

Match-play, training workloads and sensorimotor and neuromuscular performance of elite young soccer players

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ABSTRACT

Purpose: The purpose of this study was to assess sensorimotor and neuromuscular performance capabilities over an in-season microcycle in early-career professional soccer players and to examine the relationship with training and match-play workload. **Methods:** Sensorimotor and neuromuscular performance capabilities (isometric knee extensor: force replication error, peak force, electromechanical delay, rate of force development) of 12 professional soccer players were assessed over a 7-day period. Training and match-play workload was also recorded over the same period for each player (high-intensity running distance). Fluctuations in sensorimotor and neuromuscular performance and workload variables were analysed. **Results:** There was evidence of fluctuations in sensorimotor and neuromuscular performance capability over the microcycle that reached statistical ($p < .005$) and practical (18.1% [baseline-to-peak]) significance alongside heterogeneity in training and match workload (264% [coefficient of variation], $p < .0005$). Some temporal congruence among fluctuating patterns of intra-microcycle training and match-play load and concomitant electromechanical delay performance was noted ($p < .005$). Asynchronous responses were observed for peak force, but rate of force development and force replication error capabilities were unchanged during the microcycle. **Conclusion:** While some neuromuscular performance capabilities fluctuate over an in-season microcycle and are influenced partially by high-intensity running workload, sensorimotor performance capabilities were unchanged during the microcycle.

Keywords: Performance analysis, Force error, Muscle activation, Strength, Training workload, Match-play workload.

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INTRODUCTION

Effective sensorimotor (SM) performance is regulated in part by neuromuscular (NM) performance capabilities involving static (ligaments, joint capsule, cartilage) and dynamic (feedback and feedforward control over skeletal muscle) componentry (Riemann and Lephart, 2002a). Mechanistic interactions between NM fatigue and compromised SM performance have been implicated in compromised capability within the latter stages of both halves during match-play (Ekstrand et al., 2011). This expectation is corroborated indirectly by laboratory-induced fatigue detrimentally affecting proprioceptive acuity in high-level soccer players during an ankle joint repositioning task (Mohammadi and Roozdar, 2010) and by match-play decreasing postural stability in elite young soccer players (Brito et al., 2010). Similarly, research shows coincidence amongst reduced force-matching acuity, exercise-induced muscle damage (Proske et al., 2004) and reduced NM performance following eccentric exercise in healthy non-athletic populations (Twist et al., 2008). In contrast, a routine training session in elite youth soccer players failed to induce fatigue-related impairments to postural control (Gioftsidou et al., 2011), while a 90-minute treadmill protocol, designed to replicate the physical demands of match-play, failed to alter single leg stability performance (Greig and Walker Johnson, 2007). Thus the literature has only partially illuminated the extent to which evidence of compromised SM and NM performance from simulations and restricted episodes of soccer match-play might calibrate directly to what could be expected within the wider environment of professional soccer. For example, while match-play appears to provoke substantive strength impairment for up to 72 hours (Cohen's d , 0.7) (Silva et al., 2018; Rampinini et al., 2011), temporally-congruent data for SM performance remains elusive.

The in-season period in professional soccer comprises a series of competitive microcycles incorporating training, match-play and the post-match recovery epoch (Owen et al., 2013). Within each microcycle, periodisation strategies maximise stress and recovery, with the goal of maintaining peak performance leading into competition (Martin-Garcia et al., 2018). The primary aim of this study was to characterise SM and NM performance capabilities over an in-season, competitive microcycle in elite young soccer players. This was accomplished by using an array of relevant metrics and documenting the concomitant training load, through the lead-up to match-play, the event itself and during the recuperative epoch. Given the potential for elevated workloads involving high-intensity running to provoke a reduced neuromuscular capacity (Goodall et al., 2017; Griffin et al., 2010; Cormack et al., 2008) a secondary aim was to explore the extent of congruence amongst fluctuating patterns of intra-microcycle training and match loads, SM performance and NM responses. It was hypothesised that there would be correspondence amongst oscillations of intra-microcycle training workload and SM and NM performances, with a nadir in performance at the end of match-play. In so doing, we hoped to inform practitioners about the potential vulnerability of elite players to diminished NM and SM performance.

