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Repeated-Sprint Ability – Part II

Recommendations for Training

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Abstract

Short-duration sprints, interspersed with brief recoveries, are common during most team sports. The ability to produce the best possible average sprint performance over a series of sprints (≤10 seconds), separated by short (≤60 seconds) recovery periods has been termed repeated-sprint ability (RSA). RSA is therefore an important fitness requirement of team-sport athletes, and it is important to better understand training strategies that can improve this fitness component. Surprisingly, however, there has been little research about the best training methods to improve RSA. In the absence of strong scientific evidence, two principal training theories have emerged. One is based on the concept of training specificity and maintains that the best way to train RSA is to perform repeated sprints. The second proposes that training interventions that target the main factors limiting RSA may be a more effective approach. The aim of this review (Part II) is to critically analyse training strategies to improve both RSA and the underlying factors responsible for fatigue during repeated sprints (see Part I of the preceding companion article). This review has highlighted that there is not one type of training that can be recommended to best improve RSA and all of the factors believed to be responsible for performance decrements during repeated-sprint tasks. This is not surprising, as RSA is a complex fitness component that depends on both metabolic (e.g. oxidative capacity, phosphocreatine recovery and H^+ buffering) and neural factors (e.g. muscle activation and recruitment strategies) among others. While different training strategies can be used in order to improve each of these potential limiting factors, and in turn RSA, two key recommendations emerge from this review; it is important to include (i) some training to improve single-sprint performance (e.g. 'traditional' sprint training and strength/power training); and (ii) some high-intensity (80–90% maximal oxygen consumption) interval training to best improve the ability to recover between sprints. Further research is required to establish whether it is best to develop these qualities separately, or whether they can be developed concurrently (without interference effects). While research has identified a correlation between RSA and total sprint distance during soccer, future studies need to address whether training-induced changes in RSA also produce changes in match physical performance.

1. Introduction

Short-duration sprints (≤10 seconds), interspersed with brief recovery periods, are common during most team sports.[1] The ability to produce the best possible average sprint performance over a series of sprints, separated by short (≤60 seconds) recovery periods, is therefore important for all team-sport athletes and has been termed repeated-sprint ability (RSA). While RSA is often equated with a low fatigue index (i.e. the decrease in performance from the first to the last sprint), it is important to note that a good RSA is better described by a high average sprint performance, with or without a low fatigue index (e.g. a marathon runner with a low average sprint performance, but a very low fatigue index, would not be classified as having good repeated-sprint ability) [see also Part I of the preceding companion article[2]]. Mean time recorded during an RSA test predicts the distance of high-intensity running (>19.8 km/h), and the total sprint distance during a professional soccer match.[3] This suggests that improving RSA should result in greater team-sport physical performance, and that it is important to better understand training strategies that can enhance this fitness component.

Recently, there has been an increase in scientific research regarding the importance of RSA for team- and racket-sport athletes.[1,3-7] Surprisingly, however, there has been little research about the best training methods to improve this fitness component.[8] In the absence of strong scientific evidence, one concept that has emerged is that the best way to train RSA may be to perform repeated sprints.[9] While such a concept appeals to the concept of training specificity, the scientific evidence in support of this approach is currently lacking. Indeed, many studies have reported significant improvements in RSA with more generic training (e.g. interval training).[9-12] The aim of this review is to critically analyse training strategies to improve both RSA and the underlying factors responsible for fatigue during repeated sprints.

In order to obtain the necessary articles for this review, several databases were searched including SportDiscus®, PubMed, Web of Science, MEDLINE and Google Scholar. Key search terms used included 'repeated-sprint ability', 'repeated-sprint exercise', 'multiple sprints', 'team sports', 'training', 'rugby', 'soccer', 'football', 'basketball', 'conditioning', 'endurance' and 'small-sided games'. Manual searches were also made using the reference lists from recovered articles. Due to the small number of articles relating to training and RSA, there was no limit to the search period.

2. Training the Limiting Factors

During repeated-sprint exercise (RSE), the inability to reproduce performance across sprint repetitions (fatigue) is manifested by a decline in sprint speed (i.e. increased time to cover a fixed distance) or peak/mean power output. Proposed factors responsible for these performance decrements have previously been reviewed[13] (see also Part I of this review[2]) and include limitations to energy supply (e.g. phosphocreatine resynthesis, aerobic and anaerobic glycolysis) and metabolite accumulation (e.g. inorganic phosphate [P_i], H^+). Increasing evidence suggests that failure to fully activate the contracting muscle may also limit repeated-sprint performance.[14,15] Training interventions that are able to lessen the influence of these limiting factors should improve RSA.

2.1 Energy Supply

2.1.1 Phosphocreatine Resynthesis

As the brief recovery times between repeated sprints will lead to only a partial restoration of phosphocreatine stores,[16,17] it has been proposed that the ability to resynthesize phosphocreatine may be an important determinant of the ability to reproduce sprint performance.[17,18] In line with this proposition, strong relationships have been reported between phosphocreatine resynthesis and the recovery of performance during both repeated, 30-second, all-out exercise bouts[17,18] and repeated 6-second sprints (Mendez-Vallanueva A. et al., unpublished data). These findings suggest that the performance of repeated sprints may be improved by training interventions that increase the rate of phosphocreatine resynthesis.

