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SYSTEMATIC REVIEW

# Acute and Residual Soccer Match-Related Fatigue: A Systematic Review and Meta-analysis

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**Abstract**

*Background* Understanding soccer players' match-related fatigue and recovery profiles likely helps with developing conditioning programs that increase team performance and reduce injuries and illnesses. In order to improve match recovery (the return-to-play process and ergogenic interventions) it is also pivotal to determine if match simulation protocols and actual match-play lead to similar responses.

*Objectives* (1) To thoroughly describe the development of fatigue during actual soccer match play and its recovery time course in terms of physiological, neuromuscular, technical, biochemical and perceptual responses, and (2) to determine similarities of recovery responses between actual competition (11 vs. 11) and match simulations.

*Methods* A first screening phase consisted of a systematic search on PubMed (MEDLINE) and SportDiscus databases until March 2016. Inclusion criteria were: longitudinal study with soccer players; match or validated protocol; duration > 45 min; and published in English.

*Results* A total of 77 eligible studies ($n = 1105$) were used to compute 1196 effect sizes (ES). Half-time assessments revealed small to large alterations in immunological parameters (e.g. leukocytes, ES = 1.9), a moderate decrement in insulin concentration (ES = − 0.9) and a small to moderate impairment in lower-limb muscle function (ES = − 0.5 to − 0.7) and physical performance measures (e.g. linear sprint, ES = − 0.3 to − 1.0). All the systematically analyzed fatigue-related markers were substantially altered at post-match. Hamstrings force production capacity (ES = − 0.7), physical performance (2–4%, ES = 0.3−0.5), creatine kinase (CK, ES = 0.4), well-being (ES = 0.2−0.4) and delayed onset muscle soreness (DOMS, ES = 0.6–1.3) remained substantially impaired at G + 72 h. Compared to simulation protocols, 11 vs. 11 match format (CK, ES = 1.8) induced a greater magnitude of change in muscle damage (i.e. CK, ES = 1.8 vs. 0.7), inflammatory (IL-6, ES = 2.6 vs. 1.1) and immunological markers and DOMS (ES = 1.5 vs. 0.7) than simulation protocols at post-assessments. Neuromuscular performances at post-match did not differ between protocols.

*Conclusion* While some parameters are fully recovered (e.g. hormonal and technical), our systematic review shows that a period of 72 h post-match play is not long enough to completely restore homeostatic balance (e.g. muscle damage, physical and well-being status). The extent of the recovery period post-soccer game cannot consist of a 'one size fits all approach'. Additionally, the 'real match' (11 vs.

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11 format) likely induces greater magnitudes of perceptual (DOMS) and biochemical alterations (e.g. muscle damage), while neuromuscular alterations were essentially similar. Overall, coaches must adjust the structure and content of the training sessions during the 72-h post-match intervention to effectively manage the training load within this time-frame.

**Key Points**

Specific physical performance capabilities (e.g. sprint recovered at G + 72 h vs. jumping abilities still impaired at G + 72 h) likely present distinct recovery profiles, resulting in player physical performance impairments at 72 h post-match.

Post-match recovery monitoring of hamstring muscle function (eccentric and/or isometric muscle action), countermovement jump performance, DOMS and CK is of primary importance due to more profound changes (larger magnitude and extended time-course).

Medical staff and researchers should use biochemical (e.g. CK) and perceptual (DOMS) indices separately, for instance, when evaluating players readiness to return to 'real competition' or assessing the effectiveness of specific interventions (e.g. eccentric training).

## 1 Introduction

Over the last decade many excellent reviews have focused on soccer and its particular issues, including players' physiological characteristics [1–4] and performance determinants [5, 6], soccer biomechanics [7, 8] and specific training-induced effects [9–13] or periodization strategies [14]. Other reviews have also collected applicable findings in the topic of fatigue and recovery in soccer [6, 15–19]. Nevertheless, the large majority of these studies have not systematically analysed available literature, which may lead to selective reports, so that the complete picture cannot really be understood [20].

Modern players are experiencing an increase in match-play physical demands in part due to short between-match recovery periods and high neuromuscular demands (e.g. greater number of high-intensity running actions and acceleration requirements) [16, 21, 22]. This high demand may prompt transient fatigue during match-play (e.g. intense periods of the game or towards match end) and exacerbate post-match residual fatigue, implying that longer periods are needed to fully recover (e.g. several days) [15, 23]. Neuro-mechanical alterations (e.g. decrease in force production and power), physical performance impairments (e.g. sprint ability), perturbations in the biochemical milieu (e.g., creatine kinase) and worsened psychometric state are often reported acutely and in the days post-match [24–28]. Nevertheless, there are some conflicting reports regarding the time course of recovery of muscle function and selected performance-related components (e.g. jump vs. sprint ability) [18, 25, 29, 30]. In fact, several intrinsic (e.g. age, training history, playing position) and extrinsic factors (e.g. competition level, opposition standard, match importance, number of recovery days from previous match) likely influence the external and internal load experienced by each individual player with a consequent impact in the recovery time-course [31].

The habitual activity of soccer players during the competitive season consists of cycles of training, taper, competition and recovery over a weekly period [16] that may occur repetitively throughout 38–40 successive cycles. Understanding the players' recovery process therefore is critical and represents one complex issue for coaches and their support staff [18, 32]. An excessive training load prescription, while players are still 'recovering and regenerating', can result in increased injuries, reduced fitness and poor team performance [33–35]. This may explain why injuries rates are typically higher during congested competitive periods [36–39] without evidence of impairments in locomotor activities during match-play being noted [36, 38, 40]. Consequently, a clarification of the extent and time-course of solicitation of different physiological systems triggered by football match-play is warranted to prevent injuries, illness and non-functional overreaching states.

Remarkably, a considerable amount of studies used match-simulation protocols performed under laboratory conditions with the intention to replicate the overall game physical demands and gain knowledge about the magnitude of match-induced fatigue and time-course of recovery [41–49]. Soccer-match simulation protocols have also been performed on the field and in general have been validated to replicate internal (e.g. heart rate and rating of perceived exertion) and external match load metrics (e.g. running distance at different speed zones and velocity profile) associated with match play [41, 43–51]. These procedures present the advantages of a consistent longitudinal standardization of evaluation conditions (e.g. external load, exclude the unpredictable nature of the game) allowing a carefully controlled assessment of the effectiveness of any training or nutritional interventions [28, 41]. Furthermore, simulation protocols represent a valuable 'exercise

strategy' for training optimization since both players and coaches receive an objective feedback on the individual performance capability [52, 53]. Another advantage is to assist in the return-to-play process with players experiencing progressive 'real' match physiological strain [52, 53]. Nevertheless, several researchers have argued that such protocols may not accurately replicate the neuromechanical load associated with match play [25, 53, 54]. These critics were based on the following observations: (1) the uni-directional nature of the treadmill-based protocols, (2) the unpredictable nature of acceleration/deceleration and impacts occurrence, (3) reduced number of soccer-related tasks (e.g. kicking and jumping) and absence of directional change movements [25, 28, 53–55].

Consequently, there is a need for research to thoroughly quantify the importance of fatigue-causing mechanisms and identify the main factors influencing post-match recovery in soccer [18]. Since the last review in this area was published [18], there have been a large number of studies investigating one of the above mentioned specific areas. Moreover, this review and previous works are 'narrative' in nature, and so have not systematically reviewed the available evidence. To date, there has been no published systematic analysis that determines the time-course of post-match responses (e.g. variation and effect size of the different outcomes) and the most influencing factors. Therefore, our first intention was to systematically review match-related fatigue development (muscular function, physiological, technical, biochemical and perceptual responses) during actual play and post-match recovery profile in soccer. Another aim was to determine if these responses differ between actual competition and simulation protocols.

## 2 Methods

### 2.1 Research Question

The research questions were defined by the PICOS-model in accordance with the Preferred Reporting Item for systematic reviews and meta-analysis (PRISMA) statement [20, 56]: *Population* Male and female soccer players. *Intervention* Soccer-match and/or validated soccer-specific protocol performed on the field or in a laboratory environment. *Comparators* Changes between pre- (baseline), mid- (half-time) and post-match [match-end (Post) and 24-h intervals including 24-h (G + 24 h), 48-h (G + 48 h) and 72-h (G + 72 h)] time points. Similarities between soccer-match protocol formats (11 vs. 11, on-field and laboratory treadmill simulations). *Outcomes* Metabolic, physical and technical parameters and biochemical and perceptual responses. The different post-match measurements were adjusted to a 24-h period; for instance, G + 18 h measurement was included as a G + 24-h time-point. These aforementioned fatigue and recovery-related markers have been extensively used by numerous researches to understand the match-related impact and post-match recovery profile [5, 16, 18, 57, 58]. *Study Design* Randomized controlled designs, cohort and case studies investigating the acute and residual fatigue to a soccer match or a validated simulation protocol, performed on-field or in the laboratory on a treadmill.

### 2.2 Literature Search Strategies: Databases and Inclusion Criteria

The selection of studies was performed in two consecutive screening phases. The first phase consisted of identifying articles through a systematic search of the US National Library of Medicine (MEDLINE) through PubMed and the SportDiscus databases multiple times between June 2015 and March 2016. The following keyword 'soccer match' were used in combination with 'technical', 'neuromuscular', 'muscular power', 'jump', 'sprint', 'agility', 'change of direction', 'repeated sprint', 'intermittent', 'hormones', 'muscle damage', 'oxidative stress', 'inflammation', 'immunology', 'fatigue' and 'recovery'. Further search of the relevant literature was performed by using the 'related citations' function of PubMed and by scanning the reference lists. The second phase involved applying the selection criteria to the articles. Studies were chosen if they fulfilled the following six selection criteria:

1. The intervention was a soccer game or validated simulation protocols, performed on-field or in the laboratory on a treadmill. Only running-based protocols were selected for analysis.
2. The intervention had a duration of $\sim$ 45 min (i.e. half soccer match) or $\sim$ 90 min (i.e. total match).
3. The participants were soccer players ($\geq$ 18 years of age).
4. The study was published in English.
5. The study was published in a peer-reviewed journal.
6. The study reported effect sizes (ES), information needed to compute the ES or when ES was obtained from the author(s) of the study.

### 2.3 Independent Variables

Each study was read and coded by two independent investigators. An excel spreadsheet was utilized to extract all relevant information from the different studies.

1. *Players characteristics* Gender and players' training status. Three distinct levels of training status were

considered with number of training hours/sessions per week as units. Player training status was classified as 'Low' (< 4 h/two training sessions per week and one competitive game), 'Moderate' (between 5 and 7 h/three to four training sessions per week and one competitive game) and 'High' (more than 8 h/five training sessions per week and one competitive game).

2. *Methodological elements* Playing surface (artificial turf vs. natural grass), environmental temperature and type of protocol. Environmental temperatures were divided in three categories: cold (≤ 15 °C), temperate (17–25 °C) and hot (≥ 27 °C) conditions. The data from the match/simulation protocols that were included in temperate conditions reported relative humidity values below 60% [59, 60]. The type of protocol was categorized in three distinct levels: (1) 11 vs. 11 (official soccer match and friendly soccer match); (2) on-field simulation protocols [(protocols perform in a naturally-occurring environment involving different locomotor activities and/or unorthodox running patterns (e.g. sideways and backwards running) and/or change of directions)]; (3) laboratory treadmill protocols (protocols performed in a laboratory setting involving straight line running on a treadmill). In this review, the use of the word match-play/game refers to real (11 vs. 11) or simulated conditions (on-field and/or laboratory treadmill protocols).

## 2.4 Dependent Variables

The dependent variables extracted from the selected studies were grouped in *Objective* (metabolic, biochemical, physical and technical parameters) and *Subjective* (perceptual) responses.

### 2.4.1 Metabolic Alterations

Metabolic responses to match play were analysed by records of alteration in blood and muscle substract (glycogen, glucose, triglycerides, free-fatty acids, HDL, LDL), metabolites (lactate, urea, creatinine, uric acid, glycerol, bicarbonate, base excess) and pH at half-time and post-match time-points.

### 2.4.2 Biochemical Parameters

They were divided into five categories.

(a) Redox State: pro-oxidant and antioxidant status. Alterations in oxidant biomarkers [(markers of damage to lipids (malondialdehyde, *8-iso*-Prostaglandin F2α, and reactive oxygen metabolites test) and proteins (sulfhydryl groups) were recorded. Additionally, total antioxidant status evaluated by different assay techniques and specific measures of the enzymatic (e.g. glutathione peroxidase, glutathione reductase and superoxide dismutase) and non-enzymatic antioxidant component (reduced and total glutathione and uric acid) were recorded. We also considered other relevant markers of oxidative stress (homocysteine) and redox state (oxidized glutathione and reduced to oxidized glutathione ratio).

(b) Endocrinal responses: testosterone, cortisol and insulin hormones.

(c) Muscle damage: activity of intracellular enzymes (creatine kinase (CK) and lactate dehydrogenase (LDH)) and circulating concentrations of myoglobin, aspartate aminotransferase and alanine aminotransferase.

(d) Immunological state: white blood cell counts (leukocytes, lymphocytes, neutrophils and monocyte counts) and immunoglobins concentrations (IgA, IgM and IgG).

(e) Inflammatory markers: acute-phase proteins (C-reactive protein (CRP)) and anti- (interleukin 6 (IL-6)) and pro-inflammatory cytokines responses (tumour necrosis factor (TNF-α)).

### 2.4.3 Physical Performance Markers

These were divided into six categories:

(a) Neuromuscular performance measures. Lower-limb muscle function was systematically reviewed based on maximal forces/torques values measured by dynamometers, rate of force development, functional (eccentric hamstrings:concentric quadriceps ratio) and traditional muscle force ratios (concentric hamstrings:concentric quadriceps ratios) and peak torque angles of knee flexors, knee extensors and plantar flexors under concentric, eccentric and isometric muscle actions. Furthermore, data were categorized into three muscle contraction velocities ('low' ≤ 60° $s^{-1}$, 'moderate' between 60° $s^{-1}$ and 150° $s^{-1}$ and 'high' ≥ 150° $s^{-1}$) [61]. Neuro-mechanical measures were systematically review by elucidating alteration in motor output during isometric muscle contractions [(electromyographic activity (EMG) of the aforementioned muscle groups and voluntary activation level)] and intrinsic muscle properties (e.g. nerve stimulation techniques).

(b) Vertical jump ability. Vertical jump ability included the countermovement jump (CMJ) and the squat jump (SJ). Kinematic (centre of mass displacement) and

kinetic (peak and mean eccentric and concentric forces and peak power output) variables recorded during jumping tasks were also analysed.

(c) Straight-line sprint measures (SL, time) for a given distance. Best single sprint performance obtained during repeated-sprint ability test were also included in this category (e.g. fastest time or peak velocity). Kinematic (mean and peak speed, mean power output and hip flexion and extension angles) and kinetic (horizontal power and force production) variables were recorded.

(d) Sprint time during runs with change of direction (COD) was recorded (e.g. *t*-tests). In this category, we included all tests that are used to measure COD and shuttle sprint ability. Kinematic (range of motion during knee flexion variables recorded during COD tasks) were also extracted.

(e) Repeated sprint ability (RSA). Parameters such as the mean and cumulative/total sprint times and fatigue indexes (e.g. mean sprint times during 5 × 30 m sprints with 25 s of recovery) were extracted from RSA protocols. Kinematic (mean speed) and kinetic (mean power output) variables recorded during RSA were also documented.

(f) Intermittent-running endurance capacity. The capacity to perform high intensity intermittent endurance exercise (total distance covered) was recorded during the Yo–Yo intermittent endurance (YYIE2) and intermittent recovery level two tests (YYIR2).

#### *2.4.4 Technical Performance*

It was assessed by extracting records of performance (penalty shoots, time to complete a technical performance-based test, total performance, passing and shooting precision) in different technical skills tests (Loughborough soccer passing test and Loughborough soccer shooting test). Kinematic (ankle, knee and hip joint angular position and velocities) and kinetic (ground reaction forces) variables derived from technical skills (e.g. kick) were also extracted.

#### *2.4.5 Perceptual Responses*

Perceptual responses were systematically reviewed by extracting all relevant data collected by various questionnaires. Perceptual responses consisted mainly of general Delayed-Onset Muscle Soreness ($DOMS_{General}$) assessments (body regions not specified in the studies) or from specific body regions ($DOMS_{lower\ limbs}$) and quadriceps muscle groups ($DOMS_{QUADS}$). DOMS was recorded independently of the scale (e.g. 0–7, 0–10 or 0–100 visual analogue scales) and technique applied (e.g. response to squatting, muscle palpation or simply self-reporting). Recovery (Total quality recovery scale) and well-being sub-categories (Sleep, stress and fatigue) were also systematically reviewed.

### 2.5 Missing Data

The corresponding authors of the selected articles were contacted (email, social medias) requesting missing information including: (i) mean and standard deviation values from the different outcomes; (ii) surface conditions; (iii) environmental conditions; (iv) training background; and (v) other important characteristics that allow to better describe the experimental conditions (i.e. gender, players training status, period of the soccer season).

### 2.6 Analysis and Interpretation of Results

To evaluate the magnitude of the effects, percent change was calculated for each dependent variable for each study using the following equation:

$$\left[M_{post} - M_{pre}\right]/M_{pre} \times 100, \quad (1)$$

where $M_{post}$ was the post-match mean (e.g. 24 h) and $M_{pre}$ the baseline mean. ES (effect size) were computed to present standardized match-related effect on the outcome variables [62]. The different ES within individual studies were calculated with Cohen's *d*, by dividing the raw ES (difference in means) by the pooled standard deviations, as proposed by Bornstein et al. [63] as follows (Eq. 1):

$$ES = g = \frac{(M_{post} - M_{pre})}{SD_{pooled}}, \quad (2)$$

$SD_{pooled}$ is the pooled SD of the measurements and was calculated as follows (Eq. 3):

$$SD_{pooled} = \sqrt{\frac{\left((n-1) \times SD_{Pre}^2 + (n-1) \times SD_{Post}^2\right)}{(2n-2)}}, \quad (3)$$

where $SD_{Pre}^2$ is the standard deviation of the performance test completed before the match and $SD_{Post}^2$ is the standard deviation of the performance test completed after the match. To account for possible overestimation of the true population ES were corrected accounting for the magnitude of the sample size of each study [64]. Therefore, a correction factor (CF) was calculated as proposed by Hedges and Olkin [64]:

$$CF = 1 - \frac{3}{4df - 1}, \quad (4)$$

where $df = n - 1$. The corrected ES was calculated as follows:

$$\text{Corrected } ES_c = g \times CF. \quad (5)$$

Threshold values for $ES_c$ were defined as trivial (<0.2), small (0.2–0.6), moderate (0.6–1.2), large (1.2–2.0) and very large (>2.0) [65]. Results for each outcome variable are presented with number of observations (*N*) and number of $ES_c$ (Tables 2, 3, 4, 5 and 6).

All data analyses were conducted in Statistical Package for the Social Sciences version 18.0 (SPSS Science, Chicago) software and StatsDirect 3.0.152 (Altrincham, UK) was used for the meta-analysis. Mean percentages of change at different time points extracted from all studies were presented as mean (Range: minimum and maximum) for all parameters (Tables 2, 3, 4, 5 and 6). In order to investigate differences across protocols we performed meta-analysis for the reported effect sizes at match-end only where sufficient data was available. Pooled data on outcomes were analysed using random-effects meta-analyses as we assumed heterogeneity in the selected protocols and conditions. A significant difference was indicated when the 95% confidence interval (CI) of the ES did not overlap with zero.

### 2.7 Study Qualitative Assessment

We determined 12 criteria using the National Heart Blood Institute (NIH) guidelines for qualitative evaluation of observational cohort and cross-sectional studies and before-after (pre-post) studies with no control group. In addition, other versions of currently established scales used in Sports Sciences (e.g. Delphi and PEDRO Scale, Newcastle–Ottawa quality assessment scale, Downs and Black) were considered. Moreover, other relevant methodological issues for bias in the interpretation of the results (i.e. supplementary file) were considered for the creation of the 12 questions that constitute the Qualitative assessment tool. A table detailing the quality assessment criteria is included as a Supplementary file. The quality assessment was based on the reporting of study methods and results with answer categories of 'yes', 'partial' and 'no'. The quality assessment was applied to selected studies based on published information and after receiving the information from the authors (maximal score = 1).