METHODS

Participants

Twelve early career professional soccer players (age: 19.2 ± 1.1 years; height: 183.3 ± 6.1 cm; body mass: 76.1 ± 7.8 kg) (defenders = 4, midfielders = 4, forwards = 4), from the development squad (DEV) of a Scottish Premiership football club gave written informed consent to participate in the study. The physical profile of the squad is detailed in Table 1.

Design

To assess patterns of SM and NM performance over an in-season microcycle, data were collected from each of 12 players over a single training microcycle within the same phase of the competitive season (mid-season).

To ensure data was collected within a standardised time frame (within 15 minutes of the cessation of the relevant training or match-play session), this data was accumulated over a series of three separate microcycles, with each microcycle incorporating a match against a different professional team and involving a random selection of four players completing all assessments. The microcycle comprised of standardised training sessions and a match (Table 2a) for each participant. Training volumes were comparable across the three microcycles (Table 2b). Assessments were undertaken at baseline on the day prior to the match (MD-1), on the match-day [immediately post-match] (MDpost) and on the second (MD+2) and third (MD+3) days after the match. Excepting MDpost, assessments occurred at the same time each morning (± 1 hour) mitigating circadian variation and always prior to any physical exertion. All twelve players completed a minimum of 60 minutes of match-play in their designated match and participated in all training sessions within the microcycle. Prior to the study, ethical approval was granted by the institutional ethics committee (Queen Margaret University, Research Ethics Panel, Governance and Quality Enhancement) and conformed fully to the declaration of Helsinki.

Table 1. Mean (\pm SD) of physical performance profiling results from in-season assessment point.

Physical Capacity	Test	Mean (\pm SD)	Descriptive Rating*
Aerobic	Yo-Yo IE1	2576 (\pm 278) m	Moderately High
Speed	20 m Sprint	2.88 (\pm 0.09) s	Normal
Strength	Relative Back Squat 4RM	105 (\pm 15) % body mass	Moderately High

Note. *Based on unpublished comparisons from 5 seasons of profiling similar cohorts.

Table 2a. Microcycle schedule.

Day	Day Relative to Match-day (MD)	Training Session 1 Description	Training Session 2 Description	Experimental Assessment Timing
Monday	MD-1	Football session	N/A	Pre-training
Tuesday	MD	Match-play	N/A	Post-match
Wednesday	MD+1	Rest day	N/A	N/A
Thursday	MD+2	Football session	Full body strength	Pre-training
Friday	MD+3	Football session	Lower limb power	Pre-training

Table 2b. Daily high intensity running workload during microcycle trials.

Day Relative to Match-day (MD)	Microcycle 1	Microcycle 2	Microcycle 3
MD-1	203 m	241 m	140 m
MD	623 m	737 m	769 m
MD+2	149 m	121 m	88 m
MD+3	49 m	92 m	20 m

Procedures

All sensorimotor and neuromuscular assessments were carried out on a custom-built dynamometer, incorporating a load cell (range: 3 kN; Tedea-Huntleigh, Cardiff, UK; technical error, recorded: ± 0.95 N, 95% confidence limits, as described previously (Clancy et al., 2021).

The volitional neuromuscular assessment consisted of 3 maximum isometric knee extension trials (30° knee flexion) from which peak force (PF), rate of force development (RFD) and electromechanical delay (EMD) were derived. All assessments involved the participants' dominant leg, as determined by their preferred kicking leg. RFD was calculated as the average rate of force increase between 25% and 75% of PF.

Concomitant electromyographic activity (EMG) was recorded from the m. vastus lateralis during the estimation of PF as described previously, using bipolar surface electrodes that were parallel to the orientation of the muscle fibres. EMD was defined as the time delay between the onset of electrical activity and the onset of force. The latter were defined as the first points in time where the recording signals for the EMG and load cell systems consistently exceeded the 95% confidence limits of background electrical noise amplitude, respectively (Minshull et al., 2009).