The oxidative metabolism pathways are essential for phosphocreatine resynthesis during the recovery from high-intensity exercise.[19] This suggests that individuals with an elevated aerobic fitness (i.e. high maximal oxygen consumption [$\dot{V}O_{2max}$] or lactate threshold) should be able to more rapidly resynthesize phosphocreatine between repeated sprints. Indeed, cross-sectional research[17,20-23] and one training study[24] support the hypothesis that endurance training enhances phosphocreatine resynthesis following low-intensity exercise. Recently, it has also been reported that high-intensity interval training (6–12 × [2 minutes at ~100% $\dot{V}O_{2max}$: 1 minute rest]), can significantly improve phosphocreatine resynthesis during the first 60 seconds following high-intensity exercise (figure 1).[25] In contrast, no changes in the rate of phosphocreatine resynthesis have been reported following interval (8 × [30 seconds at

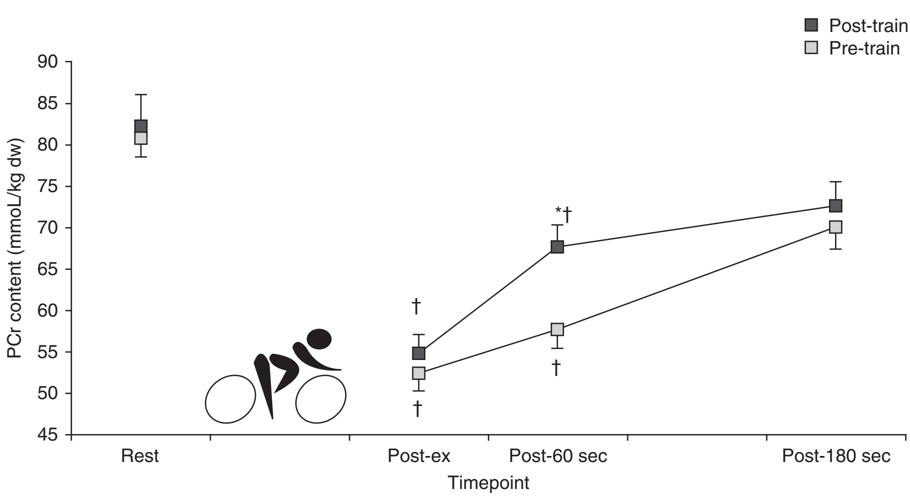

Fig. 1. Changes in resting and post-exercise phosphocreatine (PCr) content following high-intensity interval training ([25] and Bishop D. et al., unpublished research). **dw** = dry weight; * indicates significantly different from pre train; † indicates significantly different from rest.

~130% $\dot{V}O_{2max}$: 90 seconds rest]), or intermittent-sprint training (15×[6-second sprint: 1-minute jog recovery]),[12] or training involving repeated, 30-second, all-out efforts (4–7×[30 seconds 'all-out': 3–4 minutes rest]).[26] These results can possibly be attributed to the absence of significant changes in aerobic fitness (as measured by $\dot{V}O_{2max}$) with these types of training. Alternatively, these results may be related to the fact that these studies all measured phosphocreatine resynthesis 3-minutes post-exercise, a timepoint when phosphocreatine resynthesis is largely complete and therefore less likely to be influenced by training. Nonetheless, while the optimal training intensity has not yet been established, the limited research to date suggests that improvements in aerobic fitness may be required to improve phosphocreatine resynthesis. As repeated-sprint training has been reported to increase aerobic fitness,[4,27] further research is required to investigate whether this type of training can also increase the fast component (e.g. first 60 seconds) of phosphocreatine resynthesis, and whether such changes are superior to those observed following aerobic training (e.g. interval training[25]).

2.1.2 Anaerobic Glycolysis

The large drop in intramuscular phosphocreatine, along with the concomitant rise in P_i and adenosine monophosphate, stimulates the rapid activation of anaerobic glycolysis at the start of a sprint.[28] As a consequence, anaerobic glycolysis is an important source of adenosine triphosphate (ATP) during a single sprint.[29] During subsequent sprints however, there is a dramatic decrease in the ATP production, via anaerobic glycolysis, during sprint efforts that has been attributed to the acidosis resulting from the maximal anaerobic degradation of glycogen during the early sprints.[18,30] It is therefore unclear whether increasing the maximal anaerobic glycogenolytic and glycolytic rate will lead to improvements in RSA. On one hand, it could be argued that training that increases the ability to supply ATP from anaerobic glycolysis would be detrimental to RSA due to the negative correlation between anaerobic ATP production during the first sprint and sprint decrement during a repeated-sprint test.[29,31] On the other hand, it also needs to be considered that subjects with a greater glycogenolytic rate have also been reported to have a greater initial sprint performance,[29] and that researchers have reported a strong positive correlation between initial sprint performance and both final sprint performance[29] and total sprint performance[32,33] during tests of RSA. Thus, while these findings highlight the difficulties associated with interpreting contrasting effects on the various RSA test measures,[34] they suggest that increasing the anaerobic contribution is likely to improve both initial and mean sprint performance, and thus the ability to perform repeated sprints. It should be noted, however, that some researchers have reported significant increases in glycolytic enzymes following sprint training without a corresponding increase in sprint performance.[35,36] Further research is therefore required to investigate the relationship between improvements in anaerobic ATP production and RSA.

As training does not increase the amount of phosphocreatine breakdown during high-intensity exercise,[12,25,37] changes in the ability to produce ATP via anaerobic glycolysis are likely to be well reflected by training-induced changes in indirect measures of anaerobic capacity, such as maximal accumulated oxygen deficit (MAOD). A high rate of anaerobic energy release during exercise has been proposed to be an important stimulus to increase MAOD.[38] This is supported by increases in MAOD in response to high-intensity (20–120-second intervals at 100–200% $\dot{V}O_{2max}$),[38-40] but not moderate-intensity (60 minutes at 70% $\dot{V}O_{2max}$) endurance training.[40] Furthermore, the greatest changes in MAOD have typically been reported in response to interval training that produces large changes in blood lactate concentration (>10 mmol/L).[38,40] These results are consistent with the observation that training-induced changes in enzymes important for anaerobic glycolysis (e.g. phosphofructokinase and phosphorylase) are greater following training that involves repeated 30-second bouts than repeated 6-second bouts[41] or continuous training.[42] In the only study to date, 6 weeks of repeated-sprint training did not increase phosphofructokinase activity.[43] Greater increases in glycolytic enzymes have typically been reported when high-intensity intervals are separated by long (10–15 minute),[36,44] rather than by short (≤4 minute),[45-47] rest periods. This is consistent with the larger increases in peak blood or muscle lactate when 30-second all-out efforts are separated by 10- to 15-minute rest periods,[35,48] compared with 3–4-minute rest periods.[37] From this research, it is difficult to determine whether this is an effect of recovery duration *per se*, or the better maintenance of exercise intensity with longer recoveries. The above research suggests that to increase the anaerobic performance of team-sport athletes one should utilize short (20–30 second), high-intensity (all-out) intervals separated by relatively long rest periods (~10 minutes).