## 3 Results

### 3.1 Selected Studies and Characteristics

The flow chart of the search and selection process is presented in Fig. 1. In summary, the searches identified 413 relevant articles. A further 266 articles were excluded after screening titles and abstracts. Consequently 147 full text articles were assessed for eligibility. Once applying the selection criteria, 77 studies were finally selected (Fig. 1), comprising a sample size of 1105 players, allowing the computation of 1196 ES (Tables 2, 3, 4, 5 and 6). Most excluded studies resulted from criteria six (no mean and SD data available to compute ES, 38.5%) and no outcomes of interest being reported (20%).

The average methodological quality of the included articles before and after receiving the information from the authors was $0.63 \pm 0.12$ and $0.71 \pm 0.13$, respectively.

Selected studies (Table 1) consisted of 11 vs. 11 protocols (40 studies and 635 players), on-field (27 studies and 346 players) and laboratory simulations protocols (12 studies and 147 players). On-field simulations consisted of studying performance of the Loughborough intermittent shuttle test (LIST, ten studies, 139 players), soccer-specific aerobic field test (SAFT90, six studies, 76 players), a soccer game modeling protocol (one study, eight players), a soccer match simulation (SMS, one study, 16 players) and the Copenhagen soccer test (CST, three studies, 39 players). Laboratory simulations consisted of protocols performed on motorized (11 studies, 139 players) and non-motorized treadmills (one study, eight players).

Physical performance-related measures were the most assessed variables (39 studies, 514 players, 448 ES), followed by biochemical markers (32 studies, 463 players, 396 ES), metabolic alterations (26 studies, 404 players, 226 ES), perceptual responses (ten studies, 152 players, 77 ES) and technical-related performance parameters (six studies, 92 players, 49 ES). Distribution of studies across time-points was as follows: half-time (22 studies, 476 players, 123 ES), post-match (56 studies, 976 players, 507 ES), G + 24 h (17 studies, 234 players, 191 ES), G + 48 h (12 studies, 152 players, 154 ES) and G + 72 h (nine studies, 102 players, 103 ES).

### 3.2 Metabolic Alterations

The most frequently examined metabolic markers (Table 2) were carbohydrate (23 studies, 343 players, 169 ES) [29, 43, 47, 53, 66–84] followed by lipids (six studies, 96 players, 24 ES) [69, 73–75, 81, 85], protein (five studies, 102 players, 23 ES) [30, 73, 75, 78, 86] and acid–base balance (two studies, 30 players, 10 ES) [67, 73, 75] related markers. Overall, small to very large (half-time) and large to extremely large (Post) changes in the abovementioned metabolic markers were observed.

### 3.3 Biochemical Markers

As depicted in Table 3, the most common biochemical markers measured in the selected studies were muscle damage (21 studies, 309 players, 103 ES) [25, 30, 66, 71, 75, 78, 87–101] followed by redox state (13 studies, 178 players, 87 ES)

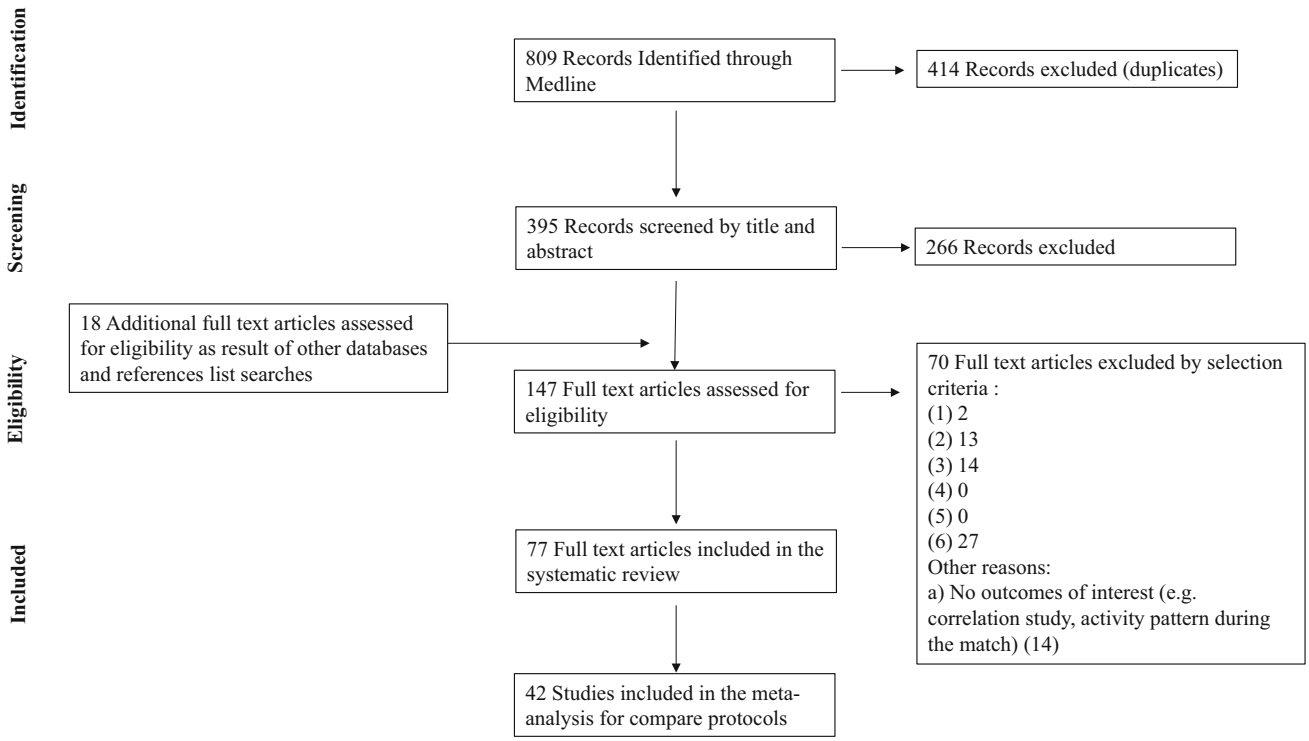


**Fig. 1** Flow chart showing the study selection process

[25, 30, 75, 78, 83, 86, 87, 94, 100, 102–105], endocrine (ten studies, 128 players, 83 ES) [25, 74, 75, 79–81, 83, 88, 90, 92], immunology (six studies, 71 players, 67 ES) [79, 87, 88, 92, 96, 106] and inflammatory biomarkers (seven studies, 130 players, 53 ES) [25, 75, 89, 90, 92, 96, 106].

### *3.3.1 Muscle Damage*

The most monitored serum markers investigating match-induced muscle injury were CK (21 studies, 284 players, 61 ES) [25, 30, 66, 71, 75, 78, 87–101], followed by myoglobin (six studies, 72 players, 24 ES) [25, 88, 96, 97, 99, 101], LDH (four studies, 66 players, 8 ES) [75, 87, 90, 101], AST (three studies, 48 players, 7 ES) [75, 87, 101] and ALT (two studies, 32 players, 3 ES) [75, 87]. These assessments occurred with greater incidence at match-end (43 ES) and G + 24 h (28 ES). Overall, throughout the recovery period and until G + 72 h, there were substantial elevations of muscle damage markers (Fig. 2).

### *3.3.2 Redox State*

Match-induced redox homeostasis alterations were examined in plasma/serum biological fluids. Investigations measured plasma total antioxidant status (five studies, 67 players, 15 ES) using distinct the assays total antioxidant status (TAS), oxygen radical absorbance capacity ($ORAC_{total}$) and ferric reduction antioxidant power (FRAP) [25, 75, 78, 83, 105]. Moreover, plasma concentration/activity of specific endogenous antioxidants molecules of non- (UA, GSH and TGSH; nine, two and one studies; 119, 32 and 16 players; 21, six and 2 ES, respectively) [25, 30, 75, 86, 94, 100, 102, 104, 105] and enzymatic nature (SOD, GPX, GR; three, two and two studies; 39, 21 and 21 players; five, four and 4 ES, respectively) [25, 75, 87] have been also investigated. Furthermore, examination of the concentration of specific oxidative stress-related markers such as GSSH (one study, 16 players, 2 ES) [104], GSH:GSSSH (two studies, 32 players, 6 ES) [104, 105], lipid peroxidation (MDA, 8-iso-PGF2a, D-roms, four studies, 54 players, 7 ES) [25, 78, 103, 104] protein oxidation products (-SH, one study, seven players, 3 ES) [25] and homocysteine levels (two studies, 37 players, 5 ES) [103, 105] were conducted. Generally, redox-related markers returned to near baseline at G + 72 h.

### *3.3.3 Inflammatory Markers*

Inflammatory responses to soccer-specific activity (Table 3) have been extensively investigated by acute phase protein CRP (five studies, 83 players, 14 ES) [25, 75, 89, 90, 92], anti-inflammatory (IL-6, five studies, 88 players, 24 ES) [89, 90, 92, 96, 106] and pro-inflammatory cytokines (TNF-α, three studies, 50 players, 15 ES) monitoring [89, 90, 105]. These assessments occurred more

**Table 1** Description of the studies included in the systematic review

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Abbey et al. [83] | 0.8 | 10 | 23 (3) | 177 (5) | 74 (6) | Randomized crossover design, ergogenic intervention (honey plus 6% carbohydrate enriched drink vs. sport drink vs. Placebo) | NCAA | PS | On-field (LIST) | M | NS | AT | NS |
| Alghannam [82] | 0.7 | 6 | 26 (2) | 180 (7) | 71 (5) | Randomized, blinded and controlled cross—over design. Ergogenic intervention. Players take part in 3 experiments (placebo (PLC), CHO-P, or isocaloric CHO beverages ingested 15 min prior to the exercise protocol and durin g half-time interval) | A | NS | Lab. T. | M | Low | T | Temp |
| Akkurt et al., [101] | 0.8 | 16 | 22 | 176 | 74 | Cohort Study, same group of players perform a match under two Surface conditions (AT vs. G) | A | OS | Match | M | Mod | AT/ G | Temp |
| Ali et al. [80] | 0.8 | 16 | 21 (3) | 180 (7) | 75 (7) | Randomized crossover design, Ergogenic interventions (6.4% CHO sport drink vs. Placebo) | A | CS | On-field (LIST) | M | Low | S | Cold |
| Ali et al. [81] | 0.8 | 17 | 21 (3) | 174 (5) | 72 (5) | Randomized crossover design, ergogenic intervention (sport drink vs. placebo) | A | CP | On-field (LIST) | M | Low | S | Cold |
| Andersson et al. [30] | 0.8 | 17 | 23 (4) | 167 (5) | 65 (7) | Randomized cohort study, active recovery group ($n = 8$) vs. passive recovery group ($n = 9$) | E | MS break | Match | F | High | AT | Cold |
| Andersson et al. [104] | 0.7 | 16 | 23 (4) | 167 (6) | 65 (7) | | E | MS break | Match | F | High | AT | Cold |
| Andersson et al. [105] | 0.8 | 16 | 22 (3) | 167 (5) | 64 (2) | | SP | CP | Match | F | High | AT | Cold |
| Andersson et al. [106] | 0.7 | 10 | 23 (3) | 167 (5) | 64 (8) | Randomized cohort study, active recovery group ($n = 8$) vs. passive recovery group ($n = 9$) | E | MS break | Match | F | High | AT | Cold |
| Apostolidis et al. [85] | 0.6 | 21 | 26 (3) | 178 (5) | 76 (4) | | A | PS | Match | M | High | G | Temp |
| Bendiksen [84] | 0.5 | 11 | 21 (5) | 169 (6) | 59 (6) | | E | NS | On-field (CST) | F | NS | NS | NS |
| Bendiksen [47] | 0.6 | 12 | 24 (5) | 181 (7) | 80 (8) | | SP | NS | On-field (CST) | | NS | NS | NS |
| Bishop et al. [79] | 0.7 | 8 | 21 (3) | 183 (6) | 78 (7) | Randomized crossover design, ergogenic intervention (carbohydrate drink participants consumed 400 ml of a lemon-flavored glucose solution (6% w/v) 10 min before the start of each 45 min of exercise and at 5 min post-exercise; a further 150 ml of this solution was consumed 14 and 29.5 min into each period of exercise vs. placebo) | A | NS | On-field (SSEP) | M | NS | NS | Temp |

**Table 1** continued

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Brito et al. [129] | 0.8 | 11 | 18 (1) | 173 (4) | 68 (4) | | A | OS | Match | M | Mod | G | Temp |
| Camarda et al. [109] | 0.8 | 21 | 23 (4) | 179 (7) | 74 (8) | Randomized crossover design, the players were allocated into one of two groups in accordance to their conventional Hcon:Qcon (BG = Hcon:Qcon > 0.60; unbalanced group, UNBG = Hcon:Qcon < 0.60) | E | NS | Lab. T. | M | Mod | T | NS |
| Costa et al. [78] | 0.7 | 10 | 18 (1) | 176 (7) | 74 (7) | | E | CP | On-field (LIST) | M | High | G | Hot |
| Cohen et al. [108] | 0.6 | 9 | 25 (1) | 179 (3) | 77 (4) | | SP | | On-field (LIST) | M | Low | NS | NS |
| Colombini et al. [86] | 0.7 | 19 | 27 (4) | 178 (3) | 78 (9) | | E | PS | Match | M | High | G | Hot |
| Coratella et al. [116] | 0.8 | 22 | 20 (2) | 177 (9) | 78 (3) | | A | MS break | On-field (LIST) | M | Mod | S | NS |
| Delextrat et al. [110] | 0.9 | 14 | 26 (5) | 168 (12) | 63 (6) | | A | PS | On-field (LIST) | F | Low | S | Temp |
| Edholm et al. [124] | 0.7 | 17 | 25 | 182 | 78.6 | Cross-over design, ergogenic intervention (effects of a half-time re-warm up on performance and movement patterns in soccer match play) | P | PS | Match | M | High | AT | Cold |
| Edwards et al. [77] | 0.5 | 7 | 24 (3) | 179 (4) | 74 (4) | | A | NS | Match | M | NS | NS | Temp |
| Fatouros et al. [76] | 0.9 | 20 | 20 (1) | 177 (1) | 75 (3) | | SP | OS | Match | M | High | G | Temp |
| Gant et al. [132] | 0.6 | 15 | 21 (3) | | 73 (8) | Randomized double-blind crossover design, ergogenic intervention (carbohydrate electrolyte solution (CON) providin g a total of 1.8 g/k g body mass (BM) of carbohydrate or a similar solution with added caffeine (CAF; 3.7 mg/k g BM) | NS | | On-field (LIST) | M | NS | S | Temp |
| Gatterer et al. [100] | 0.7 | 10 | 27 (5) | 180 (7) | 77 (7) | Single-blinded placebo-controlled crossover, ergogenic intervention (post-match massage under hypoxia vs. normoxia). 15 h after each game players rested for 1 h (30 min passive exposure followed by 30 min of massage) either at a simulated altitude of 4000 m (FIO2 of 13.5%) or under placebo conditions in a normobaric hypoxic chamber | P | CP | Match | M | High | G | NS |
| Girard et al. [27] | 0.8 | 17 | 27 (1) | 184 (1) | 80 (2) | Cohort Study, environmental conditions (temperate vs. hot) 17 male players perform two 90-min football matches in temperate (~ 20 °C and 55% rH) and hot (~ 43 °C and 20% rH) environments | E | CP | Match | M | NS | AT | Temp vs. Hot |

**Table 1** continued

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Gleeson et al. [111] | 0.9 | 8 | 23 (4) | 176 (5) | 72 (11) | | SP | OS | On-field (PIHSR) | M | Mod | AT | Temp |
| Gravina et al. [75] | 0.4 | 14 | 25 (5) | | 62 (7) | | P + SP | CP | Match | F | NS | NS | NS |
| Greco et al. [112] | 0.8 | 22 | 23 (3) | 178 (8) | 73 (7) | | P | PS | Lab. T. | M | Mod | T | NS |
| Greig et al. [127] | 0.6 | 10 | 25 (4) | | 77 (6) | | P | NS | Lab. T. | M | NS | T | NS |
| Greig [118] | 0.6 | 10 | 25 (4) | | 77 (6) | | P | NS | Lab. T. | M | NS | T | NS |
| Greig et al. [120] | 0.6 | 10 | 25 (4) | | 77 (6) | | P | NS | Lab. T. | M | NS | T | NS |
| Greig et al. [128] | 0.6 | 10 | 25 (4) | | 77 (6) | | P | NS | Lab. T. | M | NS | T | NS |
| Greig [125] | 0.5 | 10 | 25 (4) | | 77 (6) | | P | NS | Lab. T. | M | NS | T | NS |
| Gunnarsson et al. [98] | 0.5 | 16 | 24 (4) | 182 (4) | 81 (7) | Ergogenic intervention. Players were randomly assigned to a group ingesting a diet rich in carbohydrates and whey protein [CHO, protein, and fat content was 71, 21, and 8E%, respectively; high content of carbohydrates and whey protein (HCP), $n = 9$] or a group ingesting a normal diet (55, 18, and 26E%; control [CON], $n = 7$) during a 48-h recovery period | P | NS | Match + On-field (CST) | M | NS | NS | NS |
| Hughes et al. [123] | 0.8 | 17 | 22 (2) | 179 (5) | 76 (6) | Randomized crossover design. Player performed a football match simulation on high-quality artificial and natural surfaces | SP | OS | On-field (SSP) | M | Mod | AT vs. G | NS |
| Hulton et al. [74] | 0.6 | 9 | 21 (3) | 180 (8) | 74 (4) | Counterbalanced randomized crossover design, ergogenic intervention (pre-match HGI meal group vs. pre-match LGI meal group) | A | CP | Lab. T. | M | NS | T | NS |
| Ispirlidis et al. [29] | 0.5 | 14 | 20 (1) | 178 (8) | 75 (7) | | E | NS | Match | M | NS | G | NS |
| Jamurtas et al. [98] | 1 | 10 | 23 (2) | 179 (4) | 76 (5) | | SP | OS | Match | M | High | G | Temp |
| Jones et al. [113] | 0.7 | 20 | 22 (2) | 172 (6) | 68 (9) | | A | | On-field (SAFT90) | M | Mod | S | NS |
| Kellis et al. [134] | 0.7 | 10 | 23 (2) | 178 (6) | 70 (8) | | A | NS | On-field (LIST) | M | NS | AT | NS |
| Krustrup et al. [73] | 0.5 | 31 | 28 (NS) | 179 (NS) | 76 (NS) | | A | NS | Match | M | NS | NS | NS |

**Table 1** continued

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Krustrup et al. [72] | 0.8 | 23 | 23 | 170 | 60 | | E | | Match | F | Mod | NS | NS |
| Krustrup et al. [97] | 0.7 | 7 | 27 (3) | 180 (8) | 80 (6) | | P | CP | Match | M | NS | NS | NS |
| Lovell et al. [114] | 0.9 | 10 | 21 (1) | 183 (9) | 80 (7) | Randomized trial, one group under three different ergogenic interventions, each of 5 min duration during half-time (control vs. whole body vibration vs. agility) | SP | CP | On-field (SAFT90) | M | Low | NS | NS |
| Marshall et al. [119] | 0.9 | 8 | 22 (5) | 18 (6) | 76 (10) | | A | OS | On-field (SAFT90) | M | Low | S | Temp |
| Mohr et al. [130] | 0.5 | 16 | 26 (4) | 183 (5) | 77 (9) | Randomized design, ergogenic intervention during half-time (one **warm-up group** performed running and other exercises at a moderate intensity (average heart rate 135 beats min 1 or 70% of the peak heart rate reached during the game vs. one group that stay passive during half-time) | A | NS | Match | M | NS | NS | NS |
| Mohr et al. [126] | 0.6 | 10 | 19 (1) | | | | P | NS | Match | F | NS | NS | Hot |
| Mohr et al. [70] | 0.7 | 17 | 27 (5) | 184 (4) | 80 (5) | The effect of match-play in temperate vs. hot conditions | E | NS | Match | M | NS | AT/G | Temp/Hot |
| Mohr et al. [71] | 0.8 | 19 | 26 (4) | | 80 (7) | | SP | NS | Match | M | High | G | NS |
| Naclerio et al. [96] | 1 | 16 | 24. (4) | 181 (1) | 78 (9) | Double-blind, randomized, counter-balance, crossover design. One group under three ergogenic interventions. Multi-ingredient (53 g carbohydrate, 14.5 g whey protein, 5 g glutamine, 1.5 g L-carnitine-L-tartrate) supplement, carbohydrate only (69.5 g), or placebo) | A | OS | On-field (LIST) | M | Low | G | Temp |
| Nagahara et al. [122] | 0.9 | 13 | 20 (1) | 173 (5) | 70 (5) | | A | PS | Match | M | Mod | AT | Temp |
| Nedelec et al. [94] | 0.6 | 8 | 18 (1) | 182 (5) | 78 (4) | | P | NS | Lab. T. | M | NS | T | Temp |
| Nedelec et al. [121] | 0.7 | 13 | 18 (1) | 180 (6) | 72 (7) | Randomized crossover design, players perform a match under two Surface conditions (artificial turf vs. grass) | P | NS | On-field (SAFT90) | M | High | AT vs. G | Cold |
| Nedelec et al. [95] | 0.7 | 10 | 22 (3) | 178 (5) | 77 (8) | | P | NS | Match | M | High | NS | NS |
| Ostojic et al. [69] | 0.9 | 22 | 24 (2) | 185 (7) | 78 (7) | Counterbalanced randomized design. Ergogenic intervention (carbohydrate-electrolyte (CHOE) drink (carbohydrates 7%, sodium 24 mmol $l^{-1}$, chloride 12 mmol $l^{-1}$ and potassium 3 mmol $l^{-1}$) vs. placebo (plain water) | P | PS | Match | F | High | G | Temp |