After a standardised warm-up, consisting of progressive sub-maximal efforts (perceived 50 %, 75 %, 95 % of PF), and then muscular relaxation, participants were instructed to rapidly and forcefully extend the knee against the immovable lever arm of the dynamometer (~3 s), which was repeated twice more (30 s rest-recovery).

Sensorimotor performance was defined as the bias or constant force error (FE) when matching a 'blind' target force, specified as 50 % of a player's daily peak force to simulate on-field peak power (Zatsiorsky and Kraemer, 2006), with lower scores reflecting better SM performance (Peer and Gleeson, 2018). Each participant was acquainted with his concealed target force during two familiarisation trials that preceded testing (Gleeson et al., 2013). No feedback regarding performance was given to the participants during the test. The best response of two trials was used for subsequent analysis. The equation used to compute force error (FE) is outlined in Equation 1.

Equation 1. Force Error.

$$FE = \left[\frac{(\text{observed performance score} - \text{target performance score})}{\text{target performance score}} \right] \times 100 \%$$

Quantification of Training/Match Workload

Training workload (WL) was calculated for every field training session and match played over the 6-week period using a GPS (Catapult Sports, Melbourne, Australia) sampling at a frequency of 10 Hz.

Post-session, data were downloaded using the manufacturer's software and then exported and stored on a custom-built spreadsheet. The metric used to monitor player match-play and training WL over the microcycle was total high intensity running distance (all running over 19.5 km·h⁻¹).

Single-measurement reproducibility associated with the NM and SM performance indices PF, RFD, EMD and FE for the knee extensors of participants was 5.5%, 26.3%, 6.7% and 12.3%, respectively (coefficient of variation). For each measure of SM and NM performance, the participant's mean trial response of three was used.

Statistical analysis

Group means ± standard deviations (SD) described outcome scores. Pearson's product-moment correlation coefficients (*r*) were used at baseline to assess any dependency amongst indices assessing SM and NM performance (PF, RFD, EMD and FE).

Separate single-factor (time [MD-1: MDpost; MD+2; MD+3]) analyses of variance (ANOVAs), with repeated measures, tested hypotheses relating to PF, RFD, EMD, and FE (SPSS Vn. 23, IBM SPSS Illinois, USA). Intra-microcycle changes in match-play and training WL were analysed similarly. *A priori* planned reverse Helmert orthogonal difference testing located anticipated time-specific effects. Assumptions underpinning the

use of ANOVA parametrically were checked and any violations countered using data transformation (\log_{10}) and Greenhouse-Geisser (G_G) adjustments. Statistical significance was accepted at $p < .05$.

Congruence amongst fluctuating patterns of intra-microcycle match-play and training WL and SM (FE) and NM performance responses (PF; RFD; EMD) was examined using players standardised (z-score) data and separate univariate factorial (mode [WL; SM performance response; NM performance response]*time [MD-1: MDpost; MD+2; MD+3]) ANOVAs, with repeated measures on both factors. Temporal correspondence amongst dose-response patterns was indicated by an absence of factorial interaction. *A priori* planned reverse Helmert orthogonal difference and polynomial trend analyses characterised the expected intra-microcycle oscillatory patterns of fluctuation in players' match-play and training WL and concomitant adaptation of SM and NM performance.

A priori experimental design sensitivity estimation offered an approximate statistical power of 0.8 for avoiding intrusion of type-II errors for a medium relative size of change (Cohen's d , 0.5) in the study's primary outcome, FE, at the study's end-point (MDpost), requiring an approximate cohort' sample size of $n = 8$ (www.sportsci.org).

RESULTS

Group mean (\pm SD) intra-microcycle scores for NM and SM outcomes of PF, RFD, EMD and FE are shown in Figures 1(a), 1(b), 1(c) and 1(d), respectively. At baseline (MD-1), there were modest relationships amongst indices of SM and NM performance (FE with PF; r , - 0.60; $p < .05$; PF with RFD; r , 0.62; $p < .05$), suggesting that each offered an independent perspective on players' intra-microcycle performance capabilities.