2.1.3 Aerobic Metabolism

Several physiological adaptations related to an increased reliance on aerobic metabolism to resynthesize ATP, such as greater mitochondrial respiratory capacity,[49] faster oxygen uptake kinetics,[50,51] an accelerated post-sprint muscle reoxygenation rate,[52] a higher lactate threshold[53] and a higher $\dot{V}O_{2max}$,[51,54-57] have been associated with an enhanced ability to resist fatigue during repeated sprints. The most studied factor is $\dot{V}O_{2max}$ that has been reported to be moderately correlated ($0.62 < r < 0.68$; $p < 0.05$) with RSA (both mean sprint performance and sprint decrement).[51,54-56] Research has also shown that subjects with a greater $\dot{V}O_{2max}$[58] have a superior ability to resist fatigue during RSE, especially during the latter stages of a repeated-sprint test when subjects may reach their $\dot{V}O_{2max}$.[59] This suggests that improving $\dot{V}O_{2max}$ may allow for a greater aerobic contribution to repeated sprints, potentially improving RSA. However, research also indicates that there is not a linear relationship between $\dot{V}O_{2max}$ and various repeated-sprint fatigue indices.[32,60] Thus, it may be more important to develop an 'optimal', rather than a maximal, $\dot{V}O_{2max}$. Further research is required to determine what an appropriate level of $\dot{V}O_{2max}$ is, above which further increases may not be accompanied by comparable improvements in RSA. In addition, the possible links between other aerobic fitness indices (e.g. lactate threshold, economy, oxygen kinetics, the velocity associated with $\dot{V}O_{2max}$), which are relatively independent of the $\dot{V}O_{2max}$, should be the subject of further research.

Many physiologists believe that it is the reduced muscle oxygen levels during training that provide the stimulus to increase $\dot{V}O_{2max}$.[61] As the oxygen level in the muscle decreases with increases in exercise intensity up to 100% $\dot{V}O_{2max}$, but does not decrease further once the exercise intensity exceeds this point,[62] this suggests that interval training at intensities that approximate $\dot{V}O_{2max}$ may be most effective for improving $\dot{V}O_{2max}$. This is supported by previous studies that have reported greater improvements in $\dot{V}O_{2max}$ after interval training (at approximately the $\dot{V}O_{2max}$ intensity) when compared with continuous training matched for total work.[61,63-65] It should be noted, however, that most of these studies performed their continuous training at very low intensities (≤56% of the power at $\dot{V}O_{2max}$).[61,63,64] When compared with continuous training performed at intensities >60% of the power at $\dot{V}O_{2max}$, interval training has been reported to produce similar improvement in $\dot{V}O_{2max}$.[66-69] These results therefore suggest that if a minimum training intensity is exceeded (>60% of the power output at $\dot{V}O_{2max}$), and total work is equivalent, the choice of either interval or continuous training will result in similar improvements in $\dot{V}O_{2max}$. However, one advantage of interval training is that it may concurrently develop other factors (e.g. the rate of phosphocreatine resynthesis[25] and muscle buffer capacity[69]). The above research suggests that to increase the aerobic fitness of team-sport athletes, one should utilize high-intensity interval training (80–90% of $\dot{V}O_{2max}$) interspersed with rest periods (e.g. 1 minute) that are shorter than the work periods (e.g. 2 minutes).

2.2 H^+ Accumulation

It has been argued that the considerable increases in muscle[58,69,70] and blood[32,71] H^+ accumulation observed following sprinting may impair repeated-sprint performance.[72] In support of this, correlations have been observed between sprint decrement, and both muscle buffer capacity (ßm) and changes in muscle and blood pH.[32,54,55,58,73] This suggests that RSA may be improved by interventions that can increase the removal of H^+ from the muscle.[12,54,73] The removal of intracellular H^+ during intense skeletal muscle contractions (such as repeated sprints) occurs via intracellular buffering ($\beta m_{in\ vitro}$) and a number of different membrane transport systems, especially the monocarboxylate transporters (MCTs) [figure 2].[74] The MCTs appear to be the dominant regulator of muscle pH during and after high-intensity exercise.[74]

A large increase in muscle H^+ and/or lactate during exercise has been proposed to be an important stimulus for adaptations of the muscle pH regulating systems.[75] This is supported by increases in $\beta m_{in\ vitro}$ in response to high-intensity interval training (6–10 × [2 minutes at 120–140% of the lactate threshold] : 1 minute rest), but not moderate-intensity, continuous training (~30 minutes at 80–95% lactate threshold).[69] However, greater accumulation of lactate and H^+ during training has not always been associated with greater increases in MCTs[12,76] or $\beta m_{in\ vitro}$.[25] Furthermore, research suggests that too large an accumulation of H^+ during training (e.g. interval training performed at intensities >100% $\dot{V}O_{2max}$) may have a detrimental effect on adaptations to the pH regulatory systems within the muscle.[25,77] Thus, while further research is required, it appears that intramuscular accumulation of H^+ and/or lactate provides an important stimulus to improve the muscle pH regulating systems; however, maximizing H^+ accumulation during training does not maximize these adaptations. It should be noted, however, that most of this research has been conducted on moderately-trained subjects and further research is required to confirm these observations in well trained, team-sport athletes.