**Table 1** continued

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Page et al. [53] | 0.8 | 18 | 23 (4) | 177 (7) | 77 (7) | | SP | OS | Lab. T. | M | Low | T | Temp |
| Papapanagiotou et al. [103] | 0.4 | 21 | 22 (4) | 179 (4) | 75 (8) | | NS | NS | Match | M | Mod | NS | NS |
| Pettersen et al. [68] | 0.5 | 19 | 18 (1) | | 72 (7) | Randomized double bind cross -over design. Ergogenic intervention. Players ingested either a capsule of 6 m g $kg^{-1}$ b.w. caffeine or placebo (dextrose) 65 min prior to the matches | E | NS | Match | M | NS | NS | Cold |
| Rahnama et al. [115] | 0.6 | 13 | 23 (4) | 178 (5) | 75 (4) | | A | NS | Lab. T. | M | NS | T | NS |
| Rampinini et al. [133] | 0.8 | 15 | 18 (1) | 174 (7) | 67 (6) | | E | CP | Match | M | Mod | G | Cold |
| Rampinini et al. [93] | 0.8 | 19 | 19 (1) | 181 (5) | 73 (7) | | P | CP | Match | M | High | AT | Temp |
| Robineau et al. [49] | 0.5 | 8 | 20 (1) | 175 (5) | 70 (7) | | A | OS | On-field (SGM) | M | Mod | G | NS |
| Romagnoli et al. [92] | 0.8 | 22 | 19 (1) | 181 (5) | 73 (7) | | P | CP | Match | M | High | AT | Temp |
| Russel et al. [67] | 0.8 | 16 | 18 (1) | 177 (1) | 71 (2) | | E | CP | On-field (SMS) | M | High | S | Temp |
| Russel et al. [91] | 0.7 | 14 | 20 (1) | | | | P | CP | Match | M | High | G | NS |
| Sanchis-Gomar et al. [102] | 0.8 | 12 | 25 (2) | 180 (10) | 75 (8) | | E | CP | Match | M | High | G | NS |
| Silva et al. [25] | 0.8 | 7 | 30 (2) | 181 (9) | 81 (11) | | E | CP | Match | M | High | G | Temp |
| Small et al. [44] | 0.5 | 9 | 21 (3) | 185 (9) | 82 (6) | | SP | CP | On-field (SAFT90) | M | NS | NS | NS |
| Small et al. [54] | 0.6 | 16 | 21 (3) | 185 (9) | | | SP | CP | On-field (SAFT90) | M | Low | NS | NS |
| Souglis et al. [90] | 0.6 | 18 | 26 (3) | 181 (6) | 76 (6) | | P | CP | Match | M | High | G | Temp |
| Souglis et al. [89] | 0.6 | 43 | M 23 (3) F 23 (2) | M 181 (6) F 168 (3) | M 76 (6) F 61 (4) | Compares the inflammatory responses between male ($n = 22$) and female ($n = 21$) soccer players | E | CP | Match | M vs. F | High | G | Temp |
| Stone et al. [43] | 0.7 | 12 | 21 (2) | 179 (7) | 75 (7) | | SP | NS | On-field (SSP) | M | NS | AT | NS |

**Table 1** continued

| Study | QA | *N* | Anthropometric characteristics | | | Conditions | Description | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Age (years) | Height (cm) | Weight (kg) | | Level | Period | Protocol | Gender | TS | Surf. | EC |
| Stone et al. [66] | 0.5 | 8 | 20 (1) | 177 (8) | 73 (7) | Randomized crossover design, surface (artificial turf vs. grass) | SP | PS | On-field (SSP) | M | Low | AT vs. G | Temp |
| Thorlund et al. [117] | 0.5 | 9 | 18 (1) | 180 (3) | 73 (3) | | A | CP | Match | M | Mod | G | Cold |
| Thorpe et al. [88] | 0.9 | 7 | 25 (6) | 179 (6) | 75.3 (5) | | SP | CP | Match | M | Low | G | Cold |
| Tsubakihara et al. [87] | 0.5 | 18 | | 161 (4) | 56 (5) | | A | NS | Match | F | High | NS | NS |

| Study | QA | *N* | Outcomes | | | | | Time-points | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Met | NM | Bio | Tech | Perc | HT | Post | 24 h | 48 h | 72 h |
| Abbey et al. [83] | 0.8 | 10 | x | | x | | | | x | | | |
| Alghannam [82] | 0.7 | 6 | x | | | | | | | | | |
| Akkurt et al., [101] | 0.8 | 16 | | | x | | x | | x | | | x |
| Ali et al. [80] | 0.8 | 16 | x | | x | x | | | x | | | |
| Ali et al. [81] | 0.8 | 17 | x | | x | x | | x | x | | | |
| Andersson et al. [30] | 0.8 | 17 | x | x | x | | x | | x | | | x |
| Andersson et al. [104] | 0.7 | 16 | | | x | | | | x | | | x |
| Andersson et al. [105] | 0.8 | 16 | | | x | | | | x | x | | |
| Andersson et al. [106] | 0.7 | 10 | | | x | | | | x | x | x | x |
| Apostolidis et al. [85] | 0.6 | 21 | x | | | | | | | | | |
| Bendiksen [84] | 0.5 | 11 | x | | | | | | | | | |
| Bendiksen [47] | 0.6 | 12 | x | | | | | | | | | |
| Bishop et al. [79] | 0.7 | 8 | x | | x | | | x | x | | | |
| Brito et al. [129] | 0.8 | 11 | | x | | | | | x | | | |
| Camarda et al. [109] | 0.8 | 21 | | x | | | | | x | | | |
| Costa et al. [78] | 0.7 | 10 | | | x | | | | x | | | |
| Cohen et al. [108] | 0.6 | 9 | | x | | | | | x | | | |
| Colombini et al. [86] | 0.7 | 19 | x | | x | | | | x | | | |
| Coratella et al. [116] | 0.8 | 22 | | x | | | | | x | | | |
| Delextrat et al. [110] | 0.9 | 14 | | x | | | | | x | | | |
| Edholm et al. [124] | 0.7 | 17 | | x | | | | x | | | | |
| Edwards et al. [77] | 0.5 | 7 | x | | | | | | x | | | |

**Table 1** continued

| Study | QA | *N* | Outcomes | | | | | Time-points | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Met | NM | Bio | Tech | Perc | HT | Post | 24 h | 48 h | 72 h |
| Fatouros et al. [76] | 0.9 | 20 | x | | | | | | | | | |
| Gant et al. [132] | 0.6 | 15 | | | | x | | x | x | | | |
| Gatterer et al. [100] | 0.7 | 10 | | | x | | | | | | | |
| Girard et al. [27] | 0.8 | 17 | | x | | | | | x | x | | |
| Gleeson et al. [111] | 0.9 | 8 | | x | | | | | x | | | |
| Gravina et al. [75] | 0.4 | 14 | x | | x | | | | x | x | | |
| Greco et al. [112] | 0.8 | 22 | | x | | | | | x | | | |
| Greig et al. [127] | 0.6 | 10 | | x | | | | x | x | | | |
| Greig [118] | 0.6 | 10 | | x | | | | x | x | | | |
| Greig et al. [120] | 0.6 | 10 | | x | | | | x | x | | | |
| Greig et al. [128] | 0.6 | 10 | | x | | | | x | | | | |
| Greig [125] | 0.5 | 10 | | x | | | | x | x | | | |
| Gunnarsson et al. [98] | 0.5 | 16 | | | x | | | | x | x | x | |
| Hughes et al. [123] | 0.8 | 17 | | x | | | | | x | | | |
| Hulton et al. [74] | 0.6 | 9 | x | | x | | | x | x | | | |
| Ispirlidis et al. [29] | 0.5 | 14 | x | | | | | | | | | |
| Jamurtas et al. [98] | 1 | 10 | | x | x | | x | | x | | | x |
| Jones et al. [113] | 0.7 | 20 | | x | | | | x | x | | | |
| Kellis et al. [134] | 0.7 | 10 | | | | x | | | | | | |
| Krustrup et al. [73] | 0.5 | 31 | x | | | | | | | | | |
| Krustrup et al. [72] | 0.8 | 23 | x | x | | | | | x | | | |
| Krustrup et al. [97] | 0.7 | 7 | | x | x | | x | | x | x | x | x |
| Lovell et al. [114] | 0.9 | 10 | | x | | | | x | x | | | |
| Marshall et al. [119] | 0.9 | 8 | | x | | | | x | x | | | |
| Mohr et al. [130] | 0.5 | 16 | | x | | | | x | x | | | |
| Mohr et al. [126] | 0.6 | 10 | | x | | | | | x | | | |
| Mohr et al. [70] | 0.7 | 17 | x | | | | | | | | | |
| Mohr et al. [71] | 0.8 | 19 | x | | x | | | | | x | | |
| Naclerio et al. [96] | 1 | 16 | | x | x | | | | x | x | | |
| Nagahara et al. [122] | 0.9 | 13 | | x | | | | | x | | | |
| Nedelec et al. [94] | 0.6 | 8 | | x | x | | x | x | x | x | x | x |
| Nedelec et al. [121] | 0.7 | 13 | | x | | | x | x | x | x | x | |
| Nedelec et al. [95] | 0.7 | 10 | | x | x | | x | | | x | x | x |
| Ostojic et al. [69] | 0.9 | 22 | x | | | | x | x | x | | | |
| Page et al. [53] | 0.8 | 18 | x | | | | | | | | | |

**Table 1** continued

| Study | QA | *N* | Outcomes | | | | | Time-points | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Met | NM | Bio | Tech | Perc | HT | Post | 24 h | 48 h | 72 h |
| Papapanagiotou et al. [103] | 0.4 | 21 | | | x | | | | x | | | |
| Pettersen et al. [68] | 0.5 | 19 | x | | | | | | | | | |
| Rahnama et al. [115] | 0.6 | 13 | | x | | | | x | x | | | |
| Rampinini et al. [133] | 0.8 | 15 | | | | x | | x | x | | | |
| Rampinini et al. [93] | 0.8 | 19 | | x | x | x | x | | x | x | x | |
| Robineau et al. [49] | 0.5 | 8 | | x | | | | x | x | | | |
| Romagnoli et al. [92] | 0.8 | 22 | | x | x | | x | | x | x | x | |
| Russel et al. [67] | 0.8 | 16 | x | | | | | | | | | |
| Russel et al. [91] | 0.7 | 14 | | x | x | | | | | x | x | |
| Sanchis-Gomar et al. [102] | 0.8 | 12 | | | x | | | | | | | |
| Silva et al. [25] | 0.8 | 7 | | x | x | | | | x | x | x | x |
| Small et al. [44] | 0.5 | 9 | | x | | | | x | x | | | |
| Small et al. [54] | 0.6 | 16 | | x | | | | x | x | | | |
| Souglis et al. [90] | 0.6 | 18 | x | | x | | | | x | x | x | |
| Souglis et al. [89] | 0.6 | 43 | | | x | | | | | | | |
| Stone et al. [43] | 0.7 | 12 | x | | | | | | | | | |
| Stone et al. [66] | 0.5 | 8 | x | x | x | | x | | x | x | x | |
| Thorlund et al. [117] | 0.5 | 9 | | x | | | | | x | | | |
| Thorpe et al. [88] | 0.9 | 7 | | | x | | | | x | | | |
| Tsubakihara et al. [87] | 0.5 | 18 | | | x | | | | x | | | |

Age, height and weight are given as mean (SD)

*QA* qualitative assessment after receiving information from authors, *N* sample size, *TS* training status, *Surf* surface, *EC* environmental conditions, *Met* metabolic, *NM* neuromuscular, *Bio* biochemical, *Tech* technical, *Perc* perceptual, *HT* half-time, *Post* post-match, *24 h* 24 h post-match, *48 h* 48 h post-match, *72 h* 72 h post-match, *A* amateurs, *E* elite, *SP* semi-professional, *P* professional, *NCAA* university players, *PS*-preseason, *OS* off-season, *CP* competitive period, *MS* mid-season, *Lab T* laboratory treadmill protocol, *M* male, *F* female, *Mod* moderate, *AT* artificial grass, *S* synthetic, *G* natural grass, *Temp* temperate conditions, *NS* not specified, *LIST* Loughborough intermittent shuttle test, *SAFT90* soccer-specific aerobic field test, *SGM* soccer game modeling protocol, *SMS* soccer match simulation, *CST* Copenhagen soccer test, *PIHSR* prolonged intermittent high intensity shuttle run protocol

**Table 2** Metabolic alterations during and at post-match for all the three types of protocols

| Marker | Baseline-half | | | | Baseline-post | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ%(range) | ES (range) |
| **Carbohydrate metabolism** | | | | | | | | |
| Lactate plasma | 207 | 17 | 174.6 (30.1; 467.9) | 2.7 (0.7; 7.6) | 366 | 26 | 246.2 (49.6; 554.7) | 4.2 (0.9; 32.2) |
| Lactate muscle | | | | | | 1 | 210 | 2.2 |
| Glycogen | | | | | 41 | 3 | − 45.0 (− 48.8; − 39.8) | − 4.7 (− 9.5; − 1.9) |
| Glucose | 106 | 8 | 9.1 (− 8.9; 19.5) | 0.8 (− 0.5; 1.8) | 283 | 18 | 12.3 (− 16.2; 36.0) | 1.2 (− 0.9; 9.7) |
| **Protein metabolism** | | | | | | | | |
| Creatinine | | | | | 43 | 3 | 29.0 (25.9; 31.0) | 1.7 (1.5; 2.1) |
| Urea | | | | | 78 | 6 | 15.6 (− 13.3; 69.2) | 2.4 (− 1.0; 13.2) |
| Urea/creatinine | | | | | 33 | 2 | − 17.3 (− 19.1; − 15.6) | − 0.9 (− 0.9; − 0.8) |
| Ammonia | 24 | 1 | 116.9 | 1.3 | 61 | 3 | 269.5 (59.2; 511.9) | 4.0 (1.4; 8.8) |
| UA | | | | | 106 | 8 | 16.0 (0.1; 49.4) | 0.6 (0.0; 1.7) |
| **Lipid metabolism** | | | | | | | | |
| Triglycerides | | | | | 35 | 2 | − 0.1 (− 9.7; 9.6) | − 0.4 (− 1.1; 0.3) |
| Glycerol | 22 | 2 | 342.0 (313.1; 370.8) | 12.5 (11.9; 13.1) | 22 | 2 | 544.0 (527.5; 560.5) | 12.9 (9.9; 16.0) |
| Plasma FFA | 62 | 5 | 118.4 (− 20.0; 400.0) | 0.5 (− 0.9; 1.9) | 74 | 5 | 530.1 (70.0; 1590.0) | 1.9 (1.0; 2.6) |
| HDL | | | | | 35 | 2 | 10.8 (9.1; 12.4) | 1.0 (0.6; 1.5) |
| LDL | | | | | 35 | 2 | − 4.8 (− 6.7; − 3.0) | − 0.5 (− 0.9; − 0.1) |
| **Acid base** | | | | | | | | |
| Bicarbonate | 32 | 2 | − 7.6 (− 10.0; − 5.2) | − 0.9 (− 0.9; − 0.9) | 46 | 3 | − 15.4 (− 23.2; − 11.0) | − 1.6 (− 2.0; − 1.0) |
| Base excess | 16 | 1 | − 71 | − 1 | 16 | 1 | − 165 | − 2 |
| Blood pH | 16 | 1 | − 0.4 | − 0.7 | 16 | 1 | − 0.7 | − 1.2 |
| Muscle pH | | | | | 5 | 1 | − 2.3 | − 3.4 |

*N* number of observations, *ES* corrected effect size, *UA* uric acid, *FFA* free fatty acids, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein

frequently at post-match (19 ES) and G + 24 h (16 ES). Overall, throughout the recovery period and until G + 72 h, there were substantial elevations of inflammatory markers.

### *3.3.4 Immunological Markers*

Research examined the number of circulating leukocytes (three studies, 36 players, 12 ES) [79, 87, 92, 106], neutrophils (five studies, 74 players, 18 ES) [79, 87, 92, 96, 106], monocytes (two studies, 32 players, nine ES) [92, 96] and lymphocytes (five studies, 74 players, 18 ES) [79, 87, 92, 96, 106]. Additionally, match-induced alterations in immune system were examined by assessment of specific antibodies concentration such as plasma (two studies, 25 players, 2 ES) [87, 88] and saliva (one study, eight players, 4 ES) [79] immunoglobulin A (IgA, 33 players, three studies, six ES) and plasma immunoglobin G (IgG, two studies, 25 players, 2 ES) [87, 88] and M (IgM, two studies, 25 players, 2 ES) [87, 88]. Although scarcely examined, there were also reports on specific proteins of the blood complement system (C3 and C4, one study, 18 players, 2 ES) [87].

Overall, throughout the recovery period and until G + 72 h, there were substantial elevations of immunological markers.