There was a trend towards an overall significant difference in intra-microcycle PF performance capability characterised by a decline and subsequent restoration of performance ($F_{(3,33)} = 2.7$; $p = .06$). However, planned *a priori* hypothesis testing showed that the reduction between MD-1 and MDpost contributed most to the overall variation over time ($F_{(1,11)} = 12.1$; $p < .005$) (Figure 1(a)). The evidence for intra-microcycle fluctuations in PF performance was emphasised by analysis of z-scores in which variance in PF amongst participants was controlled statistically ($F_{(3,33)} = 3.1$; $p < .05$), with *a priori* planned comparisons showing depressed performance capability on MDpost relative to MD-1 ($F_{(1,11)} = 9.1$; $p < .01$). Relative to baseline (MD-1), impairment in absolute PF was substantive (ES: 0.32 [MDpost] and 0.40 [MD+2]).

By contrast, analysis of RFD absolute performance scores suggested that force-time dependent NM capability remained unchanged (2460 ± 1280 N·s⁻¹; *ns*) during the intra-microcycle period (Figure 1(b)).

Intra-microcycle EMD performance capability was characterised by a decline (18.1% reduction compared to baseline [33.2 ± 5.8 ms]) and subsequent restoration of performance ($F_{(3,24)} = 10.8$; $p < .0005$) (Figure 1(c)). Planned *a priori* hypothesis testing showed that relative to baseline (MD-1), a substantial impairment in EMD at MDpost (ES: 1.1; ($F_{(1,8)} = 9.5$; $p < .05$), followed by recovery at MD+2 ($F_{(1,8)} = 17.4$; $p < .001$) and MD+3 ($F_{(1,8)} = 10.1$; $p < .01$), which either matched or modestly surpassed baseline levels (ES: 0.9 and 1.9, respectively), contributed most to the overall significant changes in EMD.

Force error remained constant across the microcycle (13.3 ± 10.5 %; *ns*) implying that SM performance capability was preserved over the microcycle duration (Figure 1(d)).

