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REVIEW ARTICLE

Implications of Impaired Endurance Performance following Single Bouts of Resistance Training: An Alternate Concurrent Training Perspective

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Abstract A single bout of resistance training induces residual fatigue, which may impair performance during subsequent endurance training if inadequate recovery is allowed. From a concurrent training standpoint, such carry-over effects of fatigue from a resistance training session may impair the quality of a subsequent endurance training session for several hours to days with inadequate recovery. The proposed mechanisms of this phenomenon include: (1) impaired neural recruitment patterns; (2) reduced movement efficiency due to alteration in kinematics during endurance exercise and increased energy expenditure; (3) increased muscle soreness; and (4) reduced muscle glycogen. If endurance training quality is consistently compromised during the course of a specific concurrent training program, optimal endurance development may be limited. Whilst the link between acute responses of training and subsequent training adaptation has not been fully established, there is some evidence suggesting that cumulative effects of fatigue may contribute to limiting optimal endurance development. Thus, the current review will (1) explore cross-sectional studies that have reported impaired endurance performance following a single, or multiple bouts, of resistance training; (2) identify the potential impact of fatigue on chronic endurance development; (3) describe the implications of fatigue on the quality of endurance training sessions during concurrent training, and (4) explain the mechanisms contributing to resistance training-induced attenuation on endurance performance from neurological, biomechanical and metabolic standpoints. Increasing the awareness of resistance training-induced fatigue may encourage coaches to consider modulating concurrent training variables (e.g., order of training mode, between-mode recovery period, training intensity, etc.) to limit the carry-over effects of fatigue from resistance to endurance training sessions.

Key Points

Several studies have reported that a single bout of resistance training impairs subsequent endurance performance as a result of fatigue.

Resistance training-induced fatigue may impair the quality of endurance training sessions during concurrent training, thereby limiting optimisation of endurance development.

Coaches/athletes should be aware of fatigue when undertaking concurrent training to optimise endurance performance development, particularly fatigue induced by resistance training.

The original version of this article was revised: The title of the article in the Online version is incorrect. This Error has been corrected.

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1 Introduction

Incorporating exercises that induce adaptations for resistance and endurance in the one training regime irrespective of whether the training is performed on the same day or on separate days is known as concurrent training [1]. Current

literature advises health professionals to prescribe concurrent training in order to improve and sustain health-related fitness [2]. The majority of concurrent training studies to date have shown that resistance training combined with endurance training enhances endurance adaptation to a greater extent than endurance training alone [3]. Common mechanisms that have been proposed to explain this phenomenon include alterations in muscle fibre-type recruitment pattern, greater muscle force generation capacity, increased proportion of type IIA fibres and reduced proportion of type IIB fibres, and a shift toward a fatigue-resistance yet more powerful muscle phenotype, all of which can improve movement economy [4, 5].

In light of the above, resistance training is highly recommendable for endurance athletes aiming to optimise their performance. However, concurrent training prescription should be undertaken with care given that fatigue induced by resistance training may impair the quality of endurance training sessions, and, possibly, induce sub-optimal endurance development (Fig. 1). In fact, cross-sectional studies have reported attenuated repeated sprint ability [6], running time-to-exhaustion [7], arm cranking time-to-exhaustion [8] and cycling time-trial performance [9] several days following a single bout of resistance training. Whilst this phenomenon would only be observed during resistance training-induced fatigue, if training variables and adequate recovery are not appropriately accounted for, athletes may become subject to non-functional over-reaching or overtraining, which have been reported to impair chronic running [10] and cycling [11] time-trial improvements. Furthermore, whilst overloaded training is used to induce a super-compensation cycle effect [12], studies have shown no greater improvement in running and cycling time-trial performance with several weeks of overloaded training compared to normal-loaded training following tapering [10, 13], suggesting that excessive fatigue in some cases may cause unnecessary physiological stress resulting in no further benefits than normal-loaded training or increased susceptibility to injuries.

To conceptualise the possibilities of concurrent training inducing sub-optimal endurance development, the current review will (1) explore cross-sectional studies that have reported impaired endurance performance following a single, or multiple, bouts, of resistance training, hereafter referred to as resistance training-induced sub-optimisation of endurance performance (RT-SEP); (2) describe the implications of fatigue on the quality of endurance training sessions during concurrent training; (3) identify the potential impact of fatigue on chronic endurance development; and (4) explain the mechanisms contributing to RT-SEP from neurological, biomechanical and metabolic standpoints. Although training variables (i.e. intensity/volume, exercise sequence, recovery period, number of training weeks, etc.) and recovery strategies may affect the type and magnitude of resistance training-induced fatigue, an in-depth exploration of such complex factors in addition to mechanisms in a single review is beyond the scope of this review.

