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Repeated-Sprint Ability – Part I
Factors Contributing to Fatigue

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Abstract

Short-duration sprints (<10 seconds), interspersed with brief recoveries (<60 seconds), are common during most team and racket sports. Therefore, the ability to recover and to reproduce performance in subsequent sprints is probably an important fitness requirement of athletes engaged in these disciplines, and has been termed repeated-sprint ability (RSA). This review (Part I) examines how fatigue manifests during repeated-sprint exercise (RSE), and discusses the potential underpinning muscular and neural mechanisms. A subsequent companion review to this article will explain a better understanding of the training interventions that could eventually improve RSA.

Using laboratory and field-based protocols, performance analyses have consistently shown that fatigue during RSE typically manifests as a decline in maximal/mean sprint speed (i.e. running) or a decrease in peak power or total work (i.e. cycling) over sprint repetitions. A consistent result among these studies is that performance decrements (i.e. fatigue) during successive bouts are inversely correlated to initial sprint performance. To date, there is no doubt that the details of the task (e.g. changes in the nature of the work/recovery bouts) alter the time course/magnitude of fatigue development during RSE (i.e. task dependency) and potentially the contribution of the underlying mechanisms.

At the muscle level, limitations in energy supply, which include energy available from phosphocreatine hydrolysis, anaerobic glycolysis and oxidative metabolism, and the intramuscular accumulation of metabolic by-products, such as hydrogen ions, emerge as key factors responsible for fatigue. Although not as extensively studied, the use of surface electromyography techniques has revealed that failure to fully activate the contracting musculature and/or changes in inter-muscle recruitment strategies (i.e. neural factors) are also associated with fatigue outcomes. Pending confirmatory research, other factors such as stiffness regulation, hypoglycaemia, muscle damage and hostile environments (e.g. heat, hypoxia) are also likely to compromise fatigue resistance during repeated-sprint protocols.

1. Introduction

1.1 Defining Repeated-Sprint Ability (RSA)

Team and racket sports are popular with millions of participants worldwide. Athletes engaged in these disciplines are required to repeatedly produce maximal or near maximal efforts (i.e. sprints), interspersed with brief recovery intervals (consisting of complete rest or low- to moderate-intensity activity), over an extended period of time (1–4 hours), and this has been termed repeated-sprint ability (RSA).[1-8] Time-motion analysis in team sports has shown that sprinting generally constitutes 1–10% of the total distance covered (1–3% of effective playing time).[9-12]

There is potential for confusion, however, as some authors have used the word 'sprint' to describe exercise lasting 30 seconds or more.[13-15] For the purposes of this review, the definition of 'sprint' activity will be limited to brief exercise, in general ≤10 seconds, where maximal workout (i.e. power/speed) can be nearly maintained until the end of the exercise (figure 1). Longer duration, maximal-intensity exercise, where there is a considerable decrease in performance will be referred to as 'all-out' exercise, but is not the topic of this review.

When sprints are repeated, it is also useful to define two different types of exercise, i.e. intermittent-sprint and repeated-sprint exercise (RSE). Intermittent-sprint exercise can be characterized by short-duration sprints (≤10 seconds), interspersed with recovery periods long enough (60–300 seconds) to allow near complete recovery of sprint performance.[16,17] In comparison, RSE is characterized by short-duration sprints (≤10 seconds) interspersed with brief recovery periods (usually ≤60 seconds). The main difference is that during intermittent-sprint exercise there is little or no performance decrement,[18,19] whereas during RSE there is a marked performance decrement (figure 2).[20] Such a distinction is important as the factors contributing to fatigue are likely to be different for these two types of exercise.

1.2 Fatigue During Repeated-Sprint Exercise

For the purpose of this review, fatigue is defined as a RSE-induced reduction in the maximal power output (i.e. during cycling exercise) or speed (i.e. during running exercise), even though the task can

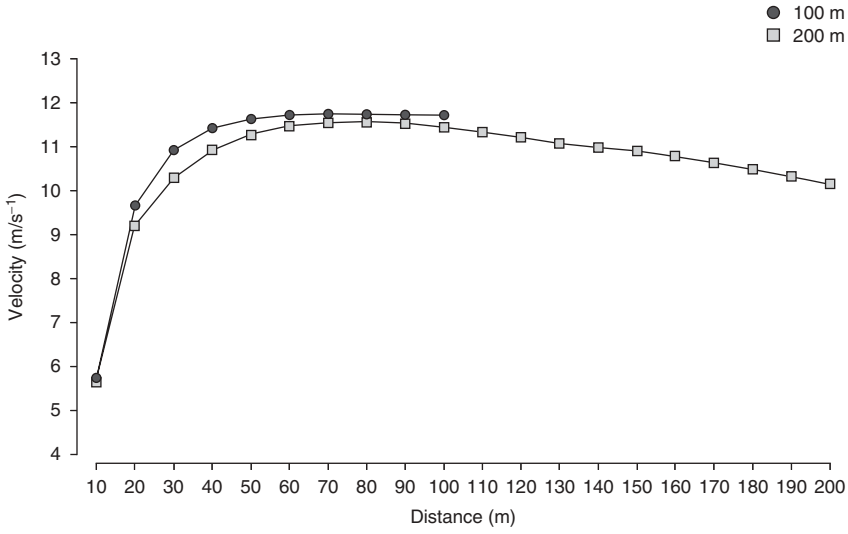

Fig. 1. Sprint profiles of 100 m and 200 m world records for men. In the above example, the 100 m performance would be defined as a sprint exercise, while the 200 m performance would be defined as an 'all-out' exercise.

be sustained. Fatigue during RSE typically develops rapidly after the first sprint (figure 2).[21] It is now accepted that fatigue can be caused by a variety of factors, ranging from the generation of an inadequate motor command in the motor cortex (i.e. neural factors) to the accumulation of metabolites within muscle fibres (i.e. muscular factors), and that there is no one global mechanism responsible for all manifestations of fatigue. The complex nature of fatigue is also highlighted by the diversity of approaches, models or indices (see section 2.1) that have been used to account for the decline in muscular performance.

In recent years, there has been an exponential growth of interest in factors underlying fatigue during RSE (figure 3). This is probably due to technological advances, the study of new potential limiting factors and the inclusion of diverse RSE protocols. However, there is still no clear explanation for the mechanisms that limit RSA.[5]

1.3 Relevance of RSA

Although performance in most multiple-sprint sports is dominated by technical and tactical proficiencies,[30] and the importance of RSA as a crucial physical component of team-sport performance[31] has been recently questioned,[12] fatigue development in team sports (e.g. soccer) has been linked with the ability to reproduce sprints.[32] In this regard, significant reductions in sprinting and high-speed running actions have been observed toward the end of elite soccer matches in men[33] and women.[34] Due to the unpredictable nature of the

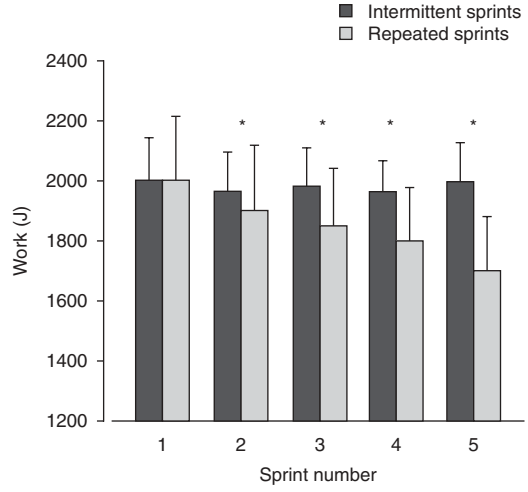

Fig. 2. Graph showing the effects of rest duration on maximal 4 sec, cycle sprint performance. Intermittent sprints were performed every 2 min,[19] whereas repeated sprints were executed every 30 sec.[20] * Significantly different from sprint 1 in the repeated-sprint condition.