The above considerations have important implications for the design of training programmes to improve the muscle pH regulating systems and hence, RSA. To increase $\beta m_{in\ vitro}$, it appears important to employ high-intensity interval training (~80–90% $\dot{V}O_{2max}$), interspersed with rest periods that are shorter than the work periods (e.g. 2 minutes of exercise followed by 1 minute of rest), so that the muscle is required to contract while experiencing a reduced pH.[69,78] Interval training at intensities >$\dot{V}O_{2max}$ does not appear to provide additional benefits, and has the potential to actually decrease $\beta m_{in\ vitro}$.[25] In addition, the use of rest periods that are longer than the work periods allows greater removal of lactate and H^+ prior to

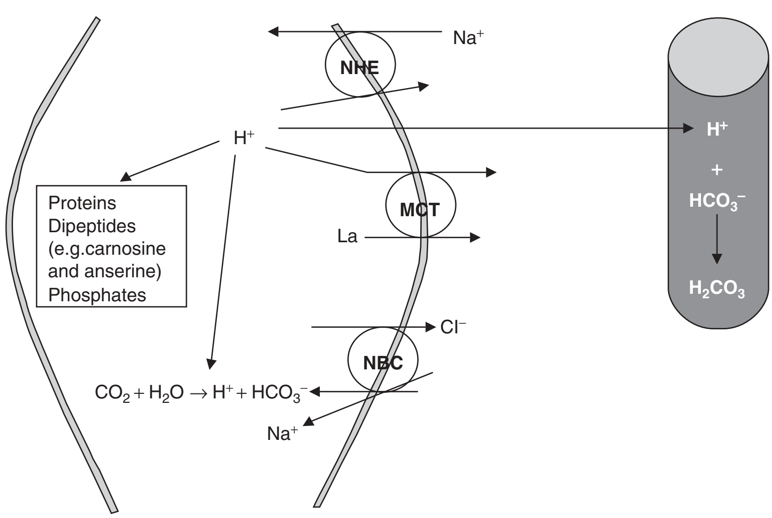

Fig. 2. Muscle (H^+) regulation. **MCT** = monocarboxylate transporters; **NBC** = sodium-bicarbonate co-transporter; **NHE** = sodium-hydrogen exchanger.

subsequent intervals[79] and typically does not result in a significant increase in $\beta m_{in\ vitro}$.[37,48] While an optimal training volume to improve $\beta m_{in\ vitro}$ is yet to be established, it appears that interval training at the above-mentioned intensities, 2–3 times per week, for 3–5 weeks, can result in significant increases in $\beta m_{in\ vitro}$.[69,75,80] In the only study to date, repeated-sprint training (5–8×[5×25–35 m sprints: 21 seconds of rest]) was reported to be less effective than high-intensity interval training (5–8×[2 minutes at ~100% $\dot{V}O_{2max}$: 1–3 minute of rest]) for improving $\beta m_{in\ vitro}$ in team-sport athletes, even when matched for total training volume.[27]

It is more difficult to recommend the ideal training programme to increase the MCTs as significant increases have been reported following both moderate-[81-83] and high-[76,84] intensity training. However, one factor that these training programmes tend to have in common is that they are associated with only modest post-training increases in blood lactate concentration (~4–8 mmol/L). When high-intensity training has been employed, the rest periods between high-intensity intervals have ranged from 90 to 240 seconds (e.g. a work-to-rest ratio of ≤1 : 2),[76,85] allowing substantial removal of lactate and H^+ prior to subsequent intervals.[79] Thus, in contrast to the high-intensity training required to increase $\beta m_{in\ vitro}$, it appears that both moderate- and high-intensity training can increase the MCTs, but that training should be structured so as to provoke only a modest increase in blood lactate concentration (~4–8 mmol/L). This might explain why, in the only study to date that has recruited well trained subjects, training at 60–70% $\dot{V}O_{2max}$ (post-exercise blood lactate concentration <1.5 mmol/L) was insufficient to maintain MCT content.[65] Significant changes in MCT content appear more likely when training is performed 2–3 times per week for 6–8 weeks. While no studies to our knowledge have investigated the influence of repeated-sprint training on changes in MCT content, intermittent-sprint training (15×[6-second sprint: 1-minute jogging recovery]) and interval training (8×[30 seconds at 130% $\dot{V}O_{2max}$: 90-seconds rest]) have been reported to be equally effective for increasing MCT1 content.[12] Further research is therefore required to determine the effects of repeated-sprint training on the muscle lactate transporters.

2.3 Muscle Activation

Sprinting requires considerable levels of neural activation.[86] Among the various potential neurally-mediated mechanisms determining RSA (in particular, sprint decrement), the ability to voluntarily fully activate the working musculature and to maintain muscle recruitment and rapid firing over sprint repetitions may critically affect fatigue resistance.[14,15,31,87] This suggests that under conditions of considerable fatigue development (e.g. sprint decrement score and fatigue index >25%) the failure to fully activate the contracting musculature may become an important factor limiting performance during RSE. Other factors, including disruption of optimal temporal sequencing of agonist and antagonist muscle activation (i.e. muscle coordination patterns) and/or motor unit recruitment strategies (e.g. decreased recruitment of fibres with faster conduction velocities), can also potentially limit RSA, as a multitude of different muscles must be activated at the appropriate times and intensities to maximize sprinting efficiency.[88,89]

A variety of training methods have been employed to successfully improve the degree of muscle activation (e.g. electromyostimulation, eccentric strength and plyometric training).[90] There is also evidence that such neural adaptations could enhance subsequent athletic performance.[91,92] While such research suggests that training which improves muscle activation has the potential to improve RSA, specific training studies are required before scientifically-based training recommendations can be given. This will not be easy as much of the fatigue experienced during RSE appears to be mediated by metabolic factors (see Part I of the preceding companion review[2]), and such research will need to demonstrate that training-induced improvements in RSA can be attributed to improvements in actual muscle activation, and not concurrent improvement in metabolic factors.