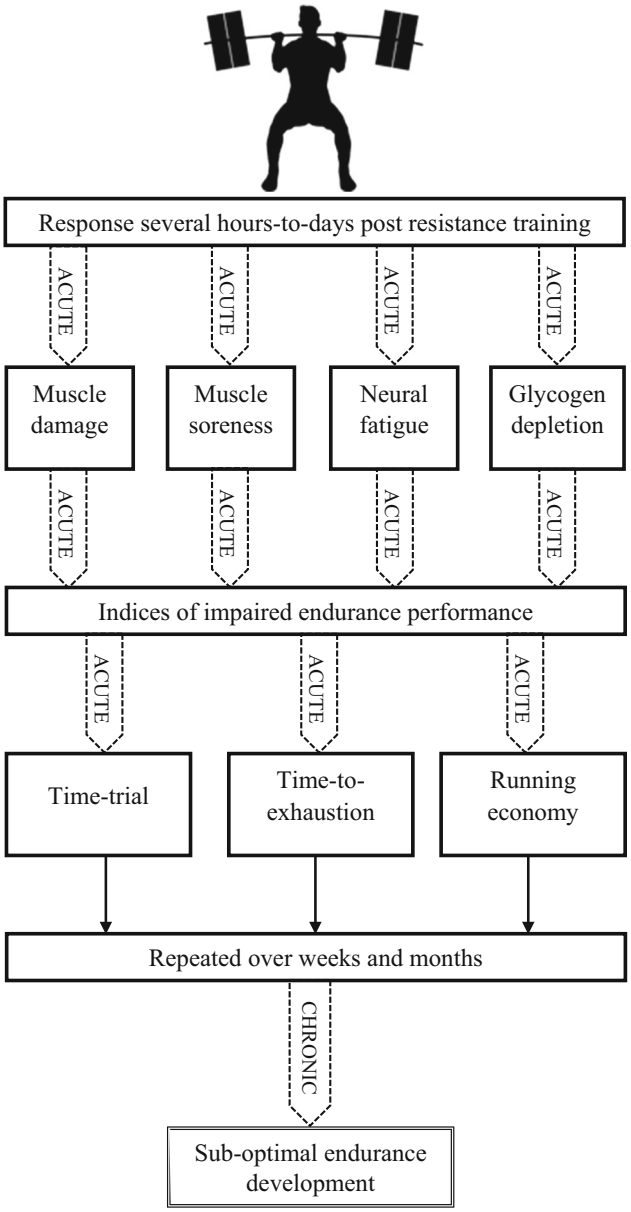

Fig. 1 The conceptual framework of resistance training-induced sub-optimisation of endurance performance and its implications for chronic endurance development

2 Acute Effects of Resistance Exercise on Indices of Endurance Performance and Their Implications

Prior to discussing the mechanisms of RT-SEP, the implications of this phenomenon should be elucidated from a concurrent training standpoint. To date, studies have

reported that a single resistance training bout impairs subsequent time-trial and time-to-exhaustion performance of various exercise modes [8, 9, 14–18] for 24–72 h post-training. For example, Doncaster and colleagues [8] examined the acute effects of bench press exercise on arm cranking endurance performance. The results showed that ten sets of six repetitions of bench press exercises at 70% of one repetition maximum (RM) reduced arm cranking time-to-exhaustion for up to 48 h post-exercise. Similarly, Doma and colleagues [14] reported reduced running time-to-exhaustion at 110% of anaerobic threshold for up to 24 h following a bout of lower body resistance training consisting of leg press, leg extension and leg curls at six repetition maximum. Furthermore, several studies have reported impaired running economy (RE) following a single resistance training bout for several days post-training [7, 19, 20]. While RE is not a measure of endurance performance per se, and compromised RE may not likewise compromise the development of endurance performance, there are observations suggesting reduced RE in a laboratory-based condition may be indicative of impaired running time-trial performance in a non-laboratory-based condition. For instance, Hoogkamer et al. [21] showed that shoes with added mass impaired RE and concomitantly slowed 3000-m running time trial performance and demonstrated that laboratory-based RE measures can accurately predict alterations in road-based running performance. Thus, impaired RE may indicate that the quality of actual running sessions could be compromised [22]. For example, attenuation of the physiological cost of aerobic exercise tends to suggest that athletes would experience difficulty covering particular distances or maintaining intensity (e.g. pacing) to meet session goals as a result of residual fatigue from previous training sessions. This implication is also attributable to studies that have shown impaired endurance time-trial performances following a single bout of resistance training [8, 14, 16–18]. Studies have also shown that resistance training-induced fatigue increases heart rate and RPE during RE tests [20, 23]. Therefore, if athletes are prescribed to train at an intensity relative to their maximum heart rate or RPE, they may have to reduce their training pace during periods of resistance training-induced fatigue, which may defeat the purpose of the training session.