### *3.3.5 Endocrine Responses*

Endocrine responses (Table 3) were mostly examined utilizing plasma fluid. The investigated variables were the peptide insulin hormone (four studies, 61 players, 11 ES) [74, 80, 81, 83], steroid hormones cortisol ($C_{plasma}$, five studies, 72 players, 14 ES) [25, 79, 83, 90, 92] and testosterone ($T_{plasma,}$ four studies, 50 players, eight ES)

**Table 3** Alterations in biochemical makers during the match and throughout the 72-h recovery period for all the three types of protocols

| | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Hormonal** | | | | | | | | | | | | |
| **Plasma (P)** | | | | | | | | | | | | |
| Insulin | 18 | 2 | − 24.0 (− 28.4; − 19.6) | − 0.9 (− 1.3; − 0.4) | 114 | 9 | − 23.1 (− 65.6; 46.6) | − 1.0 (− 2.4; 0.6) | | | | |
| P-Cortisol | 16 | 2 | − 33.9 (− 34.8; − 33.0) | − 1.4 (− 1.5; − 1.3) | 86 | 7 | 10.9 (− 46.7; 105.0) | 1.1 (− 2.0; 9.8) | 29 | 2 | − 5.2 (− 36.5; 26.1) | 0.0 (− 1.6; 1.6) |
| P-Testosterone | | | | | 36 | 2 | 4.6 (− 25.9; 35.1) | 0.0 (− 0.8; 0.7) | 43 | 3 | − 7.7 (− 22.2; 1.0) | − 0.3 (− 0.8; 0.1) |
| P-Free T/C | | | | | 22 | 1 | − 2.6 | 0 | 22 | 1 | 24.7 | 0.6 |
| P-T/C | | | | | | | | | 7 | 1 | − 20 | − 1.2 |
| **Saliva (S)** | | | | | | | | | | | | |
| S-Cortisol | | | | | 7 | 1 | 47.5 | 0.8 | | | | |
| S-Testosterone | | | | | 7 | 1 | 42.3 | 1.1 | | | | |
| S-T/C | | | | | 7 | 1 | − 3.6 | − 0.1 | | | | |
| **Muscle damage** | | | | | | | | | | | | |
| CK | | | | | 308 | 23 | 95.3 (10.5; 201.7) | 1.8 (0.1; 7.7) | 246 | 18 | 183.8 (61.2; 415.2) | 1.6 (0.5; 4.6) |
| LDH | | | | | 82 | 5 | 27.4 (13.3; 49.8) | 1.9 (0.9; 4.7) | 14 | 1 | 7.9 | 0.6 |
| Myoglobin | | | | | 113 | 9 | 1526.8 (207.4; 6066.7) | 2.3 (0.6; 8.7) | 81 | 7 | 105.8 (− 37.3; 389.6) | 0.7 (− 0.3; 3.0) |
| ALT | | | | | 32 | 2 | 12.4 (11.0; 13.8) | 0.4 (0.3; 0.6) | 14 | 1 | 7.2 | 0.3 |
| AST | | | | | 64 | 4 | 15.5 (13.5; 18.9) | 0.7 (0.6; 0.9) | 14 | 1 | 15.7 | 0.5 |
| **Inflammatory** | | | | | | | | | | | | |
| CRP | | | | | 97 | 5 | 25.1 (− 15.2; 83.3) | 0.3 (− 0.1; 1.0) | 72 | 4 | 128.0 (121.2; 133.7) | 0.9 (0.5; 1.3) |
| IL-6 | | | | | 141 | 9 | 236.6 (81.5; 440.4) | 4.2 (0.9; 26.2) | 123 | 8 | 27.8 (− 10.0; 185.7) | 0.2 (− 0.1; 0.7) |
| TNF-α | | | | | 71 | 5 | 199.5 (137.6; 242.9) | 4.2 (0.9; 12.2) | 53 | 4 | 55.1 (6.8; 142.9) | 0.7 (0.2; 1.2) |
| **Immunological** | | | | | | | | | | | | |
| Leucocyte | 16 | 2 | 37.6 (33.3; 41.9) | 1.9 (1.5; 2.2) | 61 | 5 | 78.2 (47.1; 115) | 2.0 (1.6; 2.7) | 27 | 2 | 47.0 (11.0; 83.0) | 1.5 (0.3; 2.6) |
| Lymphocyte | 16 | 2 | 12.9 (5.9; 20.0) | 0.4 (0.2; 0.6) | 109 | 8 | 1.0 (− 18.8; 33.3) | − 0.1 (− 0.8; 1.1) | 75 | 5 | 8.2 (− 10.0; 47.4) | 0.4 (− 0.3; 2.0) |
| Monocytes | | | | | 70 | 4 | 48.9 (24.1; 96.2) | 1.1 (0.7; 1.7) | 70 | 4 | 6.4 (− 9.7; 13.8) | 0.2 (− 0.3; 0.5) |
| Neutrophil | 16 | 2 | 52.4 (48.3; 56.5) | 1.9 (1.2; 2.6) | 109 | 8 | 97.7 (8.1; 221.3) | 1.6 (0.2; 2.8) | 75 | 5 | 17.3 (− 21.6; 117.2) | 0.2 (− 0.8; 2.7) |
| S-IgA | 16 | 2 | − 23.9 (− 34.9; − 13.0) | − 0.4 (− 0.5; − 0.2) | 16 | 2 | 17.8 (− 14.4; 50.0) | 0.2 (− 0.2; 0.5) | | | | |
| P-IgA | | | | | 25 | 2 | − 1.9 (− 8.0; 4.2) | − 0.1 (− 0.3; 0.1) | | | | |
| P-IgG | | | | | 25 | 2 | 3.7 (2.4; 5.1) | 0.2 (0.1; 0.2) | | | | |
| P-IgM | | | | | 25 | 2 | 2.7 (0.0; 5.4) | 0.1 (0.0; 0.2) | | | | |

**Table 3** continued

| | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Redox status** | | | | | | | | | | | | |
| UA | | | | | 106 | 8 | 16.0 (0.1; 49.4) | 0.6 (0.0; 1.7) | 63 | 5 | 6.7 (1.2; 19.1) | 0.4 (0.1; 1.2) |
| GSSG | | | | | 16 | 1 | 19.2 | 0.6 | 16 | 1 | 8.5 | 0.3 |
| GSH/GSSG | | | | | 32 | 2 | − 14.9 (− 15.2; − 14.6) | − 0.5 (− 0.5; − 0.5) | 32 | 2 | − 10.2 (− 10.6; − 9.8) | − 0.3 (− 0.4; − 0.3) |
| **Antioxidant enzymes** | | | | | 60 | 4 | 2.0 (− 21.4; 16.7) | 0.1 (− 0.6; 0.5) | 21 | 3 | 3.8 (− 8.4; 10.0) | 1.2 (− 1.1; 3.1) |
| SOD | | | | | 32 | 2 | − 2.4 (− 21.4; 16.7) | − 0.1 (− 0.6; 0.4) | 7 | 1 | 9.8 | 3.1 |
| GPX | | | | | 14 | 1 | 4.3 | 0.2 | 7 | 1 | − 8.4 | − 1.1 |
| GR | | | | | 14 | 1 | 8.5 | 0.5 | 7 | 1 | 10 | 1.7 |
| **Endogenous antioxidants** | | | | | | | | | | | | |
| TGSH | | | | | 16 | 1 | 22.9 | 1.2 | 16 | 1 | 11.4 | 1 |
| GSH | | | | | 32 | 2 | 5.3 (5.3; 5.3) | 0.2 (0.2; 0.2) | 32 | 2 | 5.3 | 0.2 (0.2; 0.2) |
| **Antioxidant capacity** | | | | | 100 | 9 | 11.2 (− 13.9; 16.9) | 0.8 (− 1.0; 1.4) | 23 | 2 | 5.4 (− 2.3; 13.1) | 0.6 (− 0.2; 1.4) |
| FRAP | | | | | 26 | 2 | − 2.2 (− 13.9; 9.5) | 0.1 (− 1; 0.8) | 16 | 1 | − 2.3 | − 0.2 |
| $ORAC_{total}$ | | | | | 30 | 3 | 15.3 (13.3; 16.9) | 1.3 (1.2; 1.4) | | | | |
| $ORAC_{plasma}$ | | | | | 30 | 3 | 15.0 (12.6; 16.8) | 0.9 (0.6; 1.1) | | | | |
| TAS | | | | | 14 | 1 | 14.1 | 0.9 | 7 | 1 | 13.1 | 1.4 |
| **Oxidative stress** | | | | | | | | | | | | |
| **Lipid peroxidation** | | | | | 47 | 3 | 13.2 (1.6; 24.8) | 0.8 (0.0; 1.9) | 23 | 2 | 32.1 (− 0.4; 64.7) | 0.7 (0.0; 1.5) |
| 8-iso-PGF2α | | | | | 21 | 1 | 13.3) | 0.6 | | | | |
| D-ROMS | | | | | 16 | 1 | 1.6 | 0 | 16 | 1 | − 0.4 | 0 |
| MDA | | | | | 10 | 1 | 24.8 | 1.9 | 7 | 1 | 64.7 | 1.5 |
| **Protein oxidation** | | | | | | | | | | | | |
| –SH | | | | | | | | | 7 | 1 | 83.6 | 3.5 |
| Homocysteine | | | | | 37 | 2 | 9.5 (1.1; 18.0) | 0.6 (0.0; 1.2) | 16 | 1 | − 3.3 | − 0.1 |

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| Hormonal | | | | | | | | |
| **Plasma (P)** | | | | | | | | |
| Insulin | | | | | | | | |
| P-Cortisol | 29 | 2 | 13.0 (− 33.5; 59.5) | 0.1 (− 1.5; 1.6) | 7 | 1 | 3.6 | 0.2 |
| P-Testosterone | 29 | 2 | − 15.6 (− 31.0; − 0.2) | − 0.6 (− 1.2; 0.0) | 7 | 1 | 0 | 0 |
| PFree T/C | 22 | 1 | 11.7 | 0.3 | | | | |

**Table 3** continued

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| P-T/C | 7 | 1 | − 33.2 | − 1.3 | 7 | 1 | − 7.8 | − 0.5 |
| **Saliva (S)** | | | | | | | | |
| S-Cortisol | | | | | | | | |
| S-Testosterone | | | | | | | | |
| S-T/C | | | | | | | | |
| **Muscle damage** | | | | | | | | |
| CK | 157 | 12 | 82.9 (9.4; 140.5) | 1.0 (0.1; 1.9) | 81 | 8 | 29.1 (0.5; 89.5) | 0.4 (0.0; 1.3) |
| LDH | | | | | 32 | 2 | − 4.0 (− 8.1; 0.1) | − 0.2 (− 0.4; 0.0) |
| Myoglobin | 33 | 4 | 11.9 (− 25.0; 38.9) | 0.3 (− 0.3; 0.7) | 46 | 4 | 2.2 (0.0; 4.9) | 0.1 (0.0; 0.2) |
| ALT | | | | | | | | |
| AST | | | | | 32 | 2 | − 7.5 (− 14.7; − 0.3) | − 0.3 (− 0.6; 0.0) |
| **Inflammatory** | | | | | | | | |
| CRP | 72 | 4 | 30.7 (− 1.3; 57.2) | 0.3 (0.0; 0.6) | 7 | 1 | 2.6 | 0 |
| IL-6 | 75 | 5 | 8.5 (− 22.2; 66.7) | 0.0 (− 0.4; 0.6) | 10 | 2 | 32.7 (− 20.4; 85.7) | 0.1 (− 0.2; 0.4) |
| TNF-α | 53 | 4 | − 1.2 (− 11.1; 14.3) | − 0.1 (− 0.4; 0.4) | 10 | 2 | 54.8 (42.9; 66.7) | 0.6 (0.6; 0.7) |
| **Immunological** | | | | | | | | |
| Leucocyte | 27 | 2 | 8.8 (5.7; 12.0) | 0.3 (0.2; 0.4) | 5 | 1 | − 5.7 | − 0.2 |
| Lymphocyte | 27 | 2 | − 2.2 (− 4.4; 0.0) | − 0.1 (− 0.2; 0.0) | 5 | 1 | − 10.5 | − 0.5 |
| Monocytes | 22 | 1 | 17.3 | 0.4 | | | | |
| Neutrophil | 27 | 2 | 18.9 (10.3; 27.4) | 0.4 (0.3; 0.6) | 5 | 1 | 0 | 0 |
| S-IgA | | | | | | | | |
| P-IgA | | | | | | | | |
| P-IgG | | | | | | | | |
| P-IgM | | | | | | | | |
| **Redox status** | | | | | | | | |
| UA | 47 | 4 | 10.9 (4.5; 19.0) | 0.6 (0.2; 1.1) | 47 | 4 | 2.6 (− 0.2; 4.7) | 0.1 (0.0; 0.3) |
| GSSG | | | | | | | | |
| GSH/GSSG | 16 | 1 | − 26.8 | − 1 | 16 | 1 | − 17.1 | − 0.7 |
| **Antioxidant enzymes** | 21 | 3 | 1.5 (− 4.1; 4.8) | 0.6 (− 0.6; 1.9) | 21 | 3 | − 0.2 (− 0.8; 0.2) | − 0.1 (− 0.3; 0.1) |
| SOD | 7 | 1 | 4.8 | 1.9 | 7 | 1 | 0.2 | 0.1 |
| GPX | 7 | 1 | − 4.1 | − 0.6 | 7 | 1 | 0 | 0 |
| GR | 7 | 1 | 3.7 | 0.6 | 7 | 1 | − 0.8 | − 0.3 |
| TGSH | | | | | | | | |
| GSH | 16 | 1 | − 26.3 | − 1.2 | 16 | 1 | − 21.1 | − 1 |

**Table 3** continued

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Antioxidant capacity** | 23 | 2 | 1.4 (− 5.5; 8.4) | 0.4 (− 0.5; 1.3) | 23 | 2 | − 3.1 (− 6.2; 0.0) | − 0.3 (− 0.5; 0.0) |
| FRAP | 16 | 1 | − 5.5 | − 0.5 | 16 | 1 | − 6.2 | − 0.5 |
| $ORAC_{total}$ | | | | | | | | |
| $ORAC_{plasma}$ | | | | | | | | |
| TAS | 7 | 1 | 8.4 | 1.3 | 7 | 1 | 0 | 0 |
| **Oxidative stress** | | | | | | | | |
| **Lipid peroxidation** | 7 | 1 | 30.8 | 1.7 | 7 | 1 | 1.8 | 0.1 |
| 8-iso-PGF2α | | | | | | | | |
| D-ROMS | | | | | | | | |
| MDA | 7 | 1 | 30.8 | 1.7 | 7 | 1 | 1.8 | 0.1 |
| **Protein oxidation** | | | | | | | | |
| –SH | 7 | 1 | − 9.8 | − 1 | 7 | 1 | 0 | 0 |
| Homocysteine | 16 | 1 | − 1.1 | 0 | 16 | 1 | − 11 | − 0.2 |

*N* number of observations, *ES* corrected effect size, *P* plasma, *S* saliva, *T/C* testosterone cortisol ratio, *CK* creatine kinase, *LDH* lactate dehydrogenase, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *CRP* C-reactive protein, *IL6* interleukin 6, *TNFα* tumor necrosis factor, *Ig* immunoglobulin, *UA* uric acid, *GSH* reduced glutathione, *GSSG* oxidized glutathione, *GSH/GSSG* ration reduced oxidized glutathione, *SOD* superoxide dismutase, *GPX* glutathione peroxidase, *GR* glutathione reductase, *TGSH* total glutathione, *GSH* glutathione, *FRAC* ferric reducing/antioxidant power, *ORAC* oxygen radical absorbance capacity, *TAS* – total antioxidant status, *8isoPGF2α 8iso*-prostaglandin F2α, *DROMS* reactive oxygen metabolites test, *MDA* malondialdehyde, *SH* sulfhydryl groups

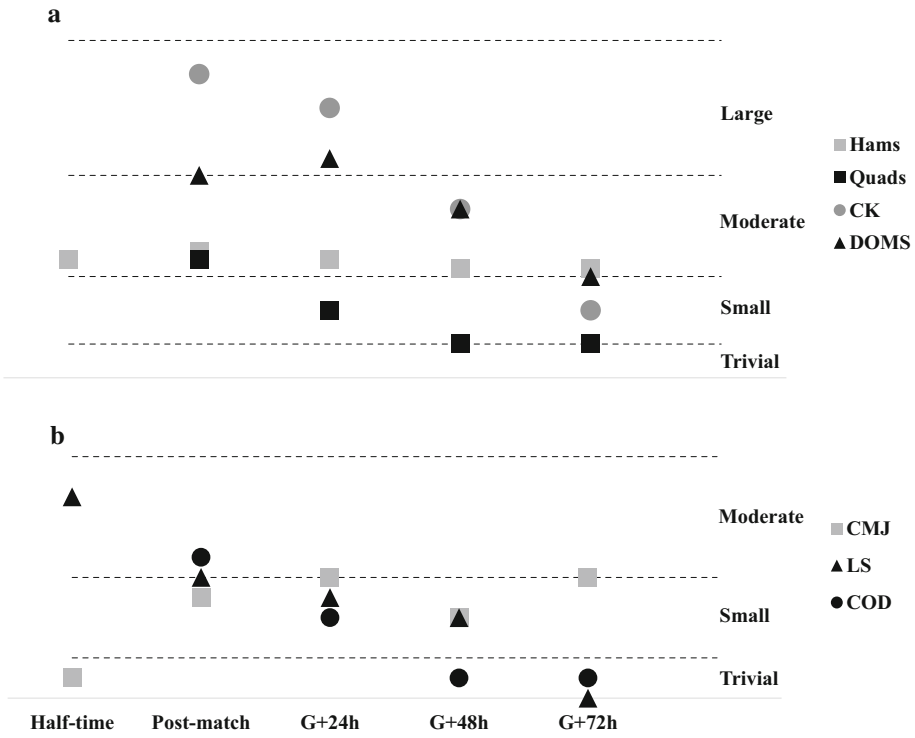


**Fig. 2** Time-course of standardized changes (average weighted effect sizes) in neuromuscular, biochemical and perceptual measures. Time points: half-time, immediately (Post), one (G + 24 h), two (G + 48 h) and three (G + 72 h) days after the match. **a** Hamstrings (Hams) and quadriceps (Quads) muscle strength, creatine kinase (CK) and delayed onset muscle soreness (DOMS); **b** physical performance assessed by the countermovement jump (CMJ), straight line sprint measures (SL) and change of direction ability (COD)

[25, 75, 92]. Nevertheless, cortisol and testosterone hormones responses were also examined in saliva (one study, seven players, 2 ES) [88]. The relationship between anabolic and catabolic hormones (testosterone/cortisol ratio, T/C, three studies, 36 players, 7 ES) [25, 88, 92] was also computed by the observed plasmatic (two studies, 29 players, six ES) [25, 92] and salivary concentrations of these steroid hormones (one study, seven players, 1 ES) [88] both in bound/total [25] and unbound/free fractions [88, 92, 107].

Overall, throughout the recovery period and until G + 72 h, there were substantial alterations in endocrine-related markers.

### 3.4 Physical Performance

The most common dependent variables measured in the selected studies were lower limb muscle function (Table 5, 22 studies, 290 players, 245 ES), followed by straight-line sprint (13 studies, 164 players, 103 ES), vertical jump (14 studies, 183 players, 56 ES), COD (five studies, 64 players, 17 ES), IE (six studies, 74 players, 15 ES), repeated-sprint ability (six studies, 64 players, 13 ES; Table 5), balance-related parameters (three studies, 31 players, 12 ES) and intermittent endurance performance (two studies, 30 players, 2 ES). These variables were mainly measured at Post (228 ES) and/or G + 24 h (58 ES).

#### *3.4.1 Lower Limb Muscle Function*

Lower limb muscle function was mainly assessed using knee joint related indices (211 ES; Table 4, Fig. 2) and included both single joint isokinetic (concentric and eccentric muscle actions, 89 ES and 41 ES, respectively) and isometric muscle actions (31 ES).

Overall, throughout the recovery period until G + 72 h, there was evidence of greater residual fatigue at G + 72 h in knee flexors compared to knee extensors muscle group (Table 4). This difference was of greater magnitude during isometric and eccentric muscle actions (e.g. knee flexors at G + 72 h).

*3.4.1.1 Knee Extensor Muscle Function* Maximal force production capacity of the knee extensors was investigated during concentric (Table 4, concQuads, 12 studies, 168 players, 47 ES) [25, 30, 108–116], isometric (MVICQuads, four studies, 57 players, 14 ES) [93, 97, 112, 117] and eccentric contractions (eccQuads, one study, 13 players, 3 ES) [115]. The rate of force development (RFD, one study, nine players, 4 ES) [117] and changes in angle-specific strength were also examined for the concQuads action (angle joint/muscle length) (three studies, 47 players, 7 ES) [44, 108, 116].