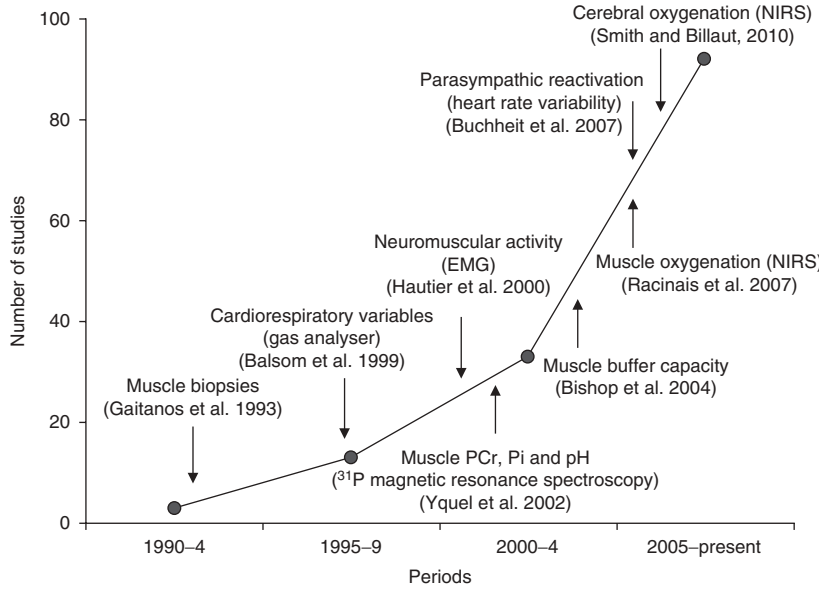

Fig. 3. Published research on the possible physiological mechanisms responsible for fatigue during repeated-sprint exercise (Gaitanos et al.,[22] Balsom et al.,[23] Hautier et al.,[24] Yquel et al.,[25] Bishop et al.,[26] Racinais et al.,[27] Buchheit et al.[28] and Smith and Billaut[29]). Time-points when new analysis methods were introduced into the investigation of repeated-sprint ability are also shown. **EMG** = electromyogram; **NIRS** = near-infrared spectroscopy; **PCr** = phosphocreatine; **Pi** = inorganic phosphate.

game, it is thought that intense periods of sprinting activity may on occasion determine the final outcome of a game, by influencing the ability to win possession of the ball or to concede goals.[35] For example, a ~0.8% impairment in sprint speed would have a substantial effect on the likelihood of a player losing possession of the ball against an opponent, when both players sprint for the ball.[36] Moreover, repeated-sprint tests performed before and after elite soccer games have demonstrated that RSA deteriorates substantially with fatigue development.[37,38] Thus, considering that the aetiology of fatigue is dependent on the exercise mode (the so-called 'task dependency'),[39] a better understanding of the factors contributing to fatigue during RSA is arguably the first step in order to design interventions (i.e. training programmes, ergogenic aids) that could delay the onset of fatigue, enhance RSA and eventually improve physical match performance in team-sport athletes.

The aim of this review, therefore, is to discuss mechanisms that have been proposed to contribute to fatigue during RSE. To achieve this objective, the databases SportDiscus®, PubMed, Web of Science and MEDLINE were searched, without any time restriction, using the following combination of terms: 'fatigue', 'repeated-sprint exercise', 'repeated-sprint ability' and 'multiple sprint'. The reference lists of the articles obtained were searched manually to obtain further studies not identified electronically. The insights gained from this review should assist the prescription of training strategies to more effectively combat factors responsible for fatigue during repeated sprints (as discussed in a subsequent companion review to this article[40]).

2. Manifestation of Fatigue

2.1 Indices

During RSE, fatigue manifests as a decline in maximal sprint speed (i.e. running), or a decrease in peak power or total work (i.e. cycling), over sprint repetitions (figure 4). To quantify the ability to resist fatigue during RSE, researchers have tended to use one of two terms, the fatigue index (FI) or the percentage decrement score (S_{dec}). The FI has generally been calculated as the drop-off in performance

from the best to worst sprint performance during an RSE (equation 1).

$$FI = 100 \times \frac{(S_{best} - S_{worst})}{S_{best}} \quad \text{(Eq. 1)}$$

where S refers to sprint performance and can be calculated for either speed, work or power scores.

In comparison, the S_{dec} attempts to quantify fatigue by comparing actual performance to an imagined 'ideal performance' (i.e. where the best effort would be replicated in each sprint) as shown in equation 2.[7,42]

$$S_{dec}(\%) = \left\{1 - \frac{(S_1 + S_2 + S_3 + \ \ldots \ + S_{final})}{S_{best} \times \text{number of sprints}}\right\} \times 100 \quad \text{(Eq. 2)}$$

A slight modification of the formula is required for sprint running performance (as times will increase as subjects fatigue) as shown in equation 3.

$$S_{dec}(\%) = \left\{\frac{(S_1 + S_2 + S_3 + \ \ldots \ + S_{final})}{S_{best} \times \text{number of sprints}} - 1\right\} \times 100 \quad \text{(Eq. 3)}$$

A possible advantage of the S_{dec} is that it takes into consideration all sprints, whereas the FI will be influenced more by a particularly good or bad first or last sprint. By comparing eight different approaches, Glaister et al.[43] concluded that the S_{dec} calculation was the most valid and reliable method to quantify fatigue in tests of RSA.

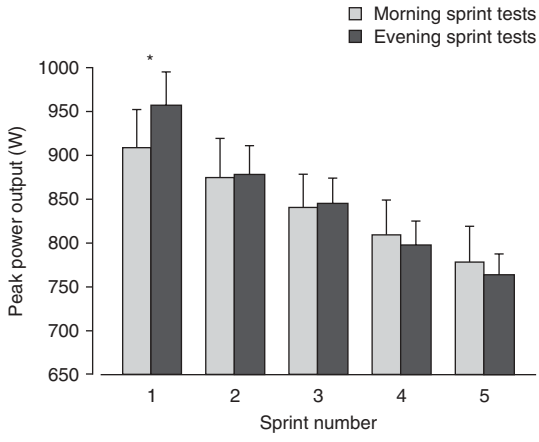

Fig. 4. Peak power output during a 5×6 sec repeated-sprint cycling test performed in the morning and evening. Note that in the evening, peak power was higher during the first sprint, but not different in the latter sprints, which produced a higher calculated sprint decrement (indicated by *) [modified from Racinais et al.[41]].

While this review only deals with fatigue outcomes, it is important to note that other performance indices, such as total mechanical work/sprint time (i.e. sum of power outputs/times for all sprint repetitions) should be used in conjunction with indices of relative decrement in performance (i.e. fatigue) to asses repeated-sprint performance.[44] In fact, it is absolutely necessary to contextualize the calculated fatigue indices when RSA is evaluated because less/greater fatigue does not always equate to better/worse performance.[45,46] An example of this is illustrated in figure 4. Here, a significant increase in performance during sprint 1 will lead to a greater calculated FI. It would be incorrect to interpret this as a decrease in RSA, when there is also an increase in total/mean sprint performance.