It has also been postulated that the ability for fast torque development depends, among other factors, on the specific ability for fast muscle activation at contraction onset (i.e. earlier recruitment of large motor units, increased synchrony, elevated motor unit firing rate[93]). Training-based studies that have reported corresponding increases in the rate of force rise and EMG development[94,95] support this viewpoint. Pending confirmatory research, these adaptations have the potential to improve rapid and forceful field/on-court movements such as sprinting involving muscle contraction times of less than 250 msec. It is therefore recommended that measurement of rate of force development and concomitant EMG activity (0–200 msec time frame) should be employed in future training studies (during standardized tests on a dynamometer or by exploring the early slope of vertical ground-reaction force-time curves during running-based RSE) to shed more light on neural adaptations to training targeting an improvement in sprint performance. Although it is tempting to propose that enhancing performance during initial sprint efforts may provide an effective strategy to improve mean sprint performance (e.g. total mechanical work), it also needs to be acknowledged that this is also likely to lead to a higher sprint decrement score.[31,32] Thus, additional training regimens may also need to be implemented to develop those fatigue-resistance factors.

3. Specific Training Strategies and Repeated-Sprint Ability

3.1 Repeated-Sprint Training

Anecdotally, repeated-sprint training is a popular training method used by team-sport athletes to improve RSA. However, despite the belief that such specific training will improve RSA more than generic training (e.g. interval training), very few studies have directly compared these two forms of training. We are aware of seven studies that have investigated adaptations to repeated-sprint training (table I). Only five of these studies incorporated a control training group, and only four of these studies recruited team-sport athletes. It is therefore difficult to make solid conclusions about the benefits of repeated-sprint training in comparison to other types of training. Nonetheless, despite the obvious need for further research, some tentative conclusions can be made.

Repeated-sprint training is able to improve $\dot{V}O_{2max}$. In the studies performed to date, 5–12 weeks of repeated-sprint training has been reported to result in a 5.0–6.1% increase in $\dot{V}O_{2max}$. Moreover, this increase is similar to that reported in the two studies, which incorporated a control group who performed interval training (5.2–6.6% increase in $\dot{V}O_{2max}$).[4,27] However, as other studies utilizing different types of interval training have reported more than 10% increases in $\dot{V}O_{2max}$,[10,101] further research, comparing repeated-sprint training and these other types of training, is required to verify the best means to improve $\dot{V}O_{2max}$ in team-sport athletes. Further research is also required to investigate additional physiological adaptations to repeated-sprint training (e.g. changes in ion regulation, anaerobic capacity, phosphocreatine resynthesis, etc). For example, the limited evidence to date suggests that, compared with repeated- or intermittent-sprint training, interval training produces superior increases in both $\beta m_{in\ vitro}$[27] and Na^+/K^+ pump isoform content.[12]

With respect to RSA, repeated-sprint training has been reported to produce greater improvements in best sprint time[12,27,96] and mean sprint time,[4,12,27,96] compared with interval-based training. In contrast, interval training appears to be superior to repeated-sprint training to decrease (i.e. improve) the sprint decrement score (or the fatigue index).[12,27] However, due to the problems associated with interpreting changes in the sprint decrement score when there are concurrent changes in best sprint time,[102] it is difficult to make universal recommendations. For example, Mohr et al.[12,103] have suggested that the greater improvement in sprint decrement following interval training (termed 'speed-endurance' training [SET] by the authors), when compared with intermittent-sprint training (ST) [figure 3], is a sign that interval training is superior for improving RSA. However, this interpretation has been questioned[34] as a closer analysis of their data suggests that the intermittent-sprint-training group had a greater improvement in single-sprint performance

Table I. A summary of the characteristics and results of training studies that have investigated changes in repeated-sprint ability following running-based training