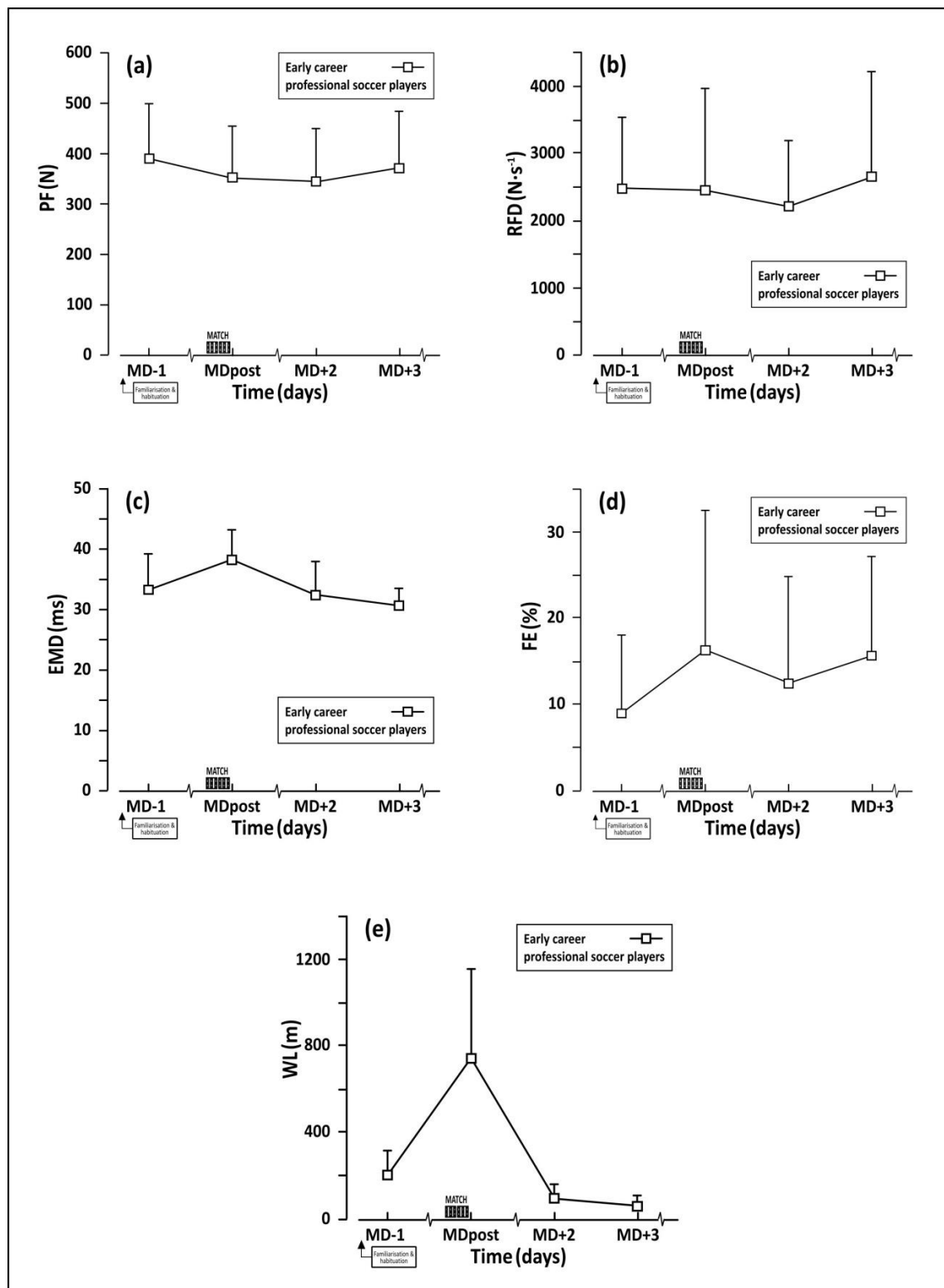


Figure 1. Group mean (\pm SD) intra-microcycle scores for NM and SM outcomes of PF (a), RFD (b), EMD (c) and FE (d) associated with the knee extensors of early career professional soccer players, with concomitant match-play and training WL (e) (high intensity running distance, over 19.5 km·h⁻¹) recorded at assessment times MD-1, MDpost, MD+2 and MD+3.

Significant fluctuations amongst match-play and training WL were observed within the microcycle ($F_{(1.1,12.1)} = 31.0$; $p < .005$), with planned *a priori* hypothesis testing showing that the greatest player exposure to high intensity running occurred on match-day (264 % greater than baseline [MD-1]; ES: 1.8) ($F_{(1,11)} = 27.8$; $p < .0005$) (Figure 1(e)).

Players' standardised (z-score) data was used to explore for congruence amongst fluctuating patterns of intra-microcycle match-play and training WL and concomitant SM and NM performance responses. Factorial interaction for WL with PF showed that patterns of intra-microcycle fluctuation amongst the amount of high intensity running during match-play and training and strength-related NM performance were incongruent over time ($F_{(3,33)} = 19.0$; $p < .0005$; Figure 2). *A priori* planned reverse Helmert difference analyses showed that the interaction associated with the period of increased match-play WL compared to baseline (MD-1) and correspondingly depressed strength capability ($F_{(1,11)} = 55.2$; $p < .0005$), together with diminished WL towards the end of the microcycle and concomitant restoration of strength performance ($F_{(1,11)} = 12.6$; $p < .01$), contributed most to the interactive incongruence between WL and this index of NM performance.