*3.4.1.2 Knee Flexors Muscle Function* Maximal force production capacity of the knee flexors was assessed during concentric (concHams, ten studies, 147 players, 42 ES;

**Table 4** Alterations in lower limb muscle function parameters during the game and throughout recovery period for all three protocols

| | Velocity | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Concentric actions** | | | | | | | | | | | | | |
| Angle ConcHams | | | | | | 141 | 7 | 7.4 (− 0.4; 10.6) | 0.4 (0.0; 0.6) | | | | |
| | High | | | | | 88 | 4 | 7.9 (4.8; 9.2) | 0.5 (0.3; 0.6) | | | | |
| | Moderate | | | | | 9 | 1 | − 0.4 | 0 | | | | |
| | Low | | | | | 44 | 2 | 10.2 (9.8; 10.6) | 0.3 (0.3; 0.4) | | | | |
| Angle ConcQuads | | | | | | 141 | 7 | 0.5 (− 2.3; 4.4) | 0.2 (− 0.3; 1.2) | | | | |
| | High | | | | | 110 | 5 | − 0.1 (− 2.3; 3.5) | 0.0 (− 0.3; 0.5) | | | | |
| | Moderate | | | | | 9 | 1 | 4.4 | 1.2 | | | | |
| | Low | | | | | 22 | 1 | − 0.6 | − 0.1 | | | | |
| ConcHams | | 95 | 8 | − 9.7 (− 17.4; − 4.9) | − 0.5 (− 0.8; − 0.2) | 378 | 26 | − 9.7 (− 17.3; − 1.2) | − 0.5 (− 1.0; − 0.1) | | | | |
| | High | 36 | 3 | − 12.0 (− 17.4; − 8.9) | − 0.6 (− 0.8; − 0.5) | 193 | 12 | − 9.5 (− 16.7; − 1.2) | − 0.5 (− 1.0; − 0.1) | | | | |
| | Moderate | 46 | 4 | − 7.8 (− 13.3; − 4.9) | − 0.5 (− 0.8; − 0.2) | 55 | 5 | − 8.6 (− 13.5; − 4.8) | − 0.5 (− 0.8; − 0.3) | | | | |
| | Low | 13 | 1 | − 10.2 | − 0.5 | 130 | 9 | − 10.7 (− 17.3; − 7.9) | − 0.6 (− 0.9; − 0.5) | 14 | 2 | − 6.0 (− 7.1; − 4.9) | − 0.3 (− 0.4; − 0.3) |
| ConcQuads | | 125 | 9 | − 6.0 (− 13.1; − 0.7) | − 0.4 (− 0.9; − 0.1) | 444 | 30 | − 7.6 (− 18.1; 2.0) | − 0.5 (− 1.3; 0.1) | | | | |
| | High | 46 | 3 | − 3.3 (− 4.0; − 2.8) | − 0.2 (− 0.2; − 0.1) | 203 | 12 | − 6.9 (− 11.7; 2.0) | − 0.4 (− 0.7; 0.1) | | | | |
| | Moderate | 46 | 4 | − 6.0 (− 13.1; − 0.7) | − 0.5 (− 0.9; − 0.1) | 83 | 7 | − 5.0 (− 15.8; − 0.1) | − 0.3 (− 1.2; 0.0) | | | | |
| | Low | 33 | 2 | − 9.9 (− 9.9; − 9.9) | − 0.6 (− 0.6; − 0.5) | 158 | 11 | − 10.0 (− 18.1; − 4.3) | − 0.7 (− 1.3; − 0.3) | 14 | 2 | − 7.8 (− 9.8; − 5.8) | − 0.3 (− 0.4 ; − 0.3) |
| **Eccentric actions** | | | | | | | | | | | | | |
| Angle EccHams | | | | | | 157 | 8 | 41.3 (9.5; 164.8) | 0.9 (0.3; 3.5) | | | | |
| | High | | | | | 88 | 4 | 20.3 (9.5; 39.1) | 0.5 (0.3; 0.8) | | | | |
| | Moderate | 16 | 1 | 30.1 | 0.6 | 25 | 2 | 100.1 (35.5; 164.8) | 2.0 (0.6; 3.5) | | | | |
| | Low | | | | | 44 | 2 | 24.6 (18.2; 31.0) | 0.5 (0.4; 0.6) | | | | |
| EccHams | | 109 | 8 | − 10.7 (− 17.4; − 6.8) | − 0.6 (− 0.9; − 0.4) | 386 | 26 | − 15.3 (− 24.0; − 6.6) | − 0.7 (− 1.2; − 0.3) | | | | |
| | High | 43 | 3 | − 11.4 (− 17.4; − 8.2) | − 0.5 (− 0.8; − 0.4) | 175 | 11 | − 16.6 (− 24.0; − 6.6) | − 0.8 (− 1.2; − 0.3) | | | | |
| | Moderate | 46 | 4 | − 10.8 (− 16.1; − 6.8) | − 0.6 (− 0.9; − 0.5) | 109 | 9 | − 13.9 (− 20.7; − 6.8) | − 0.7 (− 1.2; − 0.3) | 26 | 2 | − 8.5 (− 10.1; − 6.8) | − 0.7 (− 0.9; − 0.6) |
| | Low | 20 | 1 | − 8 | − 0.4 | 74 | 4 | − 15.6 (− 18.1; − 12.3) | − 0.7 (− 0.9; − 0.5) | | | | |
| EccQuads | | | | | | | | | | | | | |
| | High | 13 | 1 | − 2.3 | − 0.1 | 26 | 2 | − 8.1 (− 9.4; − 6.9) | − 0.4 (− 0.5; − 0.3) | | | | |

**Table 4** continued

| | Velocity | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Isometric actions** | | | | | | | | | | | | | |
| **MVIC Hams** | | 8 | 1 | − 10.2 | − 0.7 | 48 | 4 | − 12.1 (− 24.1; 0.5) | − 0.8 (− 1.4; 0.0) | 40 | 4 | − 8.4 (− 9.9; − 6.3) | − 0.7 (− 0.9; − 0.6) |
| RFD Hams (0–25/30 ms) | | 8 | 1 | − 53.2 | − 1.2 | 17 | 2 | − 43.1 (− 75.9; − 10.3) | − 1.2 (− 2.0; − 0.4) | | | | |
| RFD Hams (0–50 ms) | | 8 | 1 | − 44.6 | − 1.1 | 17 | 2 | − 39.2 (− 71.0; − 7.5) | − 1.2 (− 2.1; − 0.3) | | | | |
| RFD Hams (0–75 ms) | | 8 | 1 | − 36.5 | − 1 | 8 | 1 | − 64.4 | − 2.2 | | | | |
| RFD Hams (0–100 ms) | | 8 | 1 | − 27.2 | − 0.8 | 17 | 2 | − 28.2 (− 54.6; − 1.9) | − 1.0 (− 1.9; − 0.1) | | | | |
| RFD Hams (0–200 ms) | | | | | | 9 | 1 | − 8.6 | − 0.5 | | | | |
| **MVIC Quads** | | | | | | 73 | 6 | − 9.8 (− 13.9; − 2.7) | − 0.7 (− 1.6; − 0.1) | 33 | 3 | − 7.6 (− 9.6; − 5.7) | − 0.4 (− 0.5; − 0.3) |
| RFD Quads (0–30 ms) | | | | | | 9 | 1 | − 9.6 | − 0.6 | | | | |
| RFD Quads (0–50 ms) | | | | | | 9 | 1 | − 7 | − 0.5 | | | | |
| RFD Quads (0–100 ms) | | | | | | 9 | 1 | − 5.2 | − 0.3 | | | | |
| RFD Quads (0–200 ms) | | | | | | 9 | 1 | − 7.7 | − 0.4 | | | | |
| **Muscle joint balance** | | | | | | | | | | | | | |
| ConcHams/ConcQuads | | | | | | 214 | 12 | − 1.8 (− 9.7; 3.1) | − 0.2 (− 0.8; 0.2) | | | | |
| | High | 26 | 2 | − 7.8 (− 8.1; − 7.5) | − 0.6 (− 0.7; − 0.5) | 114 | 6 | − 0.9 (− 9.7; 3.1) | − 0.1 (− 0.6; 0.2) | | | | |
| | Small | | | | | 78 | 5 | − 2.6 (− 4.6; − 1.6) | − 0.3 (− 0.8; − 0.1) | 14 | 2 | 3.1 (2.2; 4.0) | 0.4 (0.3; 0.5) |
| EccHams/ConcQuads | | 89 | 6 | − 9.1 (− 19.6; 2.6) | − 0.5 (− 1.0; 0.1) | 301 | 18 | − 10.2 (− 22.9; 0.0) | − 0.6 (− 1.2; 0.0) | | | | |
| | High | 53 | 4 | − 12.3 (− 19.6; − 4.6) | − 0.6 (− 1.0; − 0.2) | 141 | 8 | − 12.1 (− 22.9; − 4.7) | − 0.7 (− 1.2; − 0.3) | | | | |
| | Moderate | 16 | 1 | − 8.2 | − 0.5 | 53 | 4 | − 12.3 (− 15.3; − 8.0) | − 0.7 (− 0.8; − 0.4) | | | | |
| | Low | 20 | 1 | 2.6 | 0.1 | 85 | 5 | − 5.4 (− 13.0; 0.0) | − 0.3 (− 0.8; 0.0) | | | | |

| | Velocity | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Concentric actions** | | | | | | | | | |
| Angle ConcHams | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | | | | | | | | |
| | Low | | | | | | | | |
| Angle ConcQuads | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | | | | | | | | |
| | Low | | | | | | | | |

**Table 4** continued

| | Velocity | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| ConcHams | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | | | | | | | | |
| | Low | 14 | 2 | − 3.5 (− 4.1; − 2.8) | − 0.2 (− 0.3; − 0.2) | 31 | 4 | − 1.7 (− 3.5; 0.0) | − 0.1 (− 0.2; 0.0) |
| ConcQuads | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | | | | | | | | |
| | Low | 14 | 2 | − 3.6 (− 4.7; − 2.5) | − 0.2 (− 0.2; − 0.1) | 31 | 4 | − 2.7 (− 4.2; − 1.7) | − 0.2 (− 0.4; − 0.1) |
| **Eccentric actions** | | | | | | | | | |
| Angle EccHams | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | | | | | | | | |
| | Low | | | | | | | | |
| EccHams | | | | | | | | | |
| | High | | | | | | | | |
| | Moderate | 26 | 2 | − 7.8 (− 9.5; − 6.1) | − 0.7 (− 0.9; − 0.6) | | | | |
| | Low | | | | | | | | |
| EccQuads | | | | | | | | | |
| | High | | | | | | | | |
| **Isometric actions** | | | | | | | | | |
| **MVIC Hams** | | 40 | 4 | − 8.0 (− 8.8; − 6.7) | − 0.6 (− 0.8; − 0.5) | 40 | 4 | − 6.9 (− 8.3; − 5.7) | − 0.6 (− 0.7; − 0.5) |
| RFD Hams (0–25/30 ms) | | | | | | | | | |
| RFD Hams (0–50 ms) | | | | | | | | | |
| RFD Hams (0–75 ms) | | | | | | | | | |
| RFD Hams (0–100 ms) | | | | | | | | | |
| RFD Hams (0–200 ms) | | | | | | | | | |
| **MVIC Quads** | | 33 | 3 | − 3.3 (− 6.5; 2.4) | − 0.2 (− 0.4; 0.1) | 14 | 2 | − 4.0 (− 4.4; − 3.7) | − 0.2 (− 0.3; − 0.2) |
| RFD Quads (0–30 ms) | | | | | | | | | |
| RFD Quads (0–50 ms) | | | | | | | | | |
| RFD Quads (0–100 ms) | | | | | | | | | |
| RFD Quads (0–200 ms) | | | | | | | | | |
| **Muscle joint balance** | | | | | | | | | |
| ConcHams/ConcQuads | | | | | | | | | |
| | High | | | | | | | | |
| | Small | 14 | 2 | − 0.5 (− 0.9; 0.0) | − 0.1 (− 0.1; 0.0) | 14 | 2 | − 0.3 (− 0.6; 0.0) | 0.0 (− 0.1; 0.0) |
| EccHams/ConcQuads | | | | | | | | | |

**Table 4** continued

| Velocity | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| High | | | | | | | | |
| Moderate | | | | | | | | |
| Low | | | | | | | | |

*N* number of observations, *ES* corrected effect size, *ConcHams* knee flexors peak torque during concentric actions, *ConcQuads* knee extensors peak torque during concentric actions, *EccHams* knee flexors peak torque during eccentric actions, *EccQuads* knee extensors peak torque during eccentric actions, *MVIC Hams* maximal voluntary isometric contraction of the hamstrings muscles, *MVIC Quads* maximal voluntary isometric contraction of the extensors muscles, *RFD Hams* rate force development of knee flexors, *RFD Quads* rate force development of knee extensors, *ConcHams/ConcQuads* ratio of the peak torque of ConcHmas and ConcQuads, *EccHams/ConcQuads* ratio of the peak torque of EccHmas and ConcQuads

Table 4) [25, 30, 44, 50, 108, 109, 112, 115, 116, 118], isometric ($MVIC_{Hams}$, four studies, 49 players 17 ES) [95, 112, 117, 119] and eccentric muscle actions (eccHams, ten studies, 137 players, 38 ES) [44, 50, 108–110, 113, 115, 116, 120, 121].

The hamstrings RFD (two studies, 17 players, 11 ES) [117, 119] and angle-specific strength changes during eccentric and concentric (three studies, 47 players, 15 ES) maximal muscle actions were reported [44, 108, 116].

*3.4.1.3 Knee Joint Muscle Balance* Match-induced alterations in knee joint muscles balance were reported through computation of conventional (concQuads/concHams, five studies, 74 players, 12 ES) [25, 109, 112, 115, 116] and functional ratios (eccHams/concQuads, nine studies, 136 players, 18 ES) [44, 108–110, 112, 113, 115, 116, 120].

*3.4.1.4 Ankle Joint Muscle Function* The effect of match-play on plantar flexors (PF) muscle function was also investigated (one study, 17 players, 2 ES) [27]. In this regard, no clear change in PF maximal voluntary contraction force was reported. Nevertheless, a substantial loss in PF RFD qualities (peak rate torque development; $\Delta = -7.4\%$ to $-13.4\%$, ES $= -0.2$ to $-0.4$) was observed at G + 48 h.

*3.4.1.5 Muscle Contractility* Intrinsic muscle properties (e.g. nerve stimulation techniques) have been investigated (five studies, 61 players, 32 ES) [27, 49, 93, 117, 119]. A small reduction in quadriceps peak twitch was reported at half-time ($\Delta = -16.2\%$, ES $= -0.6$) [49]. At Post there was an impairment of small magnitude at the lower stimulation intensities (1 Hz and 10 Hz; $\Delta = -7.8\%$ and $-9\%$, ES $= -0.4$, respectively) but trivial at higher intensity (100 Hz, $\Delta = -3.4\%$, ES $= -0.2$) [93]. Furthermore, within the recovery period (48-h length), values returned to near baseline independently of the evoked stimulation frequency. Plantar flexors peak twitch values displayed a small but prolonged depression until at least G + 48 h ($\Delta = -12.3$ to $-23\%$, ES $= -0.3$ to $-0.6$) [27].

*3.4.1.6 Alterations in Muscle Activity* Alterations in muscle activity have been investigated by recordings of electromyographic activity (EMG) of the quadriceps, hamstrings and plantar flexors muscles and maximal voluntary activation level during isometric muscle contractions.

Knee extensors and flexors muscles during MVIC showed a reduction in peak and average EMG amplitude at Post [117]. A reduction of moderate and small magnitudes was observed for the biceps femoris caput longum (BF,

**Table 5** Alterations in physical performance parameters during and throughout the 72-h recovery period

| | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range)Δ% (range) | ES (range) |
| **LSP (time)** | 34 | 2 | 2.9 (2.7–3.2) | 1.0 (0.9; 1.1) | 219 | 18 | 3.5 (0.7; 7.8) | 0.6 (0.1; 1.2) | 132 | 11 | 2.7 (− 0.3; 7.4) | 0.5 (0.0; 0.9) |
| 5 m | | | | | 7 | 1 | 4.9 | 0.1 | 7 | 1 | − 0.3 | 0 |
| 10 m | 34 | 2 | 2.9 (2.7; 3.2) | 1.0 (0.9; 1.1) | 29 | 3 | 2.2 (0.7; 3.6) | 0.5 (0.4; 0.7) | 16 | 2 | 2.4 (1.8; 3.0) | 0.5 (0.3; 0.6) |
| 15 m flying phase | | | | | 19 | 1 | 1.4 | 0.3 | 19 | 1 | 0.9 | 0.3 |
| 15 m | | | | | 67 | 4 | 5.7 (2.5; 7.8) | 0.8 (0.6; 1.0) | 67 | 4 | 4.4 (1.2; 7.4) | 0.6 (0.3; 0.9) |
| 20 m | | | | | 30 | 3 | 2.5 (1.4; 3.5) | 0.8 (0.7; 0.9) | | | | |
| 30 m | | | | | 17 | 2 | 2.2 (0.7; 3.8) | 0.6 (0.1; 1.0) | 7 | 1 | − 0.1 | 0 |
| 60 m | | | | | 50 | 4 | 3.1 (1.4; 5.2) | 0.7 (0.3; 1.2) | 16 | 2 | 2.4 (2.0; 2.8) | 0.6 (0.5; 0.6) |
| 6-s best sprint (mean speed) | 26 | 2 | 1.2 (0.4; 1.9) | 0.1 (0.0; 0.2) | 26 | 2 | 2.3 (1.7; 3.0) | 0.2 (0.2; 0.3) | 10 | 1 | − 0.8 | − 0.1 |
| 6-s best sprint (MPO) | | | | | 34 | 3 | − 0.9 (− 5.8; 1.7) | − 0.1 (− 0.7; 0.2) | 44 | 4 | − 0.1 (− 5.7; 4.3) | − 0.1 (− 0.7; 0.4) |
| 6-s best sprint (peak speed) | | | | | 26 | 2 | − 1.9 (− 2.0; − 1.8) | − 0.2 (− 0.3 to− 0.2) | 36 | 3 | − 1.7 (− 3.8; − 0.5) | − 0.3 (− 0.7; − 0.1) |
| **COD** | | | | | 88 | 6 | 2.1 (0.2; 4.2) | 0.7 (0.1; 1.1) | 61 | 5 | 1.3 (0.8; 2.1) | 0.4 (0.2; 0.5) |
| 20-m shuttle | | | | | 19 | 1 | 4.2 | 0.9 | 19 | 1 | 0.9 | 0.3 |
| 40-m shuttle | | | | | 19 | 1 | 2.8 | 0.8 | 19 | 1 | 1.2 | 0.4 |
| L-AR | | | | | 50 | 4 | 1.4 (0.2; 2.2) | 0.6 (0.1; 1.1) | 16 | 2 | 1.1 (0.8; 1.4) | 0.3 (0.2; 0.4) |
| t-test | | | | | | | | | 7 | 1 | 2.1 | 0.5 |
| **Jump ability** | | | | | | | | | | | | |
| CMJ | 42 | 3 | − 1.1 (− 4.2; 1.0) | − 0.1 (− 0.4; 0.1) | 163 | 13 | − 5.5 (− 11.9; 2.9) | − 0.5 (− 1.1; 0.2) | 89 | 8 | − 5.9 (− 10.4; − 2.6) | − 0.6 (− 1.1; − 0.2) |
| SJ | 8 | 1 | − 5.2 | − 0.4 | 42 | 4 | − 6.2 (− 8.6; − 2.6) | − 0.6 (− 0.8; − 0.3) | 34 | 3 | − 1.4 (− 3.3; 2.1) | − 0.2 (− 0.3; 0.2) |
| $CMJ_{15s}$ | | | | | 10 | 1 | − 2.2 | − 0.6 | | | | |
| Reactive Strength Index | | | | | 16 | 2 | − 4.7 (− 4.7; − 4.7) | − 0.2 (− 0.2; − 0.2) | 16 | 2 | 8.2 (7.0; 9.4) | 0.3 (0.3; 0.4) |
| **Repeated sprint ability** | | | | | | | | | | | | |
| RSA FI (3 × 30 m/25 s) | | | | | 8 | 1 | 252.9 | 1.6 | | | | |
| RSA FI (5 × 30 m/25 s) | | | | | 10 | 1 | 6.8 | 1.6 | | | | |
| RSA mean speed (3 × 30 m/25 s) | | | | | 8 | 1 | 2.6 | 0.7 | | | | |
| RSA mean time (3 × 30 m/25 s) | 16 | 2 | 0.9 (0.7; 1.1) | 0.3 (0.3; 0.4) | 39 | 3 | 2.8 (2.0; 4.1) | 0.7 (0.6; 0.8) | | | | |
| RSA mean time (5 × 30 m/25 s) | | | | | 10 | 1 | 3.9 | 1 | | | | |
| RSA (MPO) (6 × 6 s/20 s) | 8 | 1 | − 4.6 | − 0.6 | 8 | 1 | 2.4 | 0.5 | | | | |

**Table 5** continued

| | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range)Δ% (range) | ES (range) |
| **Intermittent Exercise** | | | | | | | | | | | | |
| YYIE2 | | | | | 23 | 1 | − 38.3 | − 1.5 | | | | |
| YYIR2 | | | | | | | | | | | | |