Study (y)	Subjects		Training programme	Adaptations			
	type	$\dot{V}O_{2max}$ (mL/kg/min)[a]		best sprint (%)	mean sprint (%)	DS (%)	$\dot{V}O_{2max}$ (%)
Buchheit et al.[9] (2008)	9, MA, M, TS	NR	2×([5–6×30–40 m shuttle sprints: 14–23 sec]: 2 min rest); 2 d/wk, 9 wk	↑ 0.3 NS	↑ 1.0 NS	↑ 19 NS	NR
	8, MA, M, TS		9–24×(15–20 sec at 105–115% $\dot{V}O_{2max}$: 15–20 sec); 2 d/wk, 9 wk	↑ 1.4*	↑ 1.5*	↑ 44 NS	
Buchheit et al.[96] (2010)	7, MA, M, TS	NR	3–4×([4–6×accelerations/sprints (<5 sec): 30 sec]: 3 min rest); 2 d/wk, 4 wk	↑ 2.7	↑ 22	↑ 35	NR
	7, MA, M, TS		3–5×(30 sec all-out shuttle sprints: 4 min rest), 2 d/wk, 4 wk (both groups also performed two other team training sessions)	↑ 0.7	↑ 0.8	↑ 39	
Dawson et al.[43] (1998)	9, MA, M	57.0±2.4	4–6×([5×30 to 80 m sprints: 30–90 sec rest]: 2–4 min rest); 3 d/wk, 6 wk	↑ 2.4*	↑ 2.2*	↑ 16 NS	↑ 6.1*
Bravo et al.[4] (2008)	21, MA, M, TS	55.7±2.3	3×([6×40 m sprint: 20 sec rest]: 4 min rest); 2 d/wk, 12 wk	NR	↑ 2.1*	NR	↑ 5.0*
	21, MA, M, TS	52.8±3.2	4×(4 min at 95% HR_{max}: 3 min at 75% HR_{max}); 2 d/wk, 12 wk (both groups also performed two other team training sessions)		↑ 0.3 NS		↑ 6.6*
Mohr et al.[12] (2007)	6, MA, M	50.2±3.7	15×(6 sec sprint:1 min jog recovery); 3–5 d/wk, 8 wk	↑ 4.0*	↑ 4.3*	↑ 13 NS	NR
	7, MA, M	49.0±4.2	8×(30 sec at 130% max: 90 sec rest); 3–5 d/wk, 8 wk	↑ 0.7 NS	↑ 2.4*	↑ 54*	
Schneiker and Bishop[27] (2008)	7, MA, M TS	56.2±6.8	5–8×(5×25 to 35 m sprints: 21 sec rest); 3 d/wk, 5 wk	↑ 1.3*	↑ 1.6*	↑ 12 NS	↑ 5.1*
	7, MA, M, TS	56.6±5.3	5–8×(2 min at 110% $\dot{V}O_{2max}$: 2 min rest); 3 d/wk, 5 wk	↓ 0.5 NS	↑ 0.6 NS	↑ 26*	↑ 5.2*
Serpiello et al.[97] (2011)	10, M, M, F	53.7±6.9	3×([5×4 sec sprint: 16 sec rest]: 4.5 min rest); 3 d/wk, 4 wk (training/tests performed on a non-motorized treadmill)	↑ 5.5*	↑ 8.8*	NR	↑ 2.0
Walklate et al.[98] (2009)	6, MA, M, TS	NR	Control (squad training)	↑ 0.6 NS	↑ 1.4 NS	↑ 2 NS	NR
	6, MA, M, TS		Squad training+7–15×(20 sec sprint: 10 sec rest); 2 d/wk, 4 wk	↓ 0.2 NS	↑ 5.0 NS	↑ 8 NS	
Buchheit et al.[99] (2009)	15, MA, M, TS	NR	Small-sided games (2–4×2.5–4 min games)	↑ 3.7*	↑ 4.6*	↑ 23 NS	NR
	17, MA, M, TS		12–24×(15 sec at 105–115% $\dot{V}O_{2max}$: 15–20 sec); 2 d/wk, 10 wk	↑ 3.5*	↑ 3.4*	↑ 3 NS	
Hill-Haas et al.[100] (2009)	10, MA, M, TS	59.3±4.5	Small-sided games 2–6×(6–13 min games:1–3 min of rest)	↑ 0.6 NS	↑ 0.2 NS	↓ 5 NS	↓ 0.7 NS
	9, MA, M, TS	60.2±4.6	Generic training (see review for more details)	↑ 1.5 NS	↓ 0.2 NS	↓ 23 NS	↑ 2.0 NS

a Data presented as mean±SD unless NR.

DS=decrement score (or fatigue index); **F**=Females; **HR_{max}**=maximal heart rate; **M**=Males; **MA**=moderate aerobic fitness; **max**=maximum; **NR**=not reported; **NS**=not significant; **TS**=team-sport athletes; **$\dot{V}O_{2max}$**=maximal oxygen consumption; * indicates significant difference between pre and post ($p<0.05$); ↑ indicates improved; ↓ indicates worsened.

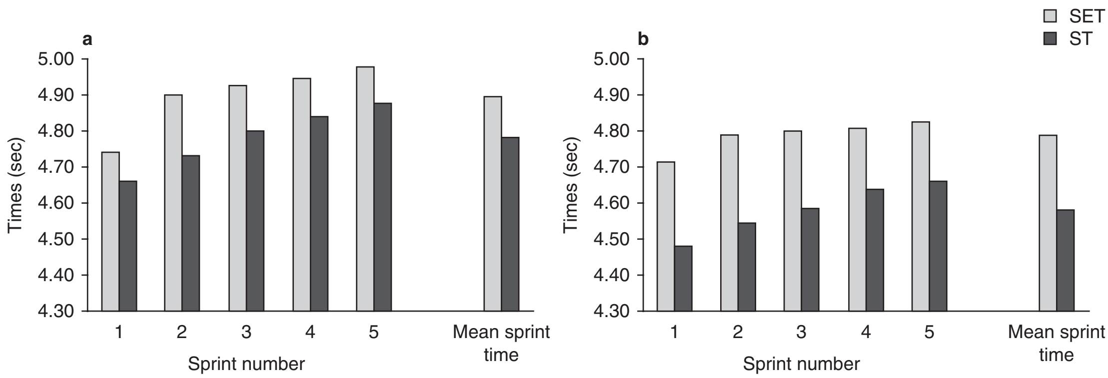

Fig. 3. (**a**) Pre-training and (**b**) post-training individual and mean sprint times derived from a repeated-sprint test consisting of five 30 m sprints, separated by 25 sec periods of active recovery during which the subjects jogged back to the starting line. ST performed intermittent-sprint training, while SET performed 'speed-endurance' training (a type of interval training). [See table II for more details of the training performed]. Post-training there was a significant decrease in initial sprint time for ST only, but a significant decrease in mean sprint time for both groups.[12]

(including the final sprint; 4.5 vs 3.2%) and mean sprint time (4.3% vs 2.4%) [figure 3]. Furthermore, the smaller improvement in the fatigue index by the intermittent-sprint training group is likely to be related to their much-improved first sprint. Thus, it appears that while interval training may be superior at minimizing the decrement during repeated sprints (possibly due to greater physiological adaptations, as outlined in section 2), intermittent- or repeated-sprint training is superior at improving the performance of individual sprints. As a result, the combination of the two (i.e. repeated-sprint training to improve sprint performance plus interval training to improve the recovery between sprints) may be the best strategy to improve RSA. Further research is required to investigate the optimal volume and duration of a repeated-sprint training macro cycle, as anecdotal evidence suggests that too much repeated-sprint training is stressful and may lead to decreases in RSA.