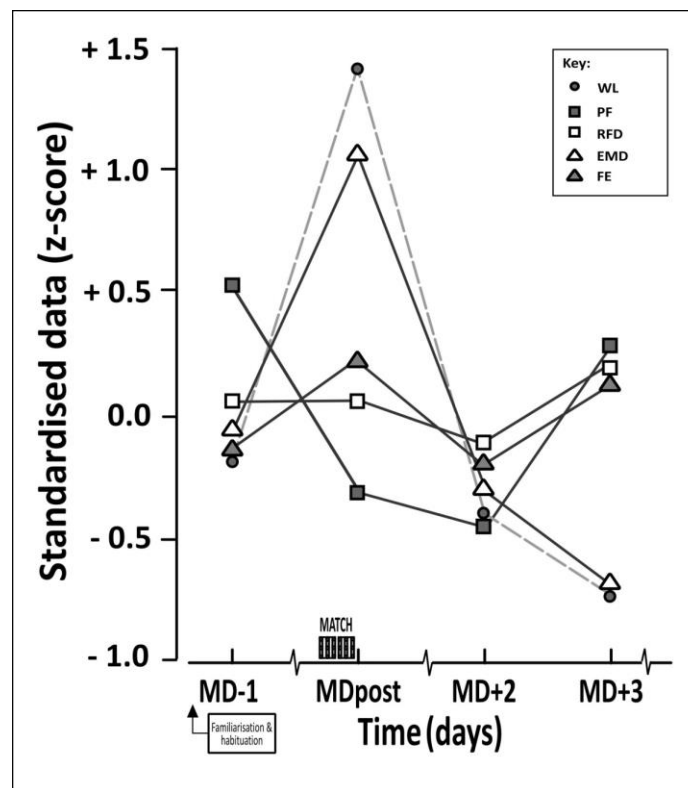


Figure 2. Group mean intra-microcycle standardised scores for indices of match-play and training WL (closed circle) and corresponding indices of SM (FE [filled triangle]) and NM performance (PF [filled square]; RFD [open square]; EMD [open triangle]) of the knee extensors of early career professional soccer players recorded at assessment times MD-1, MDpost, MD+2 and MD+3.

Similarly, temporal incongruence was noted over time for WL with the force-time dependent index of RFD ($F_{(3,33)} = 9.3$; $p < .0005$; Figure 2). Planned difference analyses characterising RFD responses, which had remained constant over the microcycle, and concomitant initial ($F_{(1,11)} = 24.4$; $p < .0005$) and end ($F_{(1,11)} =$

7.7; $p < .05$) fluctuating patterns in players' WL underscored intra-microcycle asynchrony between RFD and WL metrics.

Incongruence was also noted for WL and the SM index of FE ($F_{(3,33)} = 8.3$; $p < .0005$), with correspondingly influential and divergent patterns at the start ($F_{(1,11)} = 9.0$; $p < .05$) and end ($F_{(1,11)} = 10.6$; $p < .01$) of the microcycle.

EMD showed a non-significant (*ns*) factorial interaction in its response to WL over time, and thus a convergent but inverse pattern of WL and neuromuscular response, given that higher EMD scores reference increasing muscle activation latencies. A *priori* planned polynomial trend analyses primarily characterised the intra-microcycle oscillatory patterns in players' match-day and training WL and concomitant EMD as cubic curvilinear responses ($F_{(1,8)} = 113.9$; $p < .0005$).

DISCUSSION

The main finding of this study was that sensorimotor performance appeared to be unaffected within an in-season training microcycle. In contrast, whether expressed in absolute or standardised units, PF performance in the knee extensors was diminished substantially immediately following match-play (MDpost; ES: 0.34) and at MD+2 (ES: 0.40) compared to baseline (MD-1). The latter reduction, which corresponds to an 11.1% loss in strength is consistent with previous evidence in the same musculature following exposure to acute match-play (15%) (Goodall et al., 2017). Elevated levels of high-intensity running during match-play involving high eccentric demands of soccer actions have been hypothesised to cause damage and inhibited performance to portions of the lower limb musculature (Hortobágyi et al., 1998). A restoration of baseline levels of PF performance at MD+3 (trivial ES: -0.17) was consistent with previous research describing restoration of NM performance at 72 hours post-match play (Nédélec et al., 2012) and supports the current recommendations to reduce the volume and intensity of training for 48 hours post-match (Martin-Garcia et al., 2018).