2.2 Influence of Initial Sprint Performance

An important determinant of fatigue during RSE is the initial (i.e. first sprint) mechanical score, which has consistently been reported to be positively correlated with performance decrement over subsequent sprints.[21,47-49] This can probably be attributed to the observation that subjects with a greater initial sprint performance will have greater changes in muscle metabolites, arising secondary to a higher anaerobic contribution, which in turn has been related to larger performance decrements.[22] In support of this, Mendez-Villanueva et al.[21] have reported that individuals with lower anaerobic power reserves, implying less reliance on anaerobic metabolism, showed a higher fatigue resistance during repeated cycling sprints. This suggests that metabolic pathways supporting force production, and not the absolute force generated *per se*, might explain power decrements during RSE. Therefore, initial sprint mechanical output *per se* cannot solely account for performance decrements during RSE. Indeed, Mendez-Villanueva et al.[50] have highlighted that previous fatiguing muscle contractions (i.e. a set of repeated sprints) exacerbated the rate of fatigue development during subsequent sprints, despite being matched for initial sprint power. Similarly, Bishop and Edge[51] found a greater fatigability

(i.e. larger work decrement) across five 6-second cycling sprints repeated every 30 seconds in low versus moderately aerobically trained females matched for single-sprint performance.

2.3 Task Dependency

There is no doubt that the degree of fatigue experienced during RSE is largely influenced by the details of the task being performed (i.e. task dependency).[39] For example, fatigue resistance during RSE is directly dependent on the exercise mode (e.g. cycling vs running);[8] decrement scores during repeated-sprint cycling protocols (10–25%) have typically been reported to be greater than those for running protocols (5–15%). Fatigue development during RSE also appears to depend on resistive load (e.g. mechanically, wind or electromagnetically resisted[52,53]) or the running surface (e.g. indoor tartan track, outdoor football field). Moreover, fatigue resistance during RSE depends on the distribution (e.g. number of repetitions) and duration of the work periods,[54] and the recovery pattern; i.e. the nature,[55-58] duration[16,59-64] and intensity[65] of the recovery between sprints. Although not a universal finding,[66] performing active versus passive recovery is generally associated with a higher degree of fatigue development.[55,58,65,67] Compared with a passive recovery, low- and moderate-intensity active recoveries (~20% and 35% maximal oxygen uptake [$\dot{V}O_{2max}$], respectively) have similar effects on RSA and muscle metabolism.[65] One limitation of the above-mentioned studies, however, is that researchers have typically imposed fixed-duration recovery periods between repeated sprints, while the pattern of exercise and recovery seen in team sports can best be described as random (i.e. imposed by tactical factors and the player's ability to self-select the intensity and nature of their efforts). By manipulating different recovery durations (i.e. constant, increasing and decreasing recovery pattern) Billaut and Basset[59] have reported that reductions in peak and mean power output were delayed with the decreasing recovery pattern, but were subsequently more marked and associated with greater neuromuscular perturbations.

The fatigue experienced during RSE also appears to be influenced by priory activity. For example, when static stretching is conducted after dynamic activities during the warm-up, and immediately prior to performance, Sim et al.[68] have shown that the ability to resist fatigue during RSE is compromised. However, these results have been challenged since performing static stretching within the warm-up for 3 consecutive days did not negatively affect subsequent RSA.[69] It has also been reported that preceding high-intensity exercise may compromise RSA.[32,50] Hence, previous fatiguing RSE, followed by a rest period, exacerbates the rate of loss of muscle power output during a subsequent repeated-sprint bout.[50]

Despite the potential influence of prior activity on RSA, very few studies have employed RSA tests that mimic the actual game situation. More research is required using repeated-sprint tests that assess RSA following and prior to activities specific to a given sporting discipline, as Jougla et al.[67] did for rugby union players, while also simulating work-to-rest ratios specific to such activities in competition. In this context, Krustrup et al.[32] have reported that repeated 30 m sprint performance is impaired following a competitive football (soccer) game in Danish Premier League women players. In contrast, no differences were found in the performance decrement between repeated-sprint tests performed by young basketball players at different stages of a game.[70] A limited number of studies have investigated RSA during team-sport competition.[9,10,12] When elite field hockey players played three games within 4 days, there were significant changes in time-motion analysis, as the frequency of exercise bouts that met the criteria for 'repeated sprints' decreased across the three games.[10]

With respect to task specificity, it is important to note that most of our knowledge relative to the development of fatigue during RSE has been gained from cycle-based, repeated sprinting performed in the laboratory environment. The strength of this approach is to accurately control and manipulate most of the influencing variables (e.g. environmental conditions). However, the applicability of findings arising from laboratory settings has been questioned.[71] Further research is therefore required to assess RSA using more sport-specific tests, while still ensuring a high level of standardization and reliability of measures.

2.4 Influence of Other Factors

Other factors, such as sex,[72,73] age,[63,64,74,75] playing position (soccer[76]), training status (activity performed, level of competitiveness)[76,77] and whether or not subjects are sickle cell trait carriers,[78] have also been reported to influence RSA. In general, being female, young or aerobically trained has typically been associated with a smaller decrement score. However, further research is required to establish whether these differences can be attributed to differences in fatigability or can largely be explained by differences in initial mechanical output (see section 2.3).

The effect of the time of day on RSA has also been studied.[41,46,79] Muscle power during the first sprint was improved in the afternoon, compared with the morning, leading to a sharper decrease in performance, with no significant difference for total work (figure 4).[41] While a greater decrement score in the afternoon could be interpreted as impaired RSA, mechanical power during the final sprints was not significantly different between the morning and afternoon conditions. Thus, the higher decrement score in the afternoon is simply the consequence of the greater initial mechanical output, and, as previously mentioned (see section 2.1), a greater sprint decrement score cannot always be interpreted as a poorer RSA. This was confirmed by a subsequent study showing no difference in sprint decrement in the afternoon when initial sprint performance was matched to that performed in the morning.[46] This further highlights the need to carefully interpret changes in the decrement score if there are concomitant changes in initial sprint performance.

3. Limiting Factors

3.1 Muscular Factors

3.1.1 Muscle Excitability

At the skeletal muscle level, marked ionic disturbances, arising secondary to decreases in sodium (Na^+)/potassium (K^+)-adenosine triphosphatase (ATPase) activity, have been observed following intense dynamic contractions.[80,81] In such cases, the Na^+/K^+ pump cannot readily re-accumulate the K^+ efflux out of the muscles cells, inducing at least a doubling of muscle extra-cellular K^+ concentration ($[K^+]$).[82] These modifications will impair cell membrane excitability and depress force development, probably by slow inactivation of Na^+ channels,[83] and will be manifested indirectly by a reduction in action potential amplitude and a slowing of impulse conduction.[84] Since most of our knowledge to date has been gained from *in vitro* studies, it is still unclear whether these ionic disturbances contribute to fatigue during RSE. Unpublished observations have shown that plasma $[K^+]$, when corrected for changes in plasma volume, does not change following 5×6 second sprints (30 seconds of recovery). However, further research is required since (i) interstitial $[K^+]$ is considerably higher than venous plasma $[K^+]$ at similar work intensities; and (ii) venous $[K^+]$ values may not reflect the concentration in the interstitium (i.e. the site where K^+ may have its effects).[82]

By applying an electrical stimulus to peripheral nerves, the study of the *in vivo* muscle compound action potential (M-wave) characteristics has been used to determine whether exercise-induced fatigue alters muscle excitability. Decreased M-wave amplitude, but not duration, was reported after a repeated-sprint running protocol (12×40 m, 30 seconds of recovery), suggesting that action potential synaptic transmission, rather than propagation (i.e. impulse conduction velocity along the sarcolemma), may be impaired during such exercise (figure 5[85]). However, whether a loss of membrane excitability contributes to fatigue is equivocal since a potentiation of the M-wave response has also been reported following a repeated-sprint cycle exercise.[27] As the actual size of the M-wave depends on the sum of a number of factors, e.g. the muscle investigated, the amount of preceding activity and/or the motor unit firing rates, further research is needed to elucidate the contribution of an impairment in muscle excitability to muscle fatigue induced by the repetition of sprints with a strict control of the above-mentioned influencing factors.