3.2 Sprint Training

Given the improvements in individual sprint times following repeated- and intermittent-sprint training (see table II), a logical question is whether or not similar (or greater) improvements in individual and mean sprint times can be achieved by traditional sprint training (i.e. short sprints interspersed with complete recovery periods[107]). To date, we are unaware of research that has investigated the influence of 'traditional' sprint training on RSA. However, it is possible that such training may produce even better improvements in both best sprint time and mean sprint time,[86] and further research is warranted. In support of this, a targeted sprint/agility training protocol (incorporating incomplete rest periods) improved mean sprint time by 2.2% in a group of young soccer players. These changes in mean sprint time were associated with concurrent improvements in single-sprint performance (approximately 2.7% reduction in 10 m sprint time), while no changes in aerobic fitness were observed.[96] Despite the obvious need of more research in this area, these results seem to confirm that in well trained team-sport athletes, maximization of mean repeated-sprint time is linked to improvements in single-sprint performance.[33]

3.3 Small-Sided Games

Recently, there has been an increased emphasis on the use of small-sided games to improve both team-sport-related fitness (e.g. $\dot{V}O_{2max}$, intermittent exercise capacity) and technical skills.[108,109] To date, however, only two studies (table I) have investigated the effects of small-sided-games training on RSA, and both have reported only small, nonsignificant differences in terms of RSE performance enhancement when compared with generic training.[99,100] For example, when training twice per week for 10 weeks, a similar ~4% improvement in best and mean sprint time has been reported following both small-sided games (2–4×[2.5–4-minute 'games']) and interval training (12–24×[15 seconds at ~105–115% $\dot{V}O_{2max}$: 15 seconds of rest]).[99] As the small-sided game protocols employed in these studies targeted the development of aerobic fitness, it is likely that the mechanisms responsible for the reported improvements in RSA are also related to improvements in aerobic fitness. In addition, factors other than aerobic fitness, such as neuromuscular factors (e.g. acceleration and turning) that can also be developed with the use of small-sided games, might also explain the observed improvements in RSA.[99,100] However, given the limited research to date, further research is obviously required, especially research comparing the use of small-sided games with other types of training that have previously been demonstrated to improve RSA. Additional research is also required to determine whether small-sided games can be used to improve other factors such as H^+ regulation and phosphocreatine resynthesis.

3.4 Resistance Training

While there is good evidence to suggest that resistance training could be beneficial for single-sprint performance,[110-112] the impact of such training on RSA is less clear (table III). To date, three studies have reported that resistance training (2–5 sets of 10–15 maximal repetitions) produces similar increases in mean work during a repeated-sprint test (~12%)[113-115] compared with high-intensity interval training (~13%)[10] or sprint training (~12%).[105] Resistance training also improved both first-sprint performance (8–9%) and the sprint decrement score (~20%).[113,114] The increases in RSA reported in these studies are likely to be accounted for, at least in part, by strength gains. However, factors other than improvements in maximal strength may also be involved as we have observed greater improvements in RSA when sets of resistance training were separated by 20 seconds, compared with 80 seconds of rest, despite half the increase in maximal leg strength (20 vs 46%).[114] This suggests that resistance training that includes a high metabolic load (e.g. blood lactate concentration

Table II. A summary of the characteristics and results of training studies that have investigated changes in cycle repeated-sprint ability (RSA) following different types of training performed on a cycle ergometer

Study (y)	Subjects		Training programme	Adaptations			
	type	$\dot{V}O_{2max}$ (mL/kg/min)[a]		sprint 1 (%) [W]	total work (%) [kJ]	DS (%)	$\dot{V}O_{2max}$ (%)
Edge et al.[10] (2005)	10, MA, F	42.4±6.3	6–10×(2 min at 120–140% LT: 1 min rest); 3 d/wk, 5 wk	↑ 6.2* ↑ 6.9*	↑ 13.0*,† ↑ 8.5*	↑ 10 NS ↓ 16 NS	↑ 13.2* ↑ 10.4*
	10, MA, F	41.3±7.3	20–30 min at 80–95% LT; 3 d/wk, 5 wk				
Bishop and Edge[104] (2005)	11, MA, F	39.0±6.4	3–12×(2 min at 130–180% LT: 1 min rest); 3 d/wk + RSA test (5×6 sec sprint every 30 sec); 1 d/wk, 8 wk	↑ 21.2*	↑ 28.3*	↓ 14 NS	↑ 14.6*
Glaister et al.[11] (2007)	12, MA, M, TS	46.6±4.2	20 min at 70% $\dot{V}O_{2max}$; 3 d/wk, 6 wk	↑ 4.0*	↑ 9.4*	↑ 46*	↑ 9.9*
	9, MA, M, TS	52.1±3.6	Control (normal recreational activities)	–	↑ 1.4 NS	↑ 10 NS	–
Ortenblad et al.[105] (2000)	9, MA, M	61.3±1.7	20×(10 sec sprint: 50 sec rest); 3 d/wk, 5 wk	↑ 6.6* –	↑ 12* ↑ 1.0 NS	↑ 27* –	– –
	6, MA, M	64.0±0.5	Control (normal recreational activities)				

a Data presented as mean±SD.

DS=decrement score (or fatigue index); **F**=females; **LT**=lactate threshold (as determined using the modified Dmax method[106]); **M**=males; **MA**=moderate aerobic fitness; **NR**=not reported; **NS**=not significant; **TS**=team-sport athletes; **$\dot{V}O_{2max}$**=maximal oxygen consumption; * indicates significant difference between pre and post ($p<0.05$); † indicates significantly greater improvement than the alternate training group; ↑ indicates improved; ↓ indicates worsened; – indicates no change.