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **LSP (time)** | 84 | 8 | 1.5 (0.1; 2.9) | 0.4 (0.0; 0.9) | 17 | 2 | − 0.3 (− 0.6; 0.0) | − 0.1 (− 0.2; 0.0) |
| 5 m | 7 | 1 | 0.2 | 0 | | | | |
| 10 m | 16 | 2 | 2.1 (1.8; 2.4) | 0.5 (0.3; 0.6) | | | | |
| 15 m flying phase | 19 | 1 | − 0.5 | − 0.1 | | | | |
| 15 m | 19 | 1 | 1.2 | 0.4 | | | | |
| 20 m | | | | | 17 | 2 | − 0.3 (− 0.6; 0.0) | − 0.1 (− 0.2; 0.0) |
| 30 m | 7 | 1 | 0.1 | 0 | | | | |
| 60 m | 16 | 2 | 2.4 (1.9; 2.9) | 0.7 (0.5; 0.9) | | | | |
| 6-s best sprint (mean speed) | 36 | 3 | 0.5 (− 2.2; 2.1) | 0.0 (− 0.3; 0.2) | 10 | 1 | − 1.8 | − 0.2 |
| 6-s best sprint (MPO) | 44 | 4 | − 0.7 (− 7.3; 3.4) | − 0.1 (− 0.9; 0.3) | 18 | 2 | − 3.2 (− 5.1; − 1.3) | − 0.4 (− 0.7; − 0.1) |
| | 36 | 3 | − 2.1 (− 5.0; − 0.5) | − 0.4 (− 0.9; − 0.1) | 10 | 1 | − 4.8 | − 0.9 |
| **COD** | 61 | 5 | 0.2 (− 1.2; 1.0) | 0.1 (− 0.4; 0.4) | 7 | 1 | 0.4 | 0.1 |
| 20-m shuttle | 19 | 1 | 0.5 | 0.1 | | | | |
| 40-m shuttle | 19 | 1 | 0.6 | 0.2 | | | | |
| L-AR | 16 | 2 | 0.5 (0.0; 1.0) | 0.2 (0.0; 0.4) | | | | |
| *t*-test | 7 | 1 | − 1.2 | − 0.4 | 7 | 1 | 0.4 | 0.1 |
| **Jump ability** | | | | | | | | |
| CMJ | 89 | 8 | − 3.2 (− 9.3; 4.8) | − 0.4 (− 1.0; 0.4) | 42 | 5 | − 4.2 (− 6.9; − 0.5) | − 0.5 (− 1.0; 0.0) |
| SJ | 34 | 3 | − 2.8 (− 4.8; − 1.3) | − 0.3 (− 0.5; − 0.1) | 8 | 1 | − 3.1 | − 0.4 |
| $CMJ_{15s}$ | | | | | | | | |
| Reactive Strength Index | 16 | 2 | 11.3 (6.3; 16. | 0.5 (0.3; 0.7) | | | | |
| **Repeated sprint ability** | | | | | | | | |
| RSA FI (3 × 30 m/25 s) | | | | | | | | |
| RSA FI (5 × 30 m/25 s) | | | | | | | | |
| RSA mean speed (3 × 30 m/25 s) | | | | | | | | |
| RSA mean time (3 × 30 m/25 s) | | | | | 7 | 1 | 0 | 0 |
| RSA mean time (5 × 30 m/25 s) | | | | | | | | |

**Table 5** continued

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| RSA (MPO) (6 × 6 s/20 s) | 8 | 1 | 1.1 | 0.2 | | | | |
| **Intermittent Exercise** | | | | | | | | |
| YYIE2 | | | | | | | | |
| YYIR2 | | | | | 7 | 1 | − 8.6 | − 0.4 |

*N* number of observations, *ES* corrected effect size, *LSP* linear sprint time, *MPO* mean power output, *L-AR* L agility test, *CMJ* countermovement jump, *SJ* squat jump, $CMJ_{15S}$ countermovement jumps during 15 s, *RSA* repeated sprint ability test, *RSA FI* repeated sprint ability fatigue index, *YYIE2* Yo–Yo intermittent endurance test level two, *YYIR2* Yo–Yo intermittent recovery test level two

Δ = − 30 and − 31%, ES = − 0.9 and − 0.7) and semitendinosus (ST, Δ = − 18 and − 21%, ES = − 0.6 and − 0.5) at this time-point. A decrease in EMG activity was only observed for the vastus lateralis (VL, Δ = − 5% and − 17%, ES = − 0.3 and − 0.9) and not for rectus femoris during knee extensors MVIC (RF, Δ = − 3% and − 5%, ES = − 0.1, for peak and average EMG, respectively). A depression in the EMG root mean square was observed at Post and G + 24 h (Δ = − 12% and − 9%, ES = − 0.5 and − 0.4, respectively) [93]. These alterations were rated as trivial at G + 48 h (Δ = − 4%, ES = − 0.1) [93]. Additionally, the EMG/PPA ratio was reduced at Post only (Δ = − 12.3%, ES = − 0.4) [93].

Small increases were observed in the different time phases of the RFD (0–30, 0–50, 0–100 and 0–200 ms) for VL (Δ = 114% and 106%, ES = 0.5 and 0.3, respectively for 0–100 and 0–200 ms) and RF (Δ = 114% and 106%, ES = 0.5 and 0.3, respectively for 0–100 and 0–200 ms) at Post. Interestingly, although ST was not affected, a moderate depression in BF muscle activity occurred during the very early phase of the contraction (0–30 ms, Δ = − 72% and ES = 0.6) [117].

There were no clear changes in the maximum voluntary activation (VA) of hamstrings muscles [119]. A moderate to large decreases in normalized EMG BF activity (Δ = − 31% and − 44%, ES = − 0.9 and − 1.4, at half-time and Post) were reported and trivial to small changes were observed for medial hamstrings (Δ = − 6.4% and − 17%, ES = − 0.1 and − 0.3) [119]. Moderate to very large decreases in VA for quadriceps (Δ = − 8.5%, ES = − 0.8) [93], and PL (Δ = − 1.6%, ES = − 3.2, two ES) [27] muscles were reported at Post. Furthermore, it seemed that the recovery of the motor output measured by VA (neural input reaching the neuromuscular junction) was effectively recovered for the PF (Δ = 0.05%, ES = 0.3, 2 ES) at G + 24 h but some small impairment was still observed for quadriceps muscles at this time-point (Δ = − 3.7%, ES = − 0.4) with full recovery achieved at G + 48 h.

### 3.4.2 Straight-line Sprinting Performance

The match impact on player's straight-line sprint (SL; Table 5, Fig. 2) performance was investigated over predetermined sprinting distances ranging from 5 to 60 m (13 studies, 164 players, 103 ES) [19, 25, 30, 66, 93–96, 98, 121–124]. Generally, the impairments were moderate at Post, still small at G + 24 h and G + 48 h and unclear at G + 72H. Mean speeds, peak speeds and mean power outputs recorded during a 6-s sprint were still slightly to moderately affected at G + 72 h (− 1.8 to − 4.8%) [94, 95, 121]. Furthermore, post-match maximal sprint velocity and maximal horizontal power production during a 20-m sprint was impaired to a greater

extent ($\Delta = 4\%$ and 3.3%) than maximal horizontal force production ($\Delta = 0.8\%$) [122]. Additionally, very large changes occurred in hip biomechanics (hip flexion and extension angles, $\Delta = -15\%$ and $-80\%$, ES $= -1.5$ and $-2.9$) during a 10-m acceleration phase at post-match [54].

#### 3.4.3 Change of Direction Ability

A wide variety of COD tests have been applied (five studies, 64 players,17 ES) and consisted of shuttle sprint test [93], *t* test [25] and the L-agility run test [66, 123].

Match-induced fatigue results in a moderate decline in COD ability at Post, with a small reduction in performance at G + 24 h and no clear change the following days (Table 5, Fig. 2). Knee kinematics (e.g. range of joint movement during knee flexion phase) during COD moderately to largely changed at half-time and post-match ($\Delta = -34\%$ to 36%, ES $= -1.8$ to 1.5) [125].

#### 3.4.4 Jumping Ability

The scientific literature investigating the effects of soccer-match on muscle power has extensively relied on information obtained during different jump performances (14 studies, 183 players, 56 ES; Table 5, Fig. 2) [25, 30, 49, 66, 91, 92, 94, 95, 97, 117, 121, 123, 124, 126]. These studies suggested that match-induced fatigue impairs squat jump (three studies, 29 players, 12 ES) [49, 94, 121] and CMJ (12 studies, 151 players, 37 ES) [25, 30, 49, 66, 72, 92, 94, 95, 117, 121, 123, 124] performance until G + 72 h with a small but consistent effect. Small decrements in multiple CMJs performance (one study, ten players, 1 ES) [126] and in reactive strength index (one study, eight players, 6 ES) at post have been described [66]. Generally, force and power generating capacities were preserved during CMJ performance. No clear changes in absolute and relative force during CMJ were reported from Post to G + 48 h [92]. Similarly, there were no clear changes in relative peak eccentric and concentric forces, in mean force during all jumping movement phases [117], in power-related variables [92, 117] and RFD ($\Delta = 3.5\%$, ES $= 0.2$) [117]. No substantial changes in relative and absolute peak power output (PPO) values were observed from Post to G + 72 h [92, 117]. Nevertheless, others observed a small to moderate decreases in CMJ PPO values at G + 24 h and G + 48 h ($\Delta = -6.6\%$ and $-2.7\%$, ES $= -0.6$ and 0.3) [91]. Additionally, a trivial to small changes have been observed in the center of mass displacement during concentric ($\Delta = -3.4\%$, ES $= -0.2$) and eccentric ($\Delta = -7.9\%$, ES $= 0.3$) CMJ phases [117].

#### 3.4.5 Balance

The influence of match-related fatigue on balance-related parameters (three studies, 31 players, 12 ES) has been investigated [127–129]. Postural stability tasks have been investigated by the performance of unilateral stance tests [127–129]. In this regard, a moderate increase in reaction times during the SMART EquiTest Single-legged dynamic balance test was observed at half-time ($\Delta = 33$–38%, ES $= 1.1$–1.6) [128]. A decrease in postural stability was also observed at Post. Moderate and large increases in center of gravity sway velocity in the opened eyes condition for the dominant and non-dominant leg were also reported ($\Delta = 25\%$ and 43%, ES $= 0.9$–1.8) [129]. Nevertheless, other authors observed small deteriorations on stability indexes (antero-posterior and medio-lateral) during 30-s single-legged balance test at half-time ($\Delta = -8\%$ and 9%, ES $= -0.2$ and 0.3). There was no clear change in the overall index [127]. This preservation of postural control was extended at match-end [127] when measured by stability indexes and platform deflection in the antero-posterior plane. Interestingly, a substantial alteration was reported in the medio-lateral plane ($\Delta = 184\%$, ES $= 0.3$).

#### 3.4.6 Repeated Sprint Ability

The match impact on player's ability to repeatedly perform sprint actions (e.g. 5 × 30-m sprints with 25 s of recovery) has been investigated (6 studies, 64 players, 15 ES; Table 5) [72, 94, 97, 98, 126, 130]. Repeated sprint protocols have consisted in measurements of mean sprint times (four studies, 48 players, 7 ES) [72, 97, 98, 130] and fatigue indexes (two studies, 18 players, 2 ES) [98, 126] during RSA protocols. Kinematic (mean speed, one study, eight players, 1 ES) [126] and kinetic (mean power output, one study, eight players, 3 ES) [94] variables recorded during RSA were also documented. At Post, there was a moderate and large impairments in mean sprint time and fatigue indexes, respectively. RSA was not different from baseline values at G + 48 h and G + 72-h [94, 97].

#### 3.4.7 Intermittent Endurance Exercise (IE)

The match-induced fatigue in intermittent exercise capacity has been based on the assessment of player's ability to perform the YYIE2 and YYIR2 (two studies, 30 players, 2 ES) [72, 97]. There was a large impairment in YYIE2 performance at Post (38.3%). IE was still slightly affected at G + 72-h when evaluated from YYIE2 [97].

**Table 6** Technical and perceptual response during the match and throughout the 72-h recovery period for all three protocols

| | Baseline-half | | | | Baseline-post | | | | Baseline-24 h | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Technical ability** | | | | | | | | | | | | |
| LSPT penalties | 79 | 5 | 12.8 (− 39.1; 42) | 0.3 (− 0.2; 1.1) | 130 | 8 | 45.6 (3.2; 156.5) | 0.5 (0.1; 1.4) | 19 | 1 | 16.8 | 0.4 |
| LSPT time | 79 | 5 | 0.9 (− 3.5; 4.8) | 0.1 (− 0.4; 0.7) | 130 | 8 | 1.0 (− 2.2; 5.4) | 0.1 (− 0.3; 0.8) | 19 | 1 | − 0.3 | 0 |
| LSPT total performance | 79 | 5 | 0.1 (− 7.7; 15.5) | 0.0 (− 0.5; 1.1) | 130 | 8 | 4.9 (− 3.1; 19.8) | 0.3 (− 0.2; 1.4) | 19 | 1 | 3 | 0.4 |
| LSST mean shot speed | | | | | 32 | 2 | − 0.1 (− 0.1; 0.0) | 0 | | | | |
| LSST shooting | | | | | 32 | 2 | 1.2 (0.7; 1.7) | 0.5 (0.3; 0.6) | | | | |
| **Perceptual measures** | | | | | | | | | | | | |
| DOMS General | | | | | 51 | 5 | 40.2 (19.4; 72.0) | 1.2 (0.5; 1.8) | 44 | 4 | 52.5 (19.4; 92.0) | 1.3 (0.6; 2.6) |
| DOMS lower limbs | | | | | 51 | 1 | 250.5 | 2.7 (1.9; 4.1) | 41 | 2 | 183.1 (64.2; 302.1) | 1.3 (0.6; 2.0) |
| DOMS Quads | | | | | 33 | 4 | 967.0 (66.7; 3100.0) | 3.1 (0.5; 8.5) | 23 | 3 | 728.2 (141.7; 1700.0) | 1.0 (0.5; 1.3) |
| Hooper (fatigue) | | | | | 34 | 3 | 46.7 (25.7; 84.6) | 1.1 (0.8; 1.6) | 44 | 4 | 37.0 (13.5; 65.4) | 0.9 (0.5; 1.4) |
| Hooper (sleep) | | | | | | | | | 44 | 4 | 14.2 (− 2.9; 37.5) | 0.3 (− 0.1; 0.6) |
| Hooper (stress) | | | | | 34 | 3 | 39.8 (4.0; 106.3) | 0.5 (0.1; 1.2) | 44 | 4 | 21.8 (0.0; 56.3) | 0.2 (0.0; 0.5) |
| TQR | | | | | | | | | 36 | 3 | − 8.9 (− 15.5; − 4.2) | − 0.6 (− 1.1; − 0.3) |

| | Baseline-48 h | | | | Baseline-72 h | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | ES | Δ% (range) | ES (range) | *N* | ES | Δ% (range) | ES (range) |
| **Technical ability** | | | | | | | | |
| LSPT penalties | 19 | 1 | 14.7 | 0.4 | | | | |
| LSPT time | 19 | 1 | − 1 | − 0.2 | | | | |
| LSPT total performance | 19 | 1 | 2.8 | 0.3 | | | | |
| LSST mean shot speed | | | | | | | | |
| LSST shooting | | | | | | | | |
| **Perceptual measures** | | | | | | | | |
| DOMS general | 44 | 4 | 37.6 (11.1; 81.0) | 1.0 (0.4; 1.8) | 35 | 4 | 27.9 (0.0; 66.7) | 0.6 (0.0; 1.0) |
| DOMS lower limbs | 19 | 1 | 56.9 | 0.5 | 32 | 2 | 0.0 (0.0; 0.0) | 1.3 (0.9; 1.7) |
| DOMS quads | 23 | 3 | 411.9 (50.0; 1000.0) | 0.6 (0.6; 0.7) | 7 | 1 | 1300 | 1.3 |
| Hooper (fatigue) | 44 | 4 | 15.8 (0.0; 38.5) | 0.4 (0.0; 1.1) | 18 | 2 | 11.3 (7.7; 14.8) | 0.3 (0.2; 0.4) |
| Hooper (sleep) | 44 | 4 | 10.6 (− 7.1; 25.7) | 0.3 (− 0.2; 0.8) | 18 | 2 | 8.5 (8.3; 8.6) | 0.2 (0.1; 0.3) |
| Hooper (stress) | 44 | 4 | 20.3 (− 8.0; 75.0) | 0.3 (− 0.2; 1.1) | 18 | 2 | 12.5 (0.0; 25.0) | 0.2 (0.0; 0.4) |
| TQR | 36 | 3 | − 1.6 (− 6.2; 5.6) | 0.1 (− 0.3; 0.5) | 10 | 1 | − 4.9 | − 0.4 |

*N* number of observations, *ES* corrected effect size, *LSPT* Loughborough soccer passing test, *LSST* Loughborough soccer shooting test, *DOMS* delayed-onset muscle soreness, *TQR* total quality recovery scale

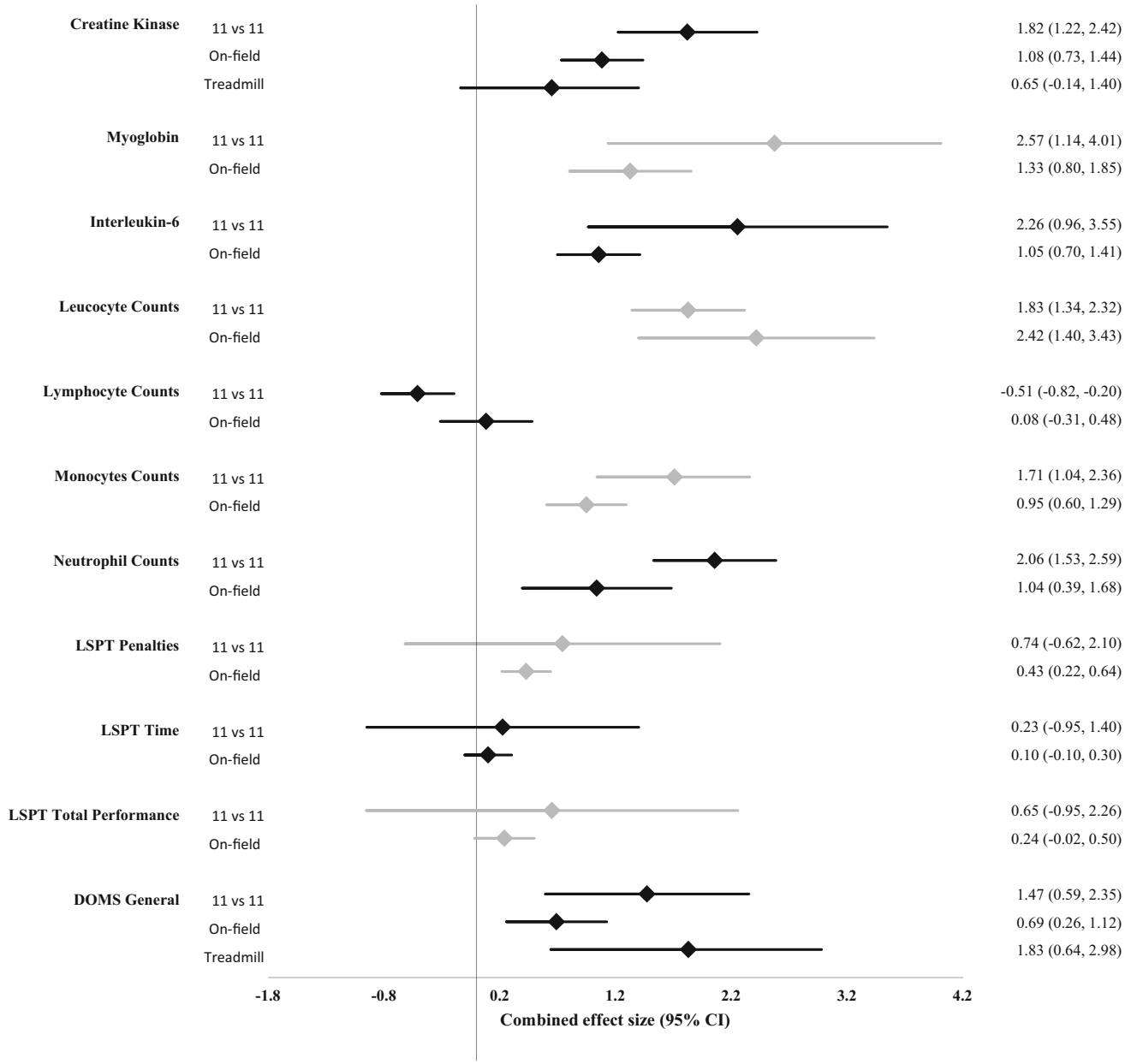


**Fig. 3** Biochemical, technical and perceptual outcomes combined effect size (95% confidence interval) at post-protocol time-point. *LSPT* Loughborough soccer passing test, *DOMS* delayed onset muscle soreness

### 3.5 Technical Performance

Technical performance parameters (six studies, 92 players, 49 ES; Table 6) were mostly measured at half-time (15 ES) and/or at Post (28 ES) consisting of the Loughborough soccer passing test (LSPT; four studies, 82 players, 45 ES) [80, 81, 93, 131–133] and/or the Loughborough soccer shooting test (LSST; one study, 16 players, four ES) performances [80].