3.1.2 Limitations in Energy Supply

Phosphocreatine Availability

With total intramuscular stores of approximately 80 mmol • kg dry muscle $(dm)^{-1}$ [figure 6] and

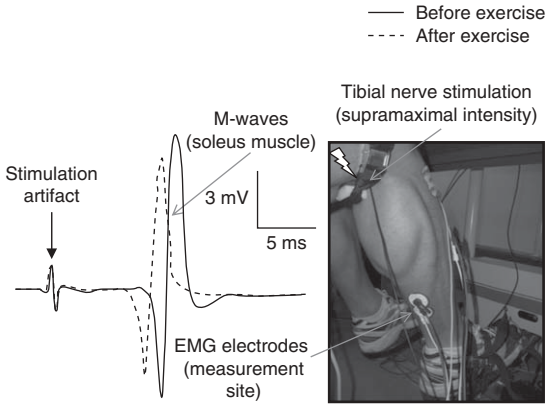

Fig. 5. Typical changes from one representative subject for the resting soleus muscle compound action potential (M-wave) before and after a repeated running sprint exercise (12×40 m with 30 sec of recovery). By analysing the changes in the electrically evoked M-wave following a supra-maximal tibial nerve stimulation, surface electromyography (EMG) can be a reliable non-invasive method to characterize sarcolemmal excitability. In human experiments, the M-wave is commonly used as an index of neuromuscular transmission (amplitude) and action potential propagation (duration) in muscle fibres. Note that the peak-to-peak amplitude of the M-wave recorded after the repeated running sprints was depressed compared with values obtained before exercise (adapted from Perrey et al.[85])

maximal turnover rates approaching 9 mmol • kg dm^{-1} s^{-1},[86] phosphocreatine represents the most immediate reserve for the rephosphorylation of adenosine triphosphate (ATP). As a consequence, phosphocreatine is particularly important during RSE, where a high rate of ATP utilization and resynthesis is required. In this respect, it is interesting to note that stores after a maximal 6-second sprint can be reduced to around 35–55% of resting levels,[22,87] and that the complete recovery of phosphocreatine stores can require more than 5 minutes.[15,88] In addition, human skeletal muscle fibres have been reported to have fibre-type-dependent differences in the usage of 'high-energy' phosphates with greater phosphocreatine reduction in fast-twitch fibres than in slow-twitch fibres.[89,90] Fast-twitch fibres dominate power production during supra-maximal exercise such as RSE. Thus, selective 'phosphocreatine deficit' of those fibres might be related to the failure to replicate performance when sprints are repeated.[91]

As recovery times during RSE generally do not exceed 60 seconds, the ATP/phosphocreatine stores may only be partially restored before the onset of subsequent exercise,[14,87] resulting in compromised performance during successive sprints.[22,92] Coupled with the fact that the recovery of power output and the resynthesis of phosphocreatine follow similar time courses, several authors have proposed

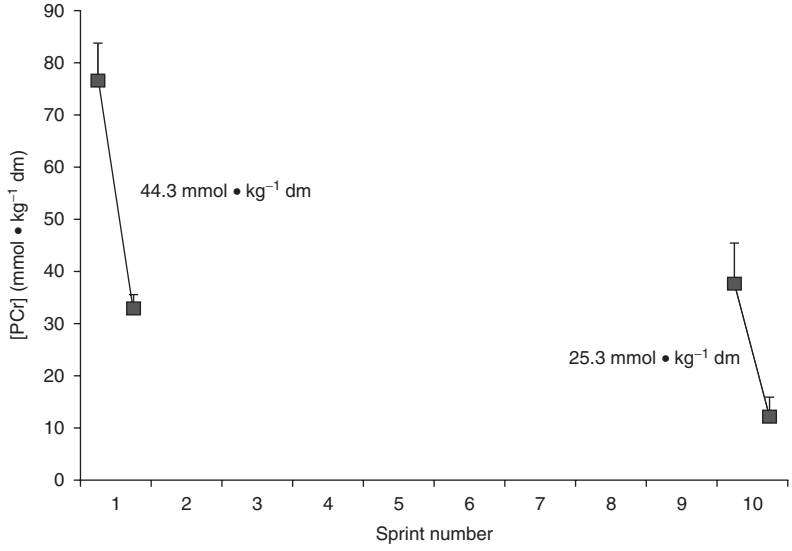

Fig. 6. Changes in phosphocreatine concentration ([PCr]) utilization before and after the first and last sprint of a 10×6 sec repeated-sprint test (with 30 sec of recovery between sprints) on a cycle ergometer (modified from Gaitanos et al.[22]). **dm** = dry muscle.

that performance during this type of work may become increasingly limited by phosphocreatine availability – i.e. a decrease in the absolute contribution of phosphocreatine to the total ATP production with each subsequent sprint (figure 6).[15,91] In line with this proposition, significant correlations have been reported between the resynthesis of phosphocreatine and power output recovery in the first 10 seconds of a second 30-second sprint ($r = 0.84$; $p < 0.05$)[14] and the partial restoration of repeated-sprint performance (i.e. total work done) following a fatigue-induced reduction in muscle phosphocreatine stores ($r = 0.67$, $p < 0.05$) [Mendez-Villanueva A, et al., unpublished observations]. This would suggest that better maintenance of muscle power output could be attributed to a faster rate of phosphocreatine resynthesis during the recovery between sprints.

Anaerobic Glycolysis

Anaerobic glycolysis supplies approximately 40% of the total energy during a single 6-second sprint, with a progressive inhibition of glycolysis as sprints are repeated (figure 7).[22,94] For instance, Gaitanos et al.[22] reported an 8-fold decrease in the absolute ATP production from glycolysis from the first to the last sprint of 10×6-second maximal sprints interspersed with 30-second recovery periods. It is unclear, however, whether increasing the maximal anaerobic glycogenolytic and glycolytic rate will lead to improvements in RSA. For example, it could be argued that training which increases the ability to supply ATP from anaerobic glycolysis would be detrimental to RSA, as individuals with the greatest decrements in power output during RSE have been reported to have the greatest glycolytic rate during the first sprint.[26] However, it also needs to be considered that subjects with a greater glycogenolytic rate have also been reported to have a greater initial sprint performance[15] and that there is a strong correlation between initial sprint performance and both final sprint performance and total sprint performance[26,47] during tests of RSA. Thus, while these findings highlight the difficulties associated with interpreting contrasting effects on the various RSA test measures, they also point out the need for future investigations that should determine whether increasing the anaerobic contribution is likely to improve both initial and mean sprint performance, and thus the ability to perform repeated sprints.

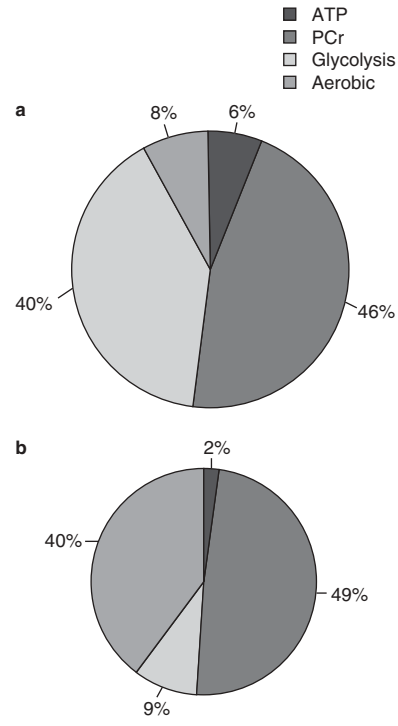

Fig. 7. Changes in metabolism during (**a**) the first and (**b**) the last sprint of a repeated-sprint exercise.[55,62,93] Note that the area of each circle represents the total absolute energy used during each sprint. **ATP** = adenosine triphosphate; **PCr** = phosphocreatine.