Impaired EMD performance (18.1%) immediately following match-play suggests that game-related exercise stress disrupted post-synaptic processes such as the transmission of force through the series elastic component and alterations within the excitation-contraction coupling process (Howatson, 2010). Comparable research evidence in elite male soccer players is lacking, but arduous soccer-activities increased EMD latencies by 58.4% (De Ste Croix et al., 2015) in female players, who have previously demonstrated prolonged EMD latencies (Blackburn et al., 2009; Granata et al., 2002; Zhou et al., 1995).

High-intensity athletic performance requires the rapid uptake of slack within the musculotendinous unit to facilitate rapid force expression (Van Hooren and Bosch, 2016). Sub-optimal EMD within soccer match-play or training has the potential to negatively impact physical performance capability or render soccer players vulnerable to ligamentous injury (Gleeson et al., 1998a), given that a limited time-frame exists in which to overcome potentially harmful dynamic forces. A more rapid restoration of capabilities to baseline levels of performance for EMD compared to PF may reference speculatively the former's greater relative biological importance to functionality in sports.

In a similar way, RFD remaining unaffected during the microcycle may indicate independence for this metric amongst recovery dynamics or mechanisms of fatigue-related resistance, whereby preservation of force-time related aspects of NM performance have been prioritised. A corollary may involve antecedent training influences having depressed RFD performance at the microcycle's commencement, with moderated responses subsequently. Indeed, this study's finding may conflict with that involving diminished RFD performance following exercise-induced muscle damage (Howatson, 2010).

Given that selected NM capabilities diminished over the course of the microcycle, the absence of a concurrent reduction in SM performance appears paradoxical. Components of the SM system contributing to the knee extensors' muscular efferent activation and force capacity showed diminished performance and a potential for compromised functional capacity, including for example, inflated joint vulnerability to injury. Nevertheless, concomitant intra-microcycle preservation of FE may imply that the knee joint's afferent sensory apparatus compensated for the negative impact of the physiological stresses within match-play and training.

Expectations for impaired SM performance within the microcycle derive from conflicting evidence of disruption (Brito et al., 2012) and maintenance (Gioftsidou et al., 2011) of performance and accumulating non-football-related research in which high intensity eccentric exercise elicited disruption of proprioception and precision of movement control (Proske et al., 2004; Twist et al., 2008). Speculatively, any match-play and training-related threats to musculoskeletal stability might provoke relative homeostasis in SM performance as a mechanism of innate biological protection.

Evidence for intra-microcycle heterogeneity in WL was demonstrated statistically for high intensity running distances and centred around a more than 2-fold greater volume during match-play compared to that within episodes of lead-in or recovery training. This finding is reflective generally of microcycle periodisation in which physical stress within the training content is managed to optimise stress, adaptation and recovery leading in to match-play (Owen et al., 2017). Intra-microcycle high intensity running distances showed large variation amongst players (as demonstrated by a within-group coefficient of variation ($\pm 61.8\%$)). Although this is surprising given that the players largely participated in the same sessions and match-play, it is likely to be at least partially reflective of the different positional demands and suggests that further exploration of an individualised or positional approach to WL tracking is justified. This finding is consistent with recent research showing peak demands (high speed running intensity) within training and competition varying by as much as 50% between centre-backs and wide-attackers (Abbott et al., 2018).