Independently of the time-points, the effect of match-induced fatigue on technical performance metrics was trivial to small. The effects of soccer-induced fatigue on biomechanical indices of soccer kick performance [134] showed that maximal ground reactions forces (vertical, horizontal and lateral) were not substantially affected. Additionally, moderate changes in ankle joint angular position ($\Delta = -8.8\%$ and $-10.5\%$, ES $= -0.8$ to $-1.1$) occurred at half-time and Post. Match-fatigue resulted in small alterations (swinging leg at impact) in knee and hip angular positions at half-time and Post ($\Delta = 3.8$–$4.2\%$, ES $= 0.3$–$0.5$). There were no clear changes at the other time-points. Additionally, small changes in the angular velocity of the hip ($\Delta = 20\%$, ES $= 0.4$) and the knee ($\Delta = -15\%$, ES $= -0.5$) were observed at Post. Ball to foot center of mass speed ratio (Vball/Vfoot ratio) of the swinging

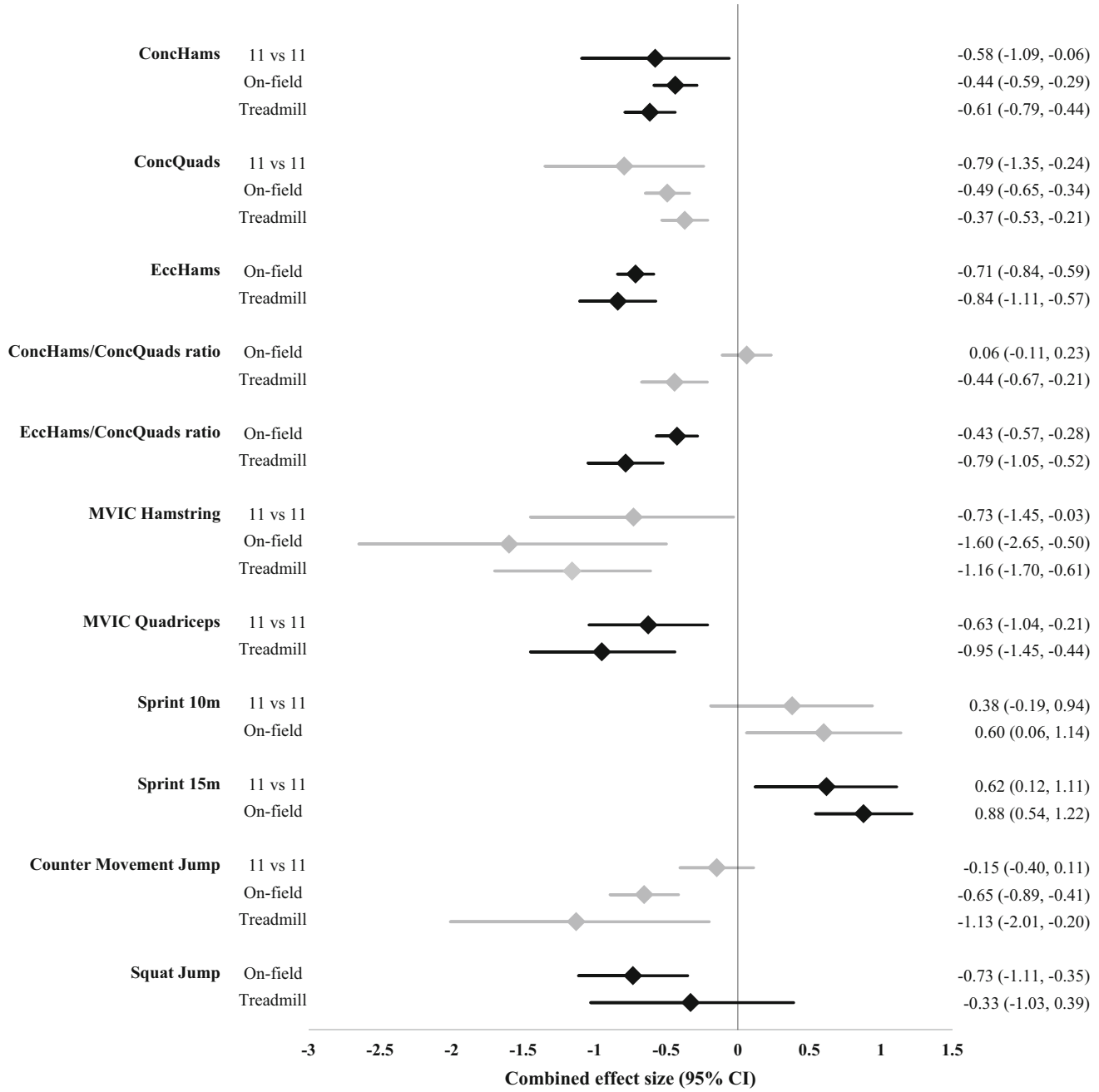


**Fig. 4** Lower limb muscle function and physical performance outcomes combined effect size (95% confidence interval) at post-protocol time-point. *ConcHams* knee flexors peak torque during concentric actions, *ConcQuads* knee extensors peak torque during concentric actions, *EccHams* knee flexors peak torque during eccentric actions, *EccQuads* knee extensors peak torque during eccentric actions, *ConcHams/ConcQuads* ratio of the peak torque of ConcHmas and ConcQuads, *EccHams/ConcQuads* ratio of the peak torque of EccHmas and ConcQuads, *MVIC Hams* maximal voluntary isometric contraction of the hamstrings muscles, *MVIC Quads* maximal voluntary isometric contraction of the extensors muscles

leg at impact was also slightly affected ($\Delta = -2.9\%$ and $-5\%$, ES $= -0.3$ to $-0.4$). There were also small to moderate alterations in joint, muscular and interactive moments for each phase in the middle ($\Delta = -29\%$ to 33%, ES $= -0.5$ to 0.8) and post-exercise protocol ($\Delta = -32\%$ to 43%, ES $= -0.8$ to 0.4).

### 3.6 Perceptual Responses

Perceptual responses (ten studies, 152 players, 77 ES; Table 6, Fig. 2) were investigated by subjective questions on DOMS (ten studies, 281 players, 34 ES), fatigue (three studies, 31 players, 13 ES), stress (three studies, 31 players,

13 ES), sleep (three studies, 31 players, ten ES) and recovery state (two studies, 23 players, 7ES). DOMS were recorded through the 7-point Likert scale [30, 94, 95, 121] and/or visual analogue scales (0–10 and/or 0–100) [66, 92, 93, 97, 98, 101]. The subjective assessment recorded general (four studies, 48 players, 17 ES) [94, 95, 121], lower-body muscle soreness (three studies, 57 players, 6 ES) [92, 93, 101] or was isolated to the quadriceps muscle group (three studies, 25 players, 11 ES) [66, 97, 98]. This analysis revealed large to very-large responses for DOMS at Post and G + 24 h. Moderate to large increases occurred until G + 72 h.

Players' well-being state in terms of fatigue, sleep and stress were investigated by a 7-point Hooper Likert scale [94, 95, 121] and by the total recovery scale questionnaire (TQR) during the 72-h observation period [95, 121]. All the parameters were small to moderately affected until G + 72 h.

### 3.7 Between-protocols Comparisons

#### *3.7.1 Biochemical Responses*

Post-match alterations in muscle damage, inflammatory and immunological parameters were of greater magnitude after 11 vs. 11 format compared to simulation protocols (Fig. 3). Large ($\Delta = 96\%$, 15 ES) and very large (315%, 5 ES) changes were observed in CK and myoglobin during the 11 vs. 11 condition. On the other hand, moderate and large CK (98%, 4 $ES_{LIST}$, 2 $ES_{SSP}$ and 2 $ES_{CST}$) and myoglobin ($\Delta = 3{,}048\%$, ES = 1.3, 3 $ES_{LIST}$ and 1 $ES_{CST}$) responses were observed for on-field simulations, respectively. These lower magnitudes of responses seemed also evident for treadmill protocols (64%, 1 ES).

Lower magnitude of IL-6 responses were observed after on-field protocols (3$ES_{LIST}$) than 11 vs. 11 condition (6 ES) with moderate (127%) and very large changes (292%) examined after each condition, respectively.

The 11 vs. 11 format resulted in greater magnitudes of changes in lymphocytes (− 13%, 3 ES), monocytes (96%, 1 ES) and neutrophils (167%, 3 ES) than the on-field condition (6%, 5 $ES_{LIST}$; 33%, 3 $ES_{LIST}$; 56%, 5 $ES_{LIST}$, respectively). On the other hand, a lower leukocytosis was evident after the 11 vs. 11 condition ($\Delta = 69\%$, ES = 1.8, 2 ES) than the on-field simulation (69%, 2 $ES_{LIST}$).

The limited number of ES plotted on the different redox parameters did not allow us to elucidate the effect of protocol on redox homeostasis.

#### *3.7.2 Neuromuscular Performance*

As depicted in Fig. 4, in general, muscle function impairments appeared to be independent of the protocols applied. Force production in concHams (− 9%, 2 ES) and $MVIC_{HAMS}$ (− 6.6%, 1 ES) were impaired to the same extent for 11 vs. 11, on-field [(concHams, − 9%, 4 $ES_{SAFT90}$ and 7 $ES_{LIST}$) and ($MVIC_{HAMS}$, − 24%, 1 $ES_{LIST}$)] and treadmill conditions [(concHams, − 11%, 13 ES) and ($MVIC_{HAMS}$, − 18%, 1 ES)]. The extent of the eccHams force impairments was similar between on-field (− 15%, 11 $ES_{LIST}$ and 8 $ES_{SAFT90}$) and treadmill protocols (− 17%, 11 ES).

Although, similar variations are observed, the standardized change of concQuads was of greater magnitude in 11 vs. 11 (− 7%, 2 ES) than in on-field simulation (− 8%, 10 $ES_{LIST}$ and 6 $ES_{SAFT90}$) and treadmill protocols (− 7%, 12 ES). There was a similar magnitude of changes between 11 vs. 11 (− 9%, five ES) and treadmill protocols (− 14%, 1 ES) for $MVIC_{QUADS}$.

Conventional and functional knee muscle joint balance indices were affected at greater magnitudes with treadmill compared to on-field protocols. While there was no clear change after on-field protocols (6 $ES_{LIST}$), the conventional ratio slightly decreased for treadmill (− 4.9%, 6 ES) protocols. Additionally, small and moderate changes were observed for the on-field (− 8%, 9 $ES_{LIST}$ and 3 $ES_{SAFT90}$) and treadmill (− 14%, 7 ES) protocols, respectively.

#### *3.7.3 Physical Performance*

The magnitude of impairments in 10-m (1 $ES_{11vs11}$ and 2 $ES_{SSP}$) and 15-m sprint (1 $ES_{11vs11}$ and 3 $ES_{LIST}$) performances was similar for on-field protocol (3.0% and 6.8%) and 11 vs. 11 match (0.7% and 2.5%) (Fig. 4). Additionally, the match-induced fatigue on CMJ was of greater magnitude with simulation protocols compared to 11 vs. 11 format. While there was no clear change after 11 vs. 11 format (− 2.3%, five ES), the magnitude of change in CMJ performance was moderate after on-field (− 7.5%, 4 $ES_{SSP}$, 2 $ES_{SAFT90}$ and 1 $ES_{CST}$) and treadmill protocols (− 8.1%, 1 ES). Nevertheless, there was not clear change in SJ performance for treadmill protocol (− 2.6%, 1 ES) and moderate decrease for the on-field (− 7.4%, 2 $ES_{SAFT90}$ and 1 $ES_{SGM}$) condition.

#### *3.7.4 Technical Performance*

While LSPT derivatives were not clearly affected by the 11 vs. 11 condition (2 ES), there were a small to moderate impairment in on-field protocols (6 $ES_{LIST}$, Fig. 3).

#### *3.7.5 Perceptual Responses*

The 11 vs. 11 format (40%, 2 ES) induced an increase in $DOMS_{General}$ of greater magnitude than compared to on-field simulations (25%, 2 $ES_{SAFT90}$). The magnitude of

$DOMS_{General}$ responses was similar between treadmill-based protocols (72%, 1 ES) and the 11 vs. 11 format. The exacerbated magnitude of change in perceptual responses induced by the 11 vs. 11 condition ($\Delta = 1715\%$, ES = 5.3, 2ES) compared to on-field protocols ($\Delta = 219\%$, ES = 1.1, 2 $ES_{SSP}$) was also observed for $DOMS_{Quads}$.

## 4 Discussion

Our systematic analysis demonstrates that actual match-play results in acute (Post) systemic alterations in metabolic, biochemical, physical performance, technical and perceptual markers. Physical performance (CMJ and hamstrings strength), muscle damage (CK) and perceptual measures (DOMS) are still substantially altered until at least G + 72 h (Tables 2, 3, 4, 5 and 6, Fig. 2). It appears that specific performance capabilities (e.g. sprint recovered at G + 72 h vs. jumping abilities still impaired at *G* + 72 h) likely present distinct recovery profiles (Table 5, Fig. 2b). Additionally, we observed that the 'real soccer match' (11 vs. 11 format) induces a greater acute magnitude (match-end) of both perceptual (DOMS) and physiological alterations (inflammatory, immunological and muscle damage) in reference to match simulations, while neuromuscular adjustments were similar (Figs. 3, 4).

### 4.1 Biochemical Milieu

Our analysis shows moderate to very large increases in several serum markers of muscle damage (CK, Myoglobin, LDH) within the first 24 h (ES = 0.6–2.3), peaking at G + 24 h and persisting until 72 h post-match for CK (ES = 0.4). The fact that soccer matches promotes exercise-induced muscle damage (EIMD) is reinforced by concomitant changes in indirect markers of EIMD such as the increase lipid peroxidation by products, decrease in force production capacity and increase in DOMS until G + 72 h (Tables 2 and 3).

The efflux of myocellular enzymes and proteins may reflect the degree of cellular and subcellular disturbances (e.g. membrane permeability) in the muscle [58, 135, 136] and is useful to determine the condition of muscle tissues (e.g. recovery of force capacity) [58, 136]. Although the precise stress mechanisms responsible for the initiation of muscle disruption are still unknown, these could be metabolic and mechanical in nature [137]. Moreover, this is a multifactorial process that consists of a complex cascade and inter-play of events involving increased oxidative stress, inflammatory and immune responses. All these events are triggered by the match, as observed in our systematic review [138]. In this regard, the energetic demands and locomotor activity pattern fit within the metabolic and mechanical stress models of EIMD [137, 139–143]. The concentric, isometric and eccentric muscles actions sustained during acceleration/deceleration, velocity (e.g. sprints) and technical actions (e.g. shooting, control of the ball against offensive pressure) [21–23, 144–146] result in the production of substantial impulses/forces [24, 122, 134, 147–150]. The mechanical stress model points out that the impulse produced during these muscle actions, with more relevance to the impulse produced during the eccentric action, leads to cellular and subcellular structural disturbances, as highlighted from biochemical responses [140, 143].

Our systematic observations of reduced blood and muscle pH (ES = − 1.2 and − 3.4), decreased glycogen store (ES = − 4.7) and increased plasma glycerol and FFA (ES = 12.0 and 1.9) reveal the high aerobic requirements throughout a game and extensive anaerobic demands during specific match periods (e.g. worst-case scenarios) [6]. We observe that already at match half (45 min within the game) there were alterations in all the metabolic pathways such as lipids (ES = up to 12.5), proteins (ES = 1.3) and carbohydrates (ES = up to 2.7). These metabolic alterations were further exacerbated during the second half of the game (Table 2). Particularly, although larger responses are observed in different markers of specific metabolic pathways (i.e. muscle and blood pH, glucose, glycerol, FFA, urea; Table 2), the very large decrement in glycogen stores has special relevance due to the soccer activity pattern and the role in sustaining high-intensity intermittent exercise performance [6]. These metabolic changes reflecting decreased energy availability may affect cellular regulation and thus contributing to fatigue development [6, 15, 17]. Interestingly, the loss of cell myofibres proteins into the blood may also be linked to match-induced substrate depletion and limited energy availability [138]. This high metabolic demand associated with playing soccer is further supported by our observations of very large post-match increases in IL-6 levels (Table 3) [151].

Match-play alters redox homeostasis and promotes oxidative damage. In this regard, our systematic review shows that from Post to G + 72 h, there are substantial increases in blood levels of certain oxidant bio-markers (e.g. accumulation of lipid peroxidation by products), changes in endogenous antioxidant molecules (total glutathione, glutathione, uric acid) and antioxidant enzymes (superoxide dismutase and glutathione peroxidase) levels (Table 3). Alterations in other recognized markers of redox state (oxidized glutathione, ratio reduced oxidize glutathione and homocysteine) further reinforce that match-play shifts the blood to a more oxidizing environment. These alterations are more prominent in the blood until G + 48 h and reflect the match-induced physiological stress. When the increase in reactive species production

overpowers the ability of antioxidant systems to render them inactive, the cellular loss of redox homeostasis occurs [152]. Alterations in redox homeostasis typically occur when there is a shift in the level of reactive species, oxidant biomarkers, antioxidants and redox/active molecules [153]. This latter occurrence is prone condition for oxidative damage to cellular lipids, proteins and DNA [152]. Moreover, apoptosis in muscle tissue subsequent to the match may also be triggered by increased oxidative stress [58, 154]. Although reactive species production has not been directly measured, the alteration of these redox markers reveals that the game constitutes a pro-oxidant insult (increased level of reactive species). Particularly, the match-related activity patterns involve series of events that enhance oxidative stress and damage and may favor additional pro-oxidant alterations such as: (i) locomotor-related actions with eccentric contractions (e.g. landing, breaking, kicking) inducing muscle damage and inflammation (e.g. increasing neutrophil oxidative burst), (ii) ischemia–reperfusion events (augmenting xanthine oxidase free-radical-generation system activity) associated with power-related actions (e.g. accelerations and isometric contractions to shield the ball), (iii) the excessive trauma (causing the disruption of iron-containing protein) that occurs during the impacts with the ground and opponents, and (iv) increased oxygen consumption during the game [28, 155]. As a result, we observed a small to large increase in circulating endogenous (e.g. total glutathione) and exogenous antioxidants more prevalent until G + 48H. These upregulation of the antioxidant defense occurs to strengthen the body reactive species scavenging capacity, blunting the overproduction of oxidants and upregulating the antioxidant system (Table 3) [104, 105].

We also observed very large match-related inflammatory and immunological responses at Post and still substantially persisting at G + 72 h. At match-end, our analyses reveal an elevation of CRP, IL-6 and TNF-α with peak values of IL-6 and TNF-α at this time point (Table 3). Interestingly, although responding with a lower magnitude, CRP shows substantial increases during the 48 h post-match with a peak at G + 24 h. Our systematic review indicates that a substantial inflammatory response can be triggered by an elevation of serum muscle damage markers (e.g. lipid peroxidation by products and CK) [156, 157]. This systemic immune response leads to cytokines secretion by inflammatory cells and the immune system activation [158, 159]. TNF-α, IL-6 and CRP are considered as blood biomarkers indicators of an inflammatory state [159, 160] and characterize the acute phase response. TNF-α is a proinflammatory cytokine, mainly a product of mononuclear phagocytes (macrophages) infiltrated along with other inflammatory cells into the injured muscle [161, 162] and affects the gene expression of acute phase proteins [162]. During exercise, IL-6 is primarily produced and released by skeletal muscle via a TNF-independent pathway [159]. This production is strongly influenced by the intensity and duration of the exercise [151] and is among the most potent mediators of the acute phase response [162]. Curiously, according to our analyses CRP peaked at G + 24 h while IL-6 and TNF-α peaked at match-end (Table 3). This is likely related to the hepatic origin of CRP that is mainly regulated by IL-6 and TNF-α [162, 163].