Oxidative Metabolism

The contribution of oxidative phosphorylation to total energy expenditure during a single short sprint is limited (<10%).[95,96] As sprints are repeated, however, the level of aerobic ATP provision progressively increases such that aerobic metabolism may contribute as much as 40% of the total energy supply during the final repetitions of a RSE (figure 8).[95] Furthermore, subjects may reach their $\dot{V}O_{2max}$ (moderate to high values generally ranging from 50 to 65 $mL \bullet min \bullet kg^{-1}$ across racket and team sports)[4,11] during the latter sprints.[95,97] This suggests that the aerobic contribution during RSE may be limited by $\dot{V}O_{2max}$ and that increasing $\dot{V}O_{2max}$ via appropriate training (as discussed in the subsequent companion review[40]))[98] and/or

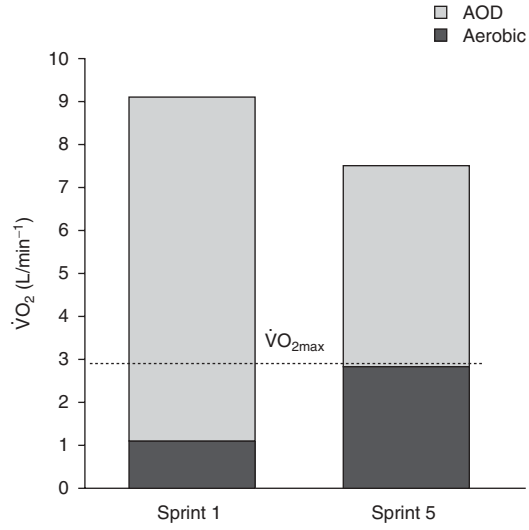

Fig. 8. As sprints are repeated, there is an increase in the aerobic contribution to individual sprints. The dashed line represents the maximal oxygen uptake ($\dot{V}O_{2max}$) [adapted from McGawley and Bishop[95]]. **AOD** = accumulated oxygen deficit; $\dot{V}O_2$ = oxygen uptake.

ergogenic aids (e.g. erythropoietin or EPO)[99] may allow for a greater aerobic contribution during the latter sprints, potentially minimizing fatigue. This hypothesis may explain why subjects with a greater $\dot{V}O_{2max}$ are more able to maintain power output/sprint times during a RSE, and is supported by significant correlations ($r = -0.45$ to -0.75) between $\dot{V}O_{2max}$ and performance fatigue indices (e.g. S_{dec} or FI).[51,77,100-102] This is not a universal finding, however, with others reporting low to nonsignificant correlations between $\dot{V}O_{2max}$ and FIs during RSE ($r = -0.20$ to 0.30).[47,93,103-108] This mixed view regarding the effect of an elevated $\dot{V}O_{2max}$ on RSA may be due to the difference between studies for the fitness level of the tested subjects and/or the nature of the RSE, together with the use of relatively homogenous samples. The absence of stronger correlations between $\dot{V}O_{2max}$ and RSA may also be related to the belief that the primary factor limiting $\dot{V}O_{2max}$ is the ability of the cardiorespiratory system to deliver O_2 to the exercising muscles (i.e. central factors), whereas RSA may be primarily limited by muscle disturbances (i.e. peripheral factors).[109] This is supported by the observation that the FI during a RSE has been reported to be inversely correlated with maximal ADP-stimulated mitochondrial respiration measured directly in muscle fibres.[110]

Interestingly, subjects who desaturated the most during a prolonged RSA test had the greatest work decrement over 20×5-second cycle sprints (and also had the lowest $\dot{V}O_{2max}$).[111] Other indirect measures of muscle oxidative capacity, such as oxygen consumption ($\dot{V}O_2$) kinetics[97,112] and the velocity at the onset of blood lactate accumulation,[113] have also been reported to be correlated with RSA. For example, subjects with faster $\dot{V}O_2$ off-kinetics during exercise of severe intensity (two runs at 120% of maximal aerobic speed, interspersed by 6 minutes of recovery, until exhaustion) are those with the smallest sprint decrement score during a repeated-sprint test.[112]

Finally, using the near-infrared spectroscopy (NIRS) technique, two studies have shown that the increase in muscle deoxyhaemoglobin engendered by sprint repetitions remains fairly constant.[27,29] This indicates that despite a progressive deoxygenation as sprints are repeated, the ability of the subjects to use available oxygen during RSE may be well preserved. Furthermore, the rate of muscle reoxygenation during the recovery periods between sprints would be an important factor limiting RSA, as evidenced by the strong correlation between the improvement of this parameter and the sprint decrement calculated for shuttle sprints after an 8-week endurance training programme.[114] Nevertheless, future studies combining pulmonary gas exchange and muscle oxygenation kinetics during RSE are needed to determine whether both 'central' (cardiovascular) and 'peripheral' (at the skeletal muscle level) impairments to oxidative pathways contribute to performance decrements.

3.1.3 Metabolite Accumulation

Acidosis

It has been argued that the considerable increases in muscle[51,65] and blood[47,64] hydrogen ion (H^+) accumulation that occur during RSE may affect sprinting performance via adverse effects on the contractile machinery and/or through the inhibition of ATP derived from glycolysis, possibly via negative effects on phosphofructokinase and glycogen phosphorylase.[115] In support of this suggestion, correlations have been observed between sprint

decrement and both muscle buffer capacity and changes in blood pH.[26,47,51] Furthermore, the content of skeletal muscle monocarboxylate transporters (i.e. MCT1), which facilitate the intramuscular lactate and H^+ removal process, has been inversely correlated with FIs calculated during ten successive 10-second cycling sprints (30 seconds of recovery).[116]

At physiological temperatures, however, acidification as a direct cause of muscle fatigue has been challenged for at least three reasons as follows: (i) the time course of the recovery of force/power following a bout of intense/maximal work is much faster than that of pH; (ii) high power outputs have been obtained under acidic conditions; and (iii) the ingestion of sodium bicarbonate (known to increase extra-cellular buffering capacity) has, in some cases, been reported to have no effect on RSA.[117,118] Furthermore, no significant correlations have been observed between the recovery of muscle pH and short-term recovery of both single 30-second 'all-out' bouts[15] and repeated, 6-second cycling sprints (Mendez-Villanueva A, et al., unpublished observation). Further research is therefore needed to clarify the effects of H^+ accumulation on the aetiology of fatigue during RSE.

Inorganic Phosphate

Indirect evidence that impairment of excitation-contraction coupling (i.e. the mechanisms that link sarcolemmal depolarization to calcium release) may contribute to fatigue during RSE has been obtained from experiments that have used electrically evoked contractions. With this approach, peripheral contractile properties can be identified and isolated from components located upstream from the neuromuscular junction.[119] In the fatigued state, lower peak twitch force in both the plantar flexors[85] and the knee extensors[27] has been observed following two different repeated-sprint protocols, suggesting that contractile properties of the active muscles had become less optimal across repetitions. In line with these results, low-frequency fatigue (i.e. a decrease in the ratio between mechanical responses to tetanus at low- and high-frequency stimulations) has also been detected after repeated running sprints.[85] *In vitro* studies provide evidence that increased inorganic phosphate levels affect calcium release from the sarcoplasmic reticulum and/or myofibrillar calcium sensitivity, which in turn decrease the number of strong binding cross-bridges.[120,121] However, whether this scenario occurs during RSE is still unknown and requires further research.

