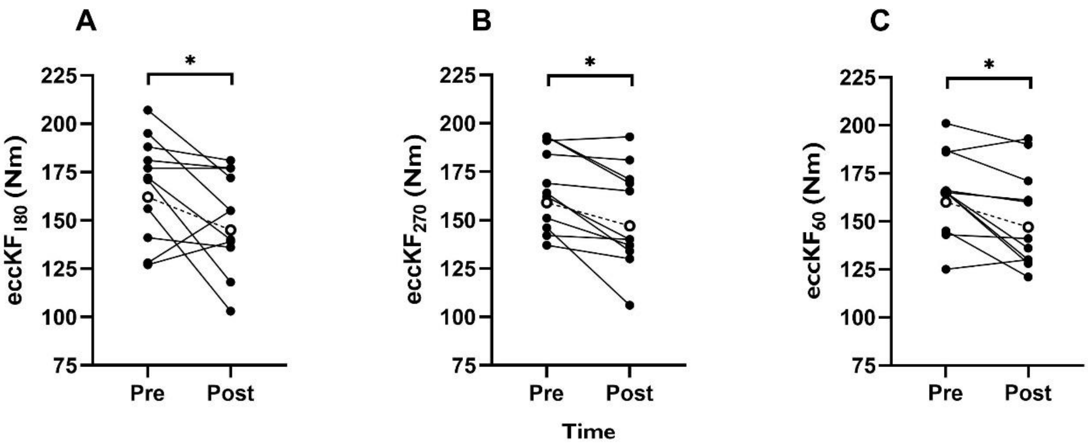
Table 2. Muscle excitation and PlayerLoad™ responses throughout the 120 min soccer simulation (Mean ± SE)

	Time							
Variable	E1 (0—15 min)	E2 (15—30 min)	E3 (30—45 min)	E4 (45—60 min)	E5 (60—75 min)	E6 (75—90 min)	E7 (90—105 min)	E8 (105—120 min)
EMG_{RF} (%)	100 ± 4	99 ± 4	97 ± 4	100 ± 4	95 ± 4	93 ± 4	95 ± 4	87 ± 4 95% CI = −24 to −1[a]; −23 to −2[b]; −23 to −1[d]
EMG_{BF} (%)	100 ± 3	100 ± 3	102 ± 3	104 ± 3	103 ± 3	99 ± 4	101 ± 3	103 ± 4
PL-T (a.u)	218 ± 7	221 ± 7 95% CI = 1 to 7[a]	221 ± 7	226 ± 7 95% CI = 2 to 13[a]; 1 to 8[c]	230 ± 7 95% CI = 5 to 18[a]; 3 to 14[b]; 4 to 13[c]; 1 to 7[d]	231 ± 7 95% CI = 6 to 20 [a]; 3 to 16 [b]; 5 to 16 [c]; 1 to 10 [d]	234 ± 7 95% CI = 3 to 13[a]; 3 to 12[b]; 4 to 12[c]; 2 to 9[d]; 1 to 6[e]; 1 to 4[f]	236 ±7 95% CI = 3 to 14[a]; 2 to 12[b]; 3 to 12[c]; 1 to 9[d]
PL-V (a.u)	115 ± 5	116 ± 5	116 ± 5	119 ± 5 95% CI = 1 to 5[c]	121 ± 5 95% CI = 1 to 9[a]; 1 to 8[b]; 2 to 7[c]	121 ± 5 95% CI = 1 to 11[a]; 1 to 9[b]; 1 to 9[c]	124 ± 5 95% CI = 3 to 14[a]; 2 to 12[b]; 4 to 12[c]; 1 to 9[d]; 1 to 6[e]; 1 to 4[f]	124 ± 5 95% CI = 3 to 14[a]; 2 to 12[b]; 3 to 12[c]; 1 to 9[d]
PL-AP (a.u)	52 ± 3	54 ± 3 95% CI = 1 to 3[a]	54 ± 3 95% CI = 1 to 3[a]	54 ± 3 95% CI = 1 to 4[a]	56 ± 3 95% CI = 2 to 5[a]; 1 to 4[b]; 1 to 4[c]; 1 to 3[d]	57 ± 3 95% CI = 2 to 6[a]; 1 to 4[b]; 1 to 4[c]; 1 to 3[d]	57 ± 3 95% CI = 3 to 7[a]; 1 to 5[b]; 2 to 5[c]; 1 to 4[d]	58 ± 3 95% CI = 4 to 8[a]; 2 to 6[b]; 3 to 6[c]; 2 to 5[d]; 1 to 3[e]; 1 to 3[f]
PL-ML (a.u)	50 ± 3	51 ± 3	51 ± 3	52 ± 3	53 ± 3	53 ± 3	54 ± 3	54 ± 3

Note. EMG_{RF} = Mean electromyography for rectus femoris; EMG_{BF} = Mean electromyography for bicep femoris; PL-T = PlayerLoad total; PL-V = PlayerLoad vertical; PL-AP = PlayerLoad anterior-posterior; PL-ML = PlayerLoad medial-lateral; CI 95% = 95% confidence intervals for the difference.

[a–g] Indicates significant differences from E1–E7 ($p \leq 0.05$), respectively.


Rectus femoris
E1
E8
EMG (µV)
Time (Seconds)
Biceps femoris
E1
E8
EMG (µV)
Time (Seconds)
EMG
Speed (km·h-1)
Time (minutes)


A
B
C
*
*
*
eccKF180 (Nm)
eccKF270 (Nm)
eccKF60 (Nm)
225
200
175
150
125
100
75
Pre
Post
Time