Players' intra-microcycle standardised scores, which moderated inter-player heterogeneity, facilitated direct comparisons amongst outcomes, including WL, and explored their temporal congruence. The complex oscillatory NM performance that had been noted for PF during the microcycle ($p < .0005$) was temporally incongruent with patterns observed for WL ($p < .005$; Figure 2). Thus, PF responses, as the net outcome amongst antecedent acute and chronic training loads, muscular damage, physiological restoration dynamics and adaptation mechanics, appeared to be independent of WL during the microcycle. Similarly, intra-microcycle responses of FE and RFD to WL fluctuations were also temporally incongruent, but FE and RFD performances had remained at a constant level, indicating limited possibilities for synchrony amongst shared mechanistic influences. In contrast, EMD had shown congruent temporal responses with intra-microcycle WL (Figure 2), and this synchrony, together with similar patterns of standardised effect, may reference an important mechanistic linkage, albeit inversely mirrored, in which peak WL levels that are associated with match-play, coincide with a nadir in EMD latencies. While NM performance's sensitivity to fluctuations in high intensity training and match workload has been noted previously (Cormack et al., 2008), the current study offers novel data linking high intensity running workload with EMD.

The extent of physiological disruption to homeostasis imposed by acute bouts of training stress, even when moderated by effective designs within periodisation, alongside match-play, is highly likely to be influenced by antecedent fitness capacity, which in turn, is influenced by prior chronic workload and genetic predisposition. It is worth noting that due to the timing of the study (mid-season), a period of acquisition of high levels of fitness had already occurred, with participants well prepared for the demands of high loads within training

and competition. Indeed, previous research has outlined the mediating influence of fitness on enhanced recovery (Johnston et al., 2015) and the athletic profile of the current participants (Table 1) may support this notion.

Overall, the significant temporal congruence shown amongst complex intra-microcycle oscillatory patterns for WL with EMD, but not for PF, together with preserved performance capabilities for RFD and FE, suggest that heterogeneous acute WLs have the potential to diminish some NM capabilities. The findings, which largely conflict with the study's prior expectations for congruence amongst SM and NM indices with WL, may have implications for optimising intra-microcycle periodisation strategies. Specifically, given the finding of diminished EMD capability following match-play, highlighting reduced functional stability of the knee joint, a shift in training focus during this recovery epoch from physical conditioning to recovery or technical drills, is recommended.

A limitation of the present study is the small sample size ($n=12$), although this is common in studies of players at a professional level. As alluded to earlier, it could be argued that a single joint force-matching trial is too simplistic to comprehensively capture complexities within soccer-related SM performance. While this approach to assessment mimics a clinical gold standard from which causal links between SM performance and musculoskeletal injury has been established, future research might usefully encompass an array of dynamic and more ecologically-valid SM assessments. Such an approach would offer adjunct information about the extent to which complex oscillatory patterns of intra-microcycle training loads might elicit SM responses as they approach physiological limits. Finally, although the use of statistical constructs such as analysis of participants' intra-microcycle z-scores was used to mitigate the effects of inter-individual variation, tactical and positional demands inevitably lead to heterogeneity in locomotor outputs during training and match-play which may in turn, elicit varied impact on NM and SM performance among players.

Practical applications

The findings of this study highlight a preservation of intra-microcycle SM performance, but reduced capacity to initiate force rapidly via increased EMD latencies. The latter may negatively impact performance and prophylactic function in the latter stages of match-play and during periods of high fixture concentration. Performance support practitioners should target muscular activation proximal to full joint extension (c. 30° of flexion) and concomitant dynamic stiffness within conditioning strategies to mitigate the effects of in-game WL, while also encouraging favourable strength adaptations and recovery.

CONCLUSION

This study explored the extent and congruence amongst fluctuating patterns of intra-microcycle WL with NM and SM performance responses. Heterogeneous training and match-play WL appears to drive fluctuations in NM performance only partially, with EMD performance compromised most during match-play. A statistical and practically significant decrease in performance was observed for PF, but RFD and SM performance capabilities were unchanged during the microcycle.

AUTHOR CONTRIBUTIONS

Colin Clancy: Research design, data collection, lead author. Nigel Gleeson: Research design, data collection, project supervisor. Tom Mercer: Research design, project supervisor.

SUPPORTING AGENCIES

No funding agencies were reported by the authors.

DISCLOSURE STATEMENT

No potential conflict of interest was reported by the authors.

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