3.2 Neural Factors

3.2.1 Neural Drive

As maximal sprint exercise demands high levels of neural drive,[122] failure to fully activate the contracting musculature, as assessed by surface electromyogram (EMG), will theoretically decrease force production and therefore reduce RSA. While not a universal finding,[24,59,85,117] a concurrent decline in mechanical scores and the amplitude of EMG signals (root mean square [RMS] and integrated EMG values) has been reported in several studies (table I).[21,27,29,50,72,111] When fatigue is mild (FI or sprint decrement score <10%), previous research has typically reported a steady level of neural activation during RSE.[24,59,85,117] However, when the fatigue level is more substantial (>10%), a concurrent decline in mechanical performance and the amplitude of EMG signals has consistently been reported across sprint repetitions.[21,27,50] This suggests that under conditions of considerable fatigue, failure to fully activate the contracting musculature may become an important factor contributing to fatigue during RSE.

Interestingly, changes in quadriceps EMG amplitude explained 97% of the variance in total work done during ten successive cycle sprints, interspersed with 30 seconds of rest.[21] Billaut and Smith[72] also add that there is no sex dimorphism in this relation ($r^2 = 0.97$ and 0.86 in men and women, respectively; $p < 0.05$). The reduction in power output during RSE, however, makes it difficult to relate neural adjustments to RSA, as the lower EMG activity could also be the consequence, rather than the cause, of the reduced power output. It is also noteworthy that resistance load affects surface EMG activity during RSE, as a decrement in the RMS value was observed from the first to the ninth repetition when ten 10-second cycling sprints were performed under light-load but not heavy-load conditions.[53] Furthermore, difficulties in inter-

Table I. A summary of the characteristics and results of studies that have investigated changes in muscle activation parameters during cycling repeated-sprint exercise (RSE)[a]

Study (y)	RSE				Fatigue effects	
	no. of subjects and training level	repetitions	sprint duration (sec)	recovery duration (sec)	mechanical changes	muscle activation changes
Billaut et al.[123] (2005)	12 physically active	10	6	30	↓ PPO (11%), PR (7%) and PT (14%) from sp1 to sp10	→ $iEMG_{sprint}$ during RSE ↓ activation time delays (~90 min) between VL and BF EMG onsets from sp1 to sp10
Billaut et al.[124] (2006)	12 physically active	10	6	30	↓ PPO (8%, 10% and 11% after sp8, 9 and 10, respectively) ↓ MVC_{post} (13%) and MVC_{+5min} (10%)	↑ VL RMS_{MVC} (~15%) post-RSE → VM RMS_{MVC} (~ +10%) post-RSE ↓ VL and VM MF_{MVC} (~15%) post-RSE
Billaut and Basset[59] (2007)	13 physically active	10	6	30	↓ PPO and MPO (~9–13%) from sp1 to sp8 ↓ MVC_{post} (~10%) → MVC_{+5min} (~ −8%) and MVC_{+10min} (~ −6%)	→ VL RMS_{MVC} (~+10%) → MF_{MVC} (~ −8%)
Billaut and Smith[72] (2009)	32 trained	20	5	25	↓ TW from s1 to sp7 (~9%) and sp20 (~25%)	↓ $iEMG_{sprint}$ (~8% from sp1 to sp9 + 15.5% across all reps) ↓ VL, RF and GM MF_{sprint} (~10–13%) from sp1 to sp20
Billaut and Smith[111] (2010)	15 trained	20	5	25	↓ TW across reps (~23.5%)	↓ $iEMG_{sprint}$ (~14.2% vs −16.4% in VL and RF, respectively)
Giacomoni et al.[79] (2006)	12 trained	10	6	30	↓ PPO (~10% and 11%), PT (~2% and 9%) and TW (~16%) from sp1 to sp10 in the morning and evening, respectively. ↓ MVC_{post} (~15% and 13%) and MVC_{+5min} (~10% and 11%) in the morning and evening, respectively	↑ VL RMS_{MVC} (~7.5% and 10% in the morning and evening) after RSE
Hautier et al.[24] (2000)	10 physically active	15	5	25	↓ PPO (~11%), moment produced (~6%) and PR (~5%) from sp1 to sp13	↓ RMS_{sprint} in BF and GL (~13% and 17%) from sp1 to sp13 → RMS_{sprint} in GMax, VL and RF from sp1 to sp13
Matsuura et al.[125] (2006)	8 trained	10	10	35	↓ PPO (~17%) from sp1 to sp10	↓ VL and RF $iEMG_{sprint}$ (~12–15% and 20%) from sp1 to sp10 → VL and RF MPF_{sprint} from sp1 to sp10
Matsuura et al.[117] (2007)	8 trained	10	10	30	↓ PPO and MPO (~25%) after sp8	→ VL RMS_{sprint} during RSE ↓ VL MPF_{sprint} (~5–10%) after sp3 and sp7 only
Mendez-Villanueva et al.[50] (2007)	8 physically active	10	6	30	↓ PPO (~24%) from sp1 to sp10 ↓ TW (~27%) from sp1 to sp10	↓ VL RMS_{sprint} (~14%) from sp1 to sp10 ↓ VL MF_{sprint} (~11%) from sp1 to sp10

Continued next page

Table I. Contd

Study (y)	RSE				Fatigue effects	
	no. of subjects and training level	repetitions	sprint duration (sec)	recovery duration (sec)	mechanical changes	muscle activation changes
Mendez-Villanueva et al.[21] (2008)	8 physically active	10	6	30	↓ PPO and MPO from sp1 to sp5 (–14% and –17%) and from sp1 to sp10 (–25 and –28%), respectively	↓ VL RMS_{sprint} from sp1 to sp5 (–9%) and from sp1 to sp10 (–14%)
Racinais et al.[27] (2007)	9 physically active	10	6	30	↓ PPO (–10%) from sp1 to sp10 ↓ MVC_{post} (16.5%)	↓ VL RMS_{sprint} (–14%) ↓ VL RMS/M_{MVC} (14.5%) and VA (2.5%)
Smith and Billaut[29] (2010)	13 trained	10	10	30	↓ TW (overall decrement –31.2%) from sp2 to sp10	↓ VL $iEMG_{sprint}$ (–12.4% vs 26.5% in normoxia and hypoxia, respectively) from sp1 to sp6–10

a All data were collected during cycle-based repeated sprinting protocols using a passive recovery mode between sprints.

BF = biceps femoris; **EMG** = electromyogram; **GL** = gastrocnemius lateralis; **GM** = gastrocnemius medialis; **GMax** = gluteus maximus; $\mathbf{iEMG_{sprint}}$ = during sprinting after integrated EMG; $\mathbf{MF_{sprint}}$ = median frequency during sprinting; $\mathbf{MF_{MVC}}$ = median frequency during maximal voluntary contraction; $\mathbf{MPF_{sprint}}$ = mean power frequency during sprinting; **MPO** = mean power output; $\mathbf{MVC_{+5min}}$ and $_{+10min}$ = maximal voluntary contraction torque measured +5min and +10min after RSE; $\mathbf{MVC_{post}}$ = maximal voluntary contraction torque post-exercise; **PPO** = peak power output; **PR** = maximal pedalling rate; **PT** = peak torque applied on the crank; **reps** = repetitions; **RF** = rectus femoris; $\mathbf{RMS/M_{MVC}}$ = normalized root mean square activity obtained during maximal voluntary contraction; $\mathbf{RMS_{MVC}}$ = RMS measured during maximal voluntary contraction; $\mathbf{RMS_{sprint}}$ = RMS measured during sprinting; **sp** = sprint; **TW** = total work; **VA** = voluntary activation; **VL** = vastus lateralis; **VM** = vastus medialis; ↓ indicates decrease; ↑ indicates increase; → indicates no significant change.