The immunological response is also evidenced by an increase in cell trafficking. A substantial increase in the circulation of leucocytes and specific monocytes, macrophages and lymphocytes populations occur at half-time (ES = 0.4–1.9) and with greater magnitude at match-end (ES = 1.1–2.0). The previous described inflammatory cytokines response aims to regulate a rapid migration of neutrophils, and later on of monocytes, into areas of injured muscle cells and other metabolically active tissues to initiate repair [164].

This increased circulation of leucocytes remains substantial at G + 24 h and G + 48 h. Interestingly, the observed likely increase in the neutrophil:lymphocytes ratio at match-end and throughout recovery reflect the match-load induced physiological stress [165]. The localized inflammatory response is consistent with the increased levels of both oxidative stress and DOMS during the G + 72 h and so may delay recovery. In fact, the accompanied accumulation of endogenous factors (e.g. cytokines) that are common with muscle damage may enhance the excitability of the sensory nerve fibers resulting in DOMS and so limiting players performance capacity by increasing perception of effort for a given task [166–168]. Moreover, the inflammatory response along with the repair process may initiate and amplify skeletal muscle injury [168–170], all these contributing factors delaying soccer match-related recovery.

Soccer match-play may also alter levels of circulating hormones; for instance, insulin levels are lowered during the game and at Post (ES = − 1.0). This substantial reduction in insulin concentration is linked to the metabolic changes triggered by match-play (e.g. increases of catecholamine production, blood glucose levels and lipolytic-related markers) [6]. Substantial increases in bound (total) and unbound (free) cortisol and testosterone levels likely occur as soon as the match ends. Nevertheless, conflicting reports on post-match testosterone on male subjects are evident in the observed substantial increases in the total [92] and decreases in the free (salivary testosterone) portion at this time-point, respectively [88]. Given that free testosterone can consist in ∼ 2% of the total hormone concentration, an increase in total testosterone should be reflected by an augmentation of the plasmatic free portion

as well [171]. However, special attention should be given to the free concentration of the steroid hormones in the blood (unbound portion), and thus, the active portion of the hormone carrying higher biological significance [107]. Generally, the studies analyzing hormonal responses as part of a biochemical analysis failed to specify the exact time (e.g. 0–30 min window) of the blood sample collection at match-end. This may be responsible for the diverging observed results (e.g. steroid hormones) since a higher variation can be observed in the short-term hormonal responses to exercise [172].

### 4.2 Perceptual Responses and Performance Recovery Profile

Soccer competition exacerbates perceptual responses with peak values observed at match-end (fatigue and stress) and after 24 h of recovery (DOMS) and substantially elevated values at G + 72 h. Notably, the perceptual measures (subjective) corroborate the peak magnitude of biochemical and neuromuscular measures within the first 24 h (objective), and substantial elevations still observed at G + 48/72 h (Tables 3, 4, 5 and 6).

A recent systematic review showed that, compared to objective measures, subjective ones may be more appropriate/sensitive to assess stress imposed by training and competition [173]. Accordingly, our systematic analysis reveals that subjective markers of competition stress are still substantially elevated at G + 72 h. Thus, the analogous responses between subjective (DOMS) and objective measures of well-being (e.g. CK, hamstrings strength) support, at least in part, the applicability of athlete self-reporting measures to monitoring fatigue level in soccer players. Subjective measures (e.g. DOMS) present the triple advantage of being easy to use, cost effective and sensitive.

Soccer match results in substantial muscle function impairments until G + 72 h. This impairment is already of small to moderate magnitude at half-time for strength-related capabilities (RFD and peak-torque, Table 3). Our systematic review reveals that match-induced fatigue moderately alters the torque–angle profile (e.g. eccentric hamstrings peak torque occurring at shorter muscle lengths) and rapid force development capabilities are largely affected at match-end. Moderate reductions in maximal force capacity are still evident at G + 72 h. This is accompanied by small decreases in knee joint stability until G + 48 h (conventional and functional muscle ratios). Particularly, magnitude of alterations is larger and last longer for knee flexors; impairments in eccentric force production are above 1.5-fold and twofold the ones observed in quadriceps and hamstrings concentric muscle actions. Biomechanical analysis of acceleration/deceleration motions (e.g. turning, changing pace and changes in direction) point to a high eccentric involvement of the hamstrings muscles during these intense movements [174–176]. Substantial eccentric impulses are produced during rapid transitions from eccentric to concentric actions as well when the knee flexors rapidly break hip flexion and/or knee extension (e.g. kicking actions) [177]. For example, specific phases of the sprint run (i.e. terminal swing phase) involve a combination of hip flexion and knee extension tasks that induce a substantial elongation stress on the biarticular hamstrings [177]. These biomechanical characteristics likely explain the higher muscle damage and higher injury risk during sprint actions [177]. Consequently, hamstrings muscles may suffer more severe ultrastructural disturbances as a result of the repeated eccentric actions performed during the game. In fact, a greater incidence and magnitude of DOMS is reported in this muscle group compared to quadriceps and plantar flexors after a soccer-specific simulation protocol [178]. Consequently, as supported by our results longer analysis periods would be needed to determine whether players fully recovered their hamstring muscle strength qualities (e.g. several days).

Our systematic analysis also points out that sprinting, jumping and COD abilities are moderately impaired at match-end. Although running abilities recover at G + 72 h, jumping performances are still slightly impaired at this time point. The small impairment in technical performance capacities at match-end is still evident at G + 48 h. Impaired performance and exacerbated perceptual responses may array from the interplay between peripheral processes and central nervous system (modulated by afferent feedback) [179–182]. The match-induced peripheral and central fatigue are evident by alterations in muscle contractile properties (e.g. peak twitch torque) and decreases in central motor output (e.g. neural input reaching the neuromuscular junction), respectively (Sect. 3.4.1) [93, 117, 119, 183]. The wide spectrum of metabolic adjustments (e.g. decrease pH, glycogen depletion), structural changes (e.g. changes in membrane permeability and sarcomere integrity) and alterations in the biochemical milieu (e.g. increases in inflammatory markers, specific metabolites, endocrine changes) examined during our systematic analysis of the literature (Table 2), justifies that decrease in muscle function may be caused by the interplay between peripheral and central processes [168, 179–181]. Notably, central fatigue-related markers exhibit a faster recovery (motor output-related capacity are restored at G + 48 h) than peripheral markers (CK still substantially elevated at G + 72 h). Such distinct restoration of neuromuscular function integrity suggests that the magnitude of post-match neuromuscular fatigue and performance recovery within the first 48 h could be explained

by central and peripheral limiting factors. A faster recovery of the neuromuscular performance (hamstrings muscle function and physical performance still impaired at G + 72 h) would be primarily dependent on the restoration of the peripheral recovery processes.

Physical performance impairments are already evident at half-time and are substantially aggravated during the second half. Our results suggest a faster recovery profile for COD and straight-line sprint performance than for jump abilities; it seems that 48 h are enough for a player to recover for both linear and non-linear sprint performance. Nevertheless, longer distances may require a longer recovery period as characterized by a greater magnitude of ES (Table 5). This is further corroborated by substantial reductions in mean power output and peak speed during a 6-s sprint (Table 5) [95, 121].

Single (CMJ) and repeated CMJ ($CMJ_{15s}$) performances are impaired at match-end. These impairments in stretch–shortening cycle jumping actions reflect an inability to effectively transit from the eccentric to the concentric component (e.g. reactive strength index; Table 5). Additionally, match play impairs jump-based SSC abilities to a greater magnitude at each time-point (Table 5). The needed recovery to perform non-SSC (SJ) and SSC jumps (CMJ) is likely more residual and still substantially reduced at G + 72 h. Notably, it has been argued that jumping variables, and more particularly with CMJ, show high repeatability with immediate and prolonged fatigue-induced changes [184] and then being more suitable for neuromuscular fatigue monitoring [184, 185]. This differentiated recovery kinetics between sprint and jump tasks may be related to changes in biomechanical strategies (e.g. eccentric and concentric phases durations) as result of match-induced neuromuscular fatigue and in turn affecting jumping performance to a greater extent [184, 186]. The substantially longer recovery period for muscle contractile properties (during eccentric and concentric actions) and jumping abilities compared to sprint and COD performances may highlight the higher recovery strength-dependent nature of the jumps motor task [13, 184]. Remarkably, the observed impairments in lower body muscle function during single-joint muscle actions (peak torque and RFD during knee extension) have not been found during multi-joint actions. Force and power-related CMJ performance variables were not affected at Post and until G + 72 h [92, 117]. Nevertheless, this is not universally confirmed [91]. RSA and high-intensity intermittent endurance exercise performance were more scarcely assessed, presumably due to the high fatigability imposed by the testing procedure. RSA and YYIE2 performances are substantially impaired at Post. Given the determinants of RSA [187, 188] and Yo–Yo test [189–191] performances, we may infer that these decreases may result from the abovementioned match-induced mechanical (e.g. force production) and metabolic (e.g. glycogen stores) impairments. The faster recovery time-course of RSA (recovered at G + 48 h) compared to YYIR1 (still impaired at G + 72 h) may be related to the recovery kinetic of different mechanical and metabolic capacities. As an example, the post-match glycogen recovery pattern after soccer match-play is muscle fiber [99] and subcellular location specific [192], which may distinctly affect RSA and YYIR1 performances recovery profile. Moreover, as we observed, the extent of muscle damage may vary within the lower-limb muscles (e.g. knee flexors vs. knee extensors) [178] and may, in turn, contribute to distinct intra-muscular glycogen recovery profile [193]. This may result in different lower-limb muscle function recovery profiles and consequently impact differently the performance recovery profile of independent physical variables (sprint vs. jump and RSA vs. YYIR1) systematically reviewed in this study. Another potential explanation could be the task specificity of the RSA protocol implemented (e.g. different work:rest ratio) [194].

### 4.3 Match and Injury Risk

A soccer match results in increases in injury-related markers towards match end and until G + 72 h. Injury occurrence can affect player's development and squad performance throughout the year [195]. Several studies reveal that between 65% and 91% of the players likely sustain an injury during a season [196] and that 90% of all muscle injuries of professional soccer players are localized in lower limbs [197]. Generally, the injury rate is substantially greater during competition than in training [198]. Notably, training-related hamstring injury rates have increased substantially since 2001 without alterations in match-related injury rates [199]. This may result from the increase match demands leading to extended periods of residual fatigue and/or players experiencing increased loads during training while still recovering/regenerating. In fact, particularities of the match-induced neuro-mechanical alterations that can be causal factors of soccer match-related injuries are (i) the greater and extended impairment in hamstrings muscle force production (threefold greater in eccHams than concQuads at post-match and until G + 72 h, Table 4), (ii) greater magnitude of decreases in RFD in hamstrings compared to quadriceps muscles, and more particularly during the early phase of force production (Table 4), (iii) the greater magnitude of disturbances in the dynamic relation around knee joint impacting its stability (i.e. fivefold greater in functional than in conventional ratio; Table 4), (iv) greater magnitude of changes in angle specific strength for eccHams compared to concQuads (Table 4), (v) the dissimilar recovery patterns of RFD

between ankle and knee joint muscles (Table 4 and Sect. 3.4.1.4) and (vi) the kinematic and kinetic alterations occurring during different motor performance (e.g. postural stability, kicking, COD, sprinting and jumping actions). In fact, soccer-related injuries likely occur under rapid movement perturbations or actions requiring rapid force development and are more prevalent in hamstring muscles group, specifically in BF [196, 200]. Moreover, singularities of neuro-physiological alterations such as muscle activity reduction are likely more evident for specific hamstrings group muscles (e.g. BF) [117, 119]. Additionally, the distinct recovery kinetic in muscle voluntary activation of lower limb muscles (e.g. plantar flexors vs. quadriceps) are all realistic contributors for a deficient motor control and decreased movement quality under explosive/unpredictable actions. Notably, although majority of the sprints performed during the game are short in nature (< 10 m) [201], players may need to perform long duration sprints during the game (> 30–40 m). The observed trend for a greater impairment in extended sprint distances, may not only limit performance during this type of game actions but may also expose players to a greater injury risk if this context occurs towards match-end and/or within congested periods [197, 199]. Observations of decreased locomotor efficiency (accelerometer derived player load) at this time-points likely supports this statement; suggesting an increase in the loading required for each meter covered [202]. All the aforementioned factors may explain the specificity of increased injury risk during (e.g. towards the later stages) and within short post-match recovery periods (G + 72 h).

### 4.4 Protocols

A second aim was to determine if post-match-responses to soccer play differ between actual competition (11 vs. 11) and simulation protocols. In general, on-field and treadmill-based simulation protocols resulted in rather similar muscle function and performance impairments. Nevertheless, the 11 vs. 11 match induced greater magnitude of change in muscle damage (e.g., CK, ES = 1.8 vs. 0.7), inflammatory (IL-6, ES = 2.6 vs. 1.1), immunological markers and DOMS (ES = 1.5 vs. 0.7) than simulation protocols at Post. These soccer match simulation protocols have been validated to replicate the internal- (e.g. heart rate, blood [La], core temperature) and external load (e.g. running distance at different speed zones and velocity profile) recorded during match play [41, 43–51]. Recent technologies (e.g. GPS) [203] allow characterization of the intermittent profile of a soccer match and subsequently match simulations under field and laboratory conditions. In this regard, simulation protocols may induce an equivalent accelerometer derived player load [53, 204], thus resulting in comparable lower limb muscle function and performance impairments. Nevertheless, our systematic observations reveal that they do not accurately recreate the biochemical strain (e.g. CK) and uncomfortable muscular perception that is associated with the 11 vs. 11 match format.

During the 11 vs. 11, players are expected to perform a greater number of high-velocity soccer-related tasks (e.g. kicking and jumping) and common unorthodox movements (e.g. sideways running) than during the on-field (e.g. LIST) and treadmill conditions. These actions requiring high eccentric impulses may result in a greater mechanical strain that most likely causes greater damage resulting in a more severe EIMD [135]. This considerable amount of lengthening based muscle actions is corroborated by greater magnitudes of DOMS, muscle damage and inflammatory and immunological responses after the 11 vs. 11 compared to on-field protocol (i.e. match-end). Interestingly, the 11 vs. 11 seemed to induce distinct immunological responses with a lymphopenia and greater magnitudes of neutrophilia and monocytosis observed. At match-end, our results reflect that the increased eccentric muscular demands during the 11 vs. 11 protocol are linked to a greater release of immune system modulators (proinflammatory cytokines and acute-phase proteins), increased ROS production by mitochondria ($H_2O_2$ production) and phagocytic cells recruitment with the potential to release ROS [205, 206]. These signals may favor additional oxidative stress and damage, as well as apoptosis in several tissues and cells (e.g. including lymphocytes) [28, 58, 154]. Additionally, the likely increase in post-match neutrophil:lymphocytes ratio may reflect the greater match-load induced physiological stress of the 11 vs. 11 format [165]. Altogether this indicates that competitive conditions (11 vs. 11) induce a greater acute stress state [207, 208]. That said, we cannot exclude the role of psychosocial factors (competition stress) in the etiology of the specific physiological changes observed here such as immunological responses [209].

Curiously, while LSPT derivatives were not clearly affected by the 11 vs. 11 condition, there was small to moderate impairments in on-field protocols (LIST). It is possible to assume that competitive matches involve higher cognitive demands than on-field protocol (LIST). It is possible that the 11 vs. 11 condition may result in higher levels of mental fatigue [210] likely linked with higher perceptual responses (perception of effort) and deterioration of technical qualities [211, 212].

## 5 Limitations

It is important to highlight some limitations inherent to this work. In this systematic review, we aggregated all the on-field protocols while the external load may differ (locomotor activities) between studies. Secondly, some studies

do not precisely report the time point of blood collection at match-end (e.g. 10, 20, 30 min Post) that may have influenced the observed concentration/activity of the analyzed marker. This limitation is also present in the different post-match measurements that were adjusted to a 24-h period, for example, a G + 18 h measurement was included as a G + 24-h time-point in our analysis. Finally, the different protocols used to characterize the games have been performed on different surfaces (grass, artificial turf vs. synthetic), under different environmental conditions, and with players from different training status and gender. Nevertheless, future systematic analysis are necessary to clarify the role played by gender, surface and/or environment conditions in modulating the physiological responses to match-play or soccer-specific endurance exercise [27, 101, 121, 123, 135, 213].

## 6 Conclusion

This paper provides a systematic review of the literature that has reported the time-course of match-related fatigue and recovery in soccer. The current findings show that soccer match alters the levels of muscle-injury markers, inflammation and immunological cell tracking, impairs physical performance and exacerbates perceptual responses until at least 72 h post-match. Specific performance capabilities (e.g. sprint vs. jumping abilities) likely present distinct recovery profiles, resulting in player physical performance impairments at 72 h post-match. Coaches must alter the structure (e.g. recovery times) and contents of the training sessions to manage efficiently the training load within this time-frame. Our results support the International Olympic Committee consensus that soccer matches should be interspersed by at least 96 h to protect players from injury [214, 215]. However, to date, this recommendation has still not been taken in consideration by some sports governing bodies [216].

Additionally, the 11 vs. 11 game may elicit greater magnitude of load than simulated protocols at specific biochemical (e.g. CK) and perceptual levels (e.g. DOMS). Nevertheless, given that on-field and treadmill-based simulation protocols resulted in similar muscle function and performance impairments, such protocols could particularly be useful to assist players in the return to play process. This would allow injured players to experience a progressive 'real' match physical strain. Another advantage is that it permits assessing the effectiveness of specific interventions (e.g. eccentric training) on these outcomes measured here in a controlled environment. Nevertheless, medical staff and researches should consider the distinct biochemical and perceptual responses when evaluating players readiness to return to 'real competition' or assessing the effectiveness of specific interventions (e.g. eccentric training) on these biochemical and perceptual-related endpoints.

## 7 Practical Applications

Our systematic analysis suggests that the extent of the recovery period cannot consist in a 'one size fits all approach' and that an inadequate exposure (high training load) during this recovery window may be harmful. Medical and technical staffs are called to implement methods that may optimize player's physiological and psychological states. Particularly, match-related monitoring techniques may allow predicting the extent of residual fatigue (e.g. acute match activity in the game vs. chronic match exposure). The extended recovery period for CMJ and hamstrings strength, CK and DOMS would suggest that these parameters should be included in a monitoring test battery. In this regard, hand-held dynamometers (cost-effective) and/or portable force plates that are reliable and sensitive [217, 218] would be easy to use devices. However, the observed changes in testing measures should be interpreted as meaningful ones (e.g. smallest worthwhile change) [65] and, to date, the use of individual reference ranges (e.g. CK) seems to constitute the better strategy for monitoring muscle recovery [219]. The similar impairments in hamstrings eccentric and isometric strength during the 48-h period further reinforce the use of MVIC to assess the recovery of hamstrings contractile properties. The more severe DOMS that is associated to eccentric testing further reinforces of the use of MVIC to assess the recovery of hamstrings contractile properties may be preferred.

Additionally, other strategies such as nutritional intake are considered as key factors to improve player's performance and recovery should be considered, and may contribute to preventing the undesirable physiological effects of match-induced peripheral (e.g. glycogen depletion) and central fatigue (e.g. branched-chain amino acids) [220–224]. Meta-analytic investigations suggest that specific recovery strategies such as massage, cold water immersion and wearing compression garments may assist in decrease the match-induced physiological and/or psychological alterations [225–227]. Finally, training status optimization by including, among others, strength training (e.g. increase maximal eccentric force and endurance capabilities of hamstrings muscle groups, prevent hamstrings shift towards shorter length and the repeat-bout-effect) and manipulation of the timing effect of neuromuscular training (e.g. under fatigue) may be key to optimizing recovery of muscle function and reducing the injury risk [13, 14, 177, 228–231]. In fact, recovery of muscle

function should remain the primary target of an intervention aiming performance and decreasing injury [183].

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