Table III. A summary of the characteristics and results of training studies that have investigated changes in cycle repeated-sprint ability (RSA) following different types of resistance training

Study (y)	Subjects		Training programme	Adaptations			
	type	$\dot{V}O_{2max}$ (mL/kg/min)[a]		sprint 1 (%) [kJ]	total work (%) [kJ]	DS (%)	$\dot{V}O_{2max}$ (%)
Edge et al.[113] (2006)	8, MA, F	42.4 ± 9.6	Control	↑ 2.7 NS	↑ 3.0 NS	↑ 3 NS	–
	8, MA, F	44.8 ± 5.5	6 leg exercises for 2–5 sets × (15–20 RM: 20 sec rest); 3 d/wk, 5 wk	↑ 8.0 NS	↑ 12.0*,†	↑ 22*,†	–
Hill-Haas et al.[114] (2007)	8, MA, F	42.4 ± 9.6	6 leg exercises for 2–5 sets × (15–20 RM: 80 sec rest); 3 d/wk, 5 wk	↑ 9.3*	↑ 5.4*	↑ 21*	–
	8, MA, F	44.8 ± 5.5	6 leg exercises for 2–5 sets × (15–20 RM: 20 sec rest); 3 d/wk, 5 wk	↑ 8.4*	↑ 12.5*,†	↑ 23*	–
Robinson et al.[115] (1995)	8, MA, M	–	2 leg exercises for 5 sets × (10 RM: 30–180 sec rest); 4 d/wk, 5 wk	↑ 6.6*	↑ 8.5*	–	–

a Data presented as mean ± SD unless no change.

DS = decrement score (or fatigue index); **F** = females; **M** = males; **MA** = moderate aerobic fitness; **NS** = not significant; $\dot{V}O_{2max}$ = maximal oxygen consumption; * indicates significant difference between pre and post ($p < 0.05$); † indicates significantly greater improvement than the alternate training group; ↑ indicates improved; ↓ indicates worsened; – indicates no change.

≥10 mmol/L), rather than resistance training which maximizes strength gains (e.g. using 1–4 maximal repetitions), may best improve RSA (possibly via greater improvements in H^+ regulation[113]). Further research is required though as the subjects involved in these studies were only moderately trained. Given that success in repeated-sprint activities is also likely to depend on an athlete's explosive power, further research is also required to investigate the importance of explosive muscle strength training on RSA.

4. Conclusions

RSA is an important fitness component of many popular team sports. This review has highlighted that there is not one type of training that can be recommended to best improve RSA and all of the factors believed to be responsible for performance decrements during repeated-sprint tasks. This is not surprising, as RSA is a complex fitness component that depends on both metabolic (e.g. oxidative capacity, phosphocreatine recovery and H^+ buffering) and neural factors (e.g. muscle activation and recruitment strategies) among others (figure 4). While different training strategies can be used in order to improve each of these potential limiting factors, and in turn RSA, the concurrent implementation of different forms of training may be the best strategy to improve RSA. However, the currently unknown synergies and interferences resulting from the combination of various training contents[116] on the metabolic, neural and mechanical determinants of RSA make guidelines on how training content should be manipulated and periodized difficult. Nonetheless, two key recommendations can be made based on the existing literature as follows:

1. It is important to include some training to improve single-sprint performance. This should include (i) specific sprint training; (ii) strength/power training; and (iii) occasional high-intensity (>$\dot{V}O_{2max}$) training (e.g. repeated, 30-second, all-out efforts separated by ~10 minutes of recovery) to increase the anaerobic capacity.
2. It is also important to include some interval training to best improve the ability to recover between sprints (if the goal is to improve fatigue resistance). High-intensity (80–90% $\dot{V}O_{2max}$) interval training, interspersed with rest periods (e.g. 1 minute) that are shorter than the work periods (e.g. 2 minutes) is efficient at improving the ability to recover between sprints by increasing aerobic fitness ($\dot{V}O_{2max}$ and the lactate threshold), the rate of phosphocreatine resynthesis and $\beta m_{in\ vitro}$.

In support of the above recommendation, to date, the greatest improvements in both single and mean sprint performance have been reported

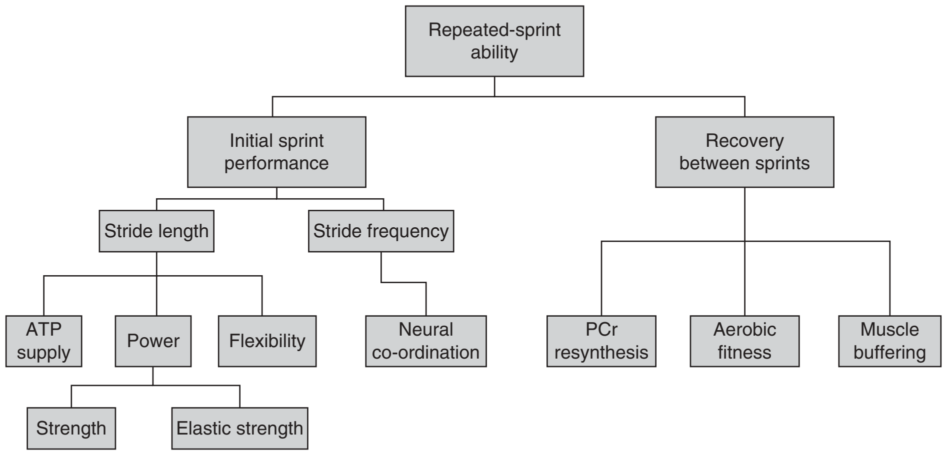

Fig. 4. A summary of factors which should be targeted by training to improve repeated-sprint ability. **ATP** = adenosine triphosphate; **PCr** = phosphocreatine.

after training that included both high-intensity interval training and repeated sprints.[104]

For most athletes, it is probably impossible to perform all of the above-described training concurrently. It is therefore paramount that a periodized training programme, designed to improve RSA, is structured such that different aspects are emphasized, at different times, in accordance with the competitive demands of each particular sport and the strengths and weaknesses of the individual athlete. As RSA requires a unique blend of power (sprint speed) and endurance (recovery between sprints), it needs to be established whether it is best to develop these qualities separately, or whether they can be developed concurrently (without interference effects). Future studies also need to address whether training-induced changes in RSA actually impact upon field performance. More importantly, as many studies to date have used untrained subjects and/or a cycle ergometer, future research must recruit highly-trained team-sport athletes and be expanded to sport-specific test settings with, in parallel, a high level of standardization and reliability of the measures.

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