preting EMG data need to be acknowledged (e.g. amplitude cancellation phenomena, excessive sweat, changes in fibre membrane and motor unit properties).[126] For instance, interpretation of EMG scores (e.g. RMS) is complicated by the fact that, at the muscle level, EMG signal can be influenced by modifications in sarcolemmal excitability.[127] Future studies should therefore control for potential modifications in M-wave amplitude (i.e. an index of sarcolemmal excitability)[27] obtained after the successive sprint bouts, and calculate the ratio of the EMG signal to the M-wave response (i.e. RMS/M-wave ratio) to ensure that neural input reaching the neuromuscular junction effectively decreases with fatigue. Nonetheless, the suboptimal motor unit activity (i.e. a decrease in recruitment, firing rate or both), inferred by changes in surface EMG activity, has also been highlighted via the MRI technique[128] and interpolated-twitch results obtained during post-RSE assessment of neuromuscular function (figure 9).[27,85] These results have implications in the context of multiple-sprint sports, since muscle activation is known to influence the sensorimotor control of force with fatigue,[129] which may in turn negatively affect the quality of specific sporting skills, and potentially increase the risk of injury (e.g. extreme range of motion and/or higher mechanical stress/load imposed on joints).[130]

The mechanisms that lead to a decreased motor unit activity of the active muscle – notably in the context of RSE – are still not well understood. Nevertheless, it has been proposed that the CNS receives sensory input from muscle afferents (e.g. muscle spindles, Golgi tendon organs, free endings of group III and IV nerves), which are incorporated into the determination of central neural drive, to adjust for the rate of intramuscular fatigue-related metabolite accumulation (e.g. H^+ and phosphate), with the purpose of avoiding the development of peripheral fatigue beyond a given individual threshold.[131] This viewpoint is supported by studies demonstrating a correlation between H^+ accumulation and change in group III and IV muscle afferents discharge rate.[132] To date, however, it remains unclear whether afferent feedback from fatigued muscle differs according to the continuous/intermittent nature of the exercise task.[133,134] Regarding RSE, it is reasonable to expect that the

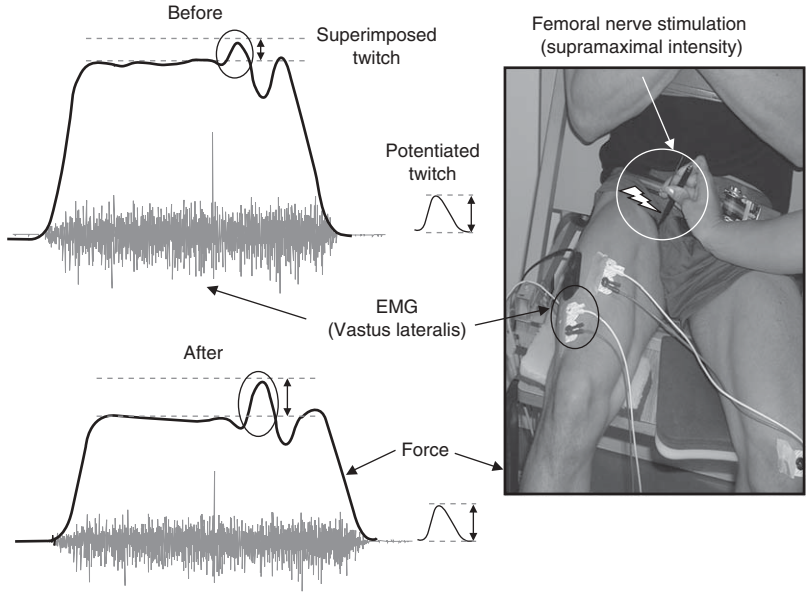

Fig. 9. Typical force and electromyogram (EMG) [vastus lateralis muscle] traces during knee extensor maximal voluntary contractions (MVC) before and after a repeated-sprint cycling exercise (10×6 sec with 30 sec of recovery). By comparing the twitch superimposed to a MVC and the twitch evoked on the relaxed muscle (i.e. femoral nerve supra-maximal stimulation), the twitch interpolation technique in conjunction with surface EMG (i.e. root mean square [RMS] value normalized by the maximal muscle compound action potential [M-wave]) can be a reliable non-invasive technique to characterize muscle activation. Voluntary activation level (%) was estimated according to the following formula: (1 – [superimposed twitch/potentiated twitch)]×100). Note that both the voluntary activation level and the normalized RMS activity were depressed (–2.5% and –14.5%, respectively) compared with values obtained before exercise (adapted from Racinais et al.[27]).

dramatic metabolic disruptions occurring within the muscle cell (see section 3.1.3) might also have consequences for central neural drive. However, the manipulation of intramuscular pH by oral administration of sodium bicarbonate did not affect surface EMG activity or peak/mean power output during repeated cycling sprints.[117] In this regard, it has also been suggested, in a sprint-like exercise, that sensory feedback signalling peripheral fatigue might be suppressed, blocked or simply counteracted by a stronger central command.[135,136] In addition to muscle afferents sensitive to metabolite accumulation, the reduction in arterial O_2 saturation (and brain oxygenation) has been shown to be a major determinant of the attenuation in motor unit activity.[111] When 15 national-level soccer players performed 20 × 5-second cycle sprints (25 seconds of rest), Billaut and Smith[111] have reported that the progressive arterial O_2 desaturation that develops across repetitions is strongly correlated with reductions in mechanical work and in the surface integrated EMG ($r^2 = 0.68$ and 0.62, respectively; $p < 0.05$). These findings are compatible with those of several *in vitro* and *in vivo* experiments, demonstrating that motor cortex excitability and neuromuscular activity are influenced by O_2 availability.[137,138]

A decrease in motor unit activity can theoretically arise from changes at the spinal level and/or supraspinal factors. At the spinal level, changes in the number of α-motoneurons that are recruited in the motoneuron pool (i.e. motoneuron excitability) can be detected with the Hoffman (H)-reflex recordings.[139] As no modification in the soleus resting H-reflex response was observed following 12×40 m sprints (30 seconds of recovery), preliminary evidence therefore questions the possibility of a reduced reflex excitability of spinal α-motoneurons as a fatigue agent during RSE.[85] Under fatigue, adaptations in neural function can also result in a reduced efficiency in the generation of the motor command due to supraspinal factors, as potentially

reflected by disturbances in brain electrical activity, cortical excitability and/or brain neurotransmitter (e.g. serotonin, dopamine, acetylcholine) concentration.[140,141] While no study has directly investigated whether these changes can compromise RSA, those observations are consistent with high-intensity, exercise-induced, post-fatigue impairments in supraspinal function as reductions in cortical drive[142,143] and corticospinal synaptic transmission.[144,145] However, the actual relevance that these changes in the CNS can have on RSA remains largely unknown. In this regard, the maintenance of motor neuron activity and power output may be due to entirely different mechanisms.[146] For example, it is known that exercise-induced fatigue can be caused by slowed muscle contractile properties.[142,146] As the relaxation rate of the muscle declines during fatigue, the lower neural stimulation frequency (i.e. firing rates) maintains the tetanus and thus optimizes maximum force production because the metabolic activity involved in the contractions changes the muscle properties in tandem.[147,148] Further studies are therefore needed to better understand the role of decreases in neural drive on the aetiology of fatigue during RSE and the spinal/supraspinal mechanisms of these adaptations. To reach this target, neural adaptations should be studied in terms of neurotransmitter turnover (e.g. serotonin and dopamine concentrations), cerebral oxygenation (e.g. NIRS), brain electrical activity (e.g. electroencephalography), and cortical excitability (e.g. transcranial magnetic stimulation).

3.2.2 Muscle Recruitment Strategies

An additional neural factor that may contribute to fatigue during RSE is a modification of muscle recruitment strategies. Billaut et al.[123] have reported that the time delay between the knee extensor and the flexor EMG activation onsets was reduced during the last sprint of a RSE, owing to earlier antagonist activation with fatigue occurrence. In the same study, no modification of the integrated EMG score was noted across sprint repetitions. This suggests that changes in inter-muscle coordination (e.g. vastus lateralis/biceps femoris coordination pattern) could contribute to the power output reduction under fatigue during RSE. Using 15 × 5-second cycle sprints separated by 25 seconds of recovery, Hautier et al.[24] observed a significant reduction in RMS for the knee flexor muscles in the thirteenth compared with the first sprint without changes in RMS for the knee extensor muscles, highlighting a possible decrease in muscle coactivation with fatigue. The lower activation of antagonist muscles after fatigue has been interpreted by these authors as an efficient adaptation of the inter-muscular coordination to transfer reduced force and power to the pedal.

A shift in median frequency values (obtained during maximal isometric voluntary contractions) toward lower frequencies has also been reported post-RSE.[124] This has been interpreted as a modification in the pattern of muscle fibre recruitment; i.e. decreased recruitment of fibres with faster conduction velocities. More tellingly, it is probable that the relative contribution of type I muscle fibres involved in force generation may increase during RSE as a result of the greater fatigability of type II fibres, highly solicited during this exercise mode.[149] Caution is needed, however, when interpreting these data since the external validity of standard tests for strength production capacity (maximal voluntary contractions) to reflect fatigue resistance could be challenged due to methodological differences in subject's positioning and/or muscle contraction mode.[150] However, using EMG recordings during cycle-based repeated sprinting, Matsuura et al.[125] concluded that greater fatigue was linked to a decreased preferential recruitment of fast-twitch motor units as mean power frequency was higher with 35-second than with 350-second recovery periods.

4. Additional Factors Affecting RSA

4.1 Stiffness Regulation

Although not as extensively studied, changes in mechanical behaviour (stiffness regulation) may also indirectly alter fatigue resistance during repeated running sprints.[122] It is generally believed that a stiffer system allows for a more efficient elastic energy contribution, potentially enhancing force production during the concentric phase of the movement.[151] Supported by the close relationship between leg stiffness and sprint running per-

formance,[152] it has been argued that stiffness regulation is a vital component for setting stride frequency.[151] In line with this statement, decreased stride frequencies have been shown to accompany fatigue development during run-based repeated sprinting.[55,64] The clear relationship between impairment of spring-mass model properties of runners' lower limbs and the decrease in performance induced by the repetition of all-out efforts (4×100 m interspersed with 2 minutes of recovery) has previously been highlighted.[153] Marked alterations in leg-spring behaviour were also reported by Girard et al.[154] when 16 active subjects performed 12×40 m sprints interspersed with 30 seconds of passive recovery. As peak vertical force was not modified by sprint repetitions, fatigue-induced slower stride frequencies, stemming from changes in vertical displacement of the centre of mass (decreased vertical stiffness) rather than changes in leg length (preserved leg stiffness), are likely to explain the longer sprint times across trials.[154] Interestingly, athletes participating in a training programme that includes field, resistance, plyometric and eccentric training are more successful than recreational sporting participants at regulating joint stiffness during repeated running sprints.[155] These findings may support the view that the ability to maintain a high level of stiffness condition may improve fatigue resistance during RSE. Further research is required to verify this hypothesis.

4.2 Environmental Perturbations

RSA is also likely to be further compromised for subjects who are exposed to environmental stresses, such as when competing in hot environments (Tennis Australian Open) or at altitude (2010 Football World Cup in South Africa). The experimental approach of using an additional environmental stress to further perturb homeostasis is also potentially useful to gain knowledge regarding the nature of the mechanisms limiting RSA. To date, little is known about the impact of such influencing factors on RSA since most of our knowledge in this area comes from intermittent-sprint exercises (e.g. heat exposure;[17,156] simulated altitude[157,158]). Nevertheless, investigating how thermal stress affects power output during repeated 30-second bouts of maximal, cycling exercise – each bout being separated by 4 minutes of recovery – Ball et al.[13] have reported increased peak and mean power outputs (~25% and 15%, respectively) when the 30-second all-out bouts were performed in a warm (~30°C, 55% relative humidity) versus a thermo-neutral (~19°C, 40% relative humidity) environment; this was due to achieving a higher pedal cadence in the heat, probably through an elevated rate of anaerobic ATP turnover and muscle fibre conduction velocity.[159] When the intermittent-sprint and repeated-sprint performance (i.e. a pattern of $5 \times$ 2-second sprints, separated by ~21 seconds, reproduced twice) of subjects performing 0, 10 and 20 minutes of active warm-up prior to a 36-minute intermittent-sprint test performed in hot conditions (35.5°C, 49% relative humidity) was measured, Bishop and Maxwell[160] found a greater increase in core temperature following the longer warm-up, which was associated with a compromised repeated-sprint performance. Drust et al.[161] have also reported an impaired ability to produce power during five 15-second all-out efforts on a cycle ergometer when core and muscle temperatures are simultaneously elevated. This impairment in performance occurred after the completion of the first sprint and was observed following both the completion of a 40-minute intermittent exercise in a hot environment and after passively induced hyperthermia. In the absence of metabolic changes (i.e. muscle lactate, extra-cellular potassium), these authors have associated the larger reduction in peak and mean power output in the heat to the negative influence of a high core temperature on the function of the CNS (e.g. alterations in brain activity, reductions in cerebral blood flow, increases in whole-brain energy turnover, reduced muscle activation). Despite a greater physiological strain experienced with the greatest level of hypo-hydration, reductions in work performed and peak power output were only observed after a second, intense RSE during the latter stages of a 36-minute, cycling, intermittent-sprint test.[162]

To date, little research has investigated the role of hypoxia on RSA.[29,163] In one study, Balsom et al.[163] examined the influence of oxygen availability on factors responsible for fatigue during 10×6-second cycle sprints interspersed with

30-second rest periods. While subjects were instructed to try and maintain a pedalling frequency of 140 revolutions per minute throughout each sprint, pedalling frequencies during the final 2 seconds of the last two repetitions were lower under hypoxic than under normoxic conditions. A slower on-transient $\dot{V}O_2$ response, as a result of reduced oxygen availability, would increase the magnitude of the O_2 deficit incurred during each sprint and thereby place more demand on anaerobic sources to maintain the required rate of ATP provision. The increased rate of fatigue under hypoxic conditions may have also been the result of a more rapid accumulation of inorganic phosphate during each sprint and a reduced rate of removal during recovery.[164] Nevertheless, the consequences of reduced oxygen content are not exclusively ascribed to peripheral sources, as CNS function during exercise in hypoxia also has the potential to curtail muscle activation and therefore alter fatigue resistance.[165] Using NIRS measurements, it has been reported that an earlier and larger degree of deoxygenation of the prefrontal cortex in acute moderate hypoxia (i.e. insufficient O_2 delivery and/or low pressure gradient to drive the diffusion of O_2 from the capillaries to the mitochondria) was associated with impairments in RSA.[29] Little is known, however, about these cortical dysfunctions, especially as most of our knowledge in this area concerns continuous locomotor tasks.

5. Conclusions

During RSE, the inability to reproduce performance in subsequent sprints (fatigue) is manifested by a decline in sprint speed (running) or peak/mean power output (cycling). Although many issues remain unresolved, proposed factors responsible for fatigue include limitations in energy supply (e.g. phosphocreatine content and $\dot{V}O_2$) and metabolic by-product accumulation (e.g. inorganic phosphate, H^+). Although not as extensively studied, failure to fully activate the contracting muscle may also compromise fatigue resistance during RSE. Moreover, the details of the task (e.g. changes in the nature of the work/recovery bouts) and additional environmental perturbations will determine the relative contribution of the underlying mechanisms (task dependency) to fatigue. Interventions (e.g. ergogenic aids or training) that can lessen the influence of these limiting factors should improve RSA (as discussed in the subsequent companion review[40]).

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