3 Effects of Fatigue on Chronic Endurance Development

A number of studies have attempted to link acute fatigue responses to training sessions, then to subsequent endurance development. For example, Lamberts et al. [24] retrospectively examined the relationship between changes in heart rate recovery and cycling performance in trained cyclists. Following 4 weeks of high intensity endurance training, the results showed that the level of improvement was greater for those that demonstrated better heart rate recovery, with authors suggesting that training-induced fatigue may have impacted on the level of training adaptation. Furthermore, Halson et al. [13] investigated the effects of cumulative exercise stress and subsequent recovery on performance changes and fatigue indicators in trained cyclists during a 6-week training period. The cyclists completed normal training, intensified training and recovery training for 2 weeks, respectively. The results showed that maximal cycling power output and cycling time-trial performance was worsened following the intensified training period, despite the provision of recovery training. These findings suggest that athletes are unable to perform at optimum levels as a result of cumulative effects of fatigue from intensified training. Whilst speculative, if intensified endurance training alone induces fatigue sufficient to affect endurance development, then combining resistance with endurance training may induce similar to or further levels of cumulative effects of fatigue if proper recovery and/or training variables are not accounted for. This is because the physiological stress caused by a typical resistance training bout (i.e., 40–60 min) can continue for several days post-exercise [15, 19] as opposed to a full recovery observed within 24 h following a typical endurance training bout (i.e. 40–60 min) [14, 25, 26]. However, further studies are warranted to confirm whether endurance development is impacted specifically from over-reaching or overtraining induced by concurrent training.

Whilst full restoration of homeostasis is not required between training sessions [27], general consensus suggests that recovery and psychophysiological 'readiness' for each training session may impact on the overall magnitude of training adaptation [28]. In fact, the accumulation of specific transcriptional and translational alterations that occur from one session to the next may dictate the course of training adaptation [29]. Thus, the benefits of resistance training on endurance development could be further optimised by limiting the carry-over effects of fatigue from resistance to endurance training sessions by modulating the concurrent training variables (e.g. order of training mode, between-mode recovery period, training intensity, etc.). For example, Chtara et al. [30] reported greater improvement in 4-km running time-trial performance following 12 weeks of concurrent training for the group that undertook endurance training sessions prior to resistance training compared to the group that carried out each mode of training in the reverse order. Izquierdo-Gabarren and colleagues [31] reported improved 20-min rowing time-trial performance following 8 weeks of concurrent training compared to the control group that solely undertook endurance training. However, when concurrent training

Table 1 A summary of concurrent training studies demonstrating between-group differences in endurance performance outcomes

Authors	Training groups	Participant background	Training duration	Training volume	Outcome measure	Improvement
Chtara et al. [30] (PMID: 16046343)	E + S ($n = 10$)	Healthy male moderately active individuals	12 weeks	$4 \cdot wk^{-1}$	4-km running time trial	↑8.6%*
	S + E ($n = 10$)			$4 \cdot wk^{-1}$		↑4.7%
Izquierdo et al. [31] (PMID: 19997025)	NRF ($n = 15$)	Trained male rowers	8 weeks	$8 \cdot wk^{-1}$	20-min rowing time trial	↑9.0%*
	RF ($n = 14$)					↑4.6%
Robineau et al. [32] (PMID: 25546450)	C-24 h ($n = 12$)	Amateur male rugby players	7 weeks	$4 \cdot wk^{-1}$	Running VO_{2peak}	↑8.0% ≠
	C-0 h ($n = 15$)					↑3.7%
Gravelle et al. [131]	LR ($n = 6$)	Healthy active women	11 weeks	$3 \cdot wk^{-1}$	Rowing VO_{2max}	↑7.5%*
	RL ($n = 7$)					↑5.1
Gravelle et al. [131]	LR ($n = 6$)	Healthy active women	11 weeks	$3 \cdot wk^{-1}$	30-s Wingate performance	↑10.2%*
	RL ($n = 7$)					↑0.3%
Schumann et al. [132]	AD ($n = 18$)	Healthy moderately active men and women	24 weeks	$4 \cdot wk^{-1}$	Sub-maximal cycling VO_2	↑3% ≠
	E + S ($n = 15$)			$2 \cdot wk^{-1}$		↓2%

E + S endurance training following by strength training on the same day, *S + E* strength training followed by endurance training on the same day, *NRF* no repetiton to failure, where participants undertook resistance exercises without reaching failure for each set, *RF* repetition to failure, where participants undertook resistance exercises and reaching failure for each set, C-24 h participants receiving 24 h of rest between resistance and endurance training sessions; 0–24 participants undertaking resistance training followed by endurance training with no rest in-between, *LR* lifting weights (i.e. resistance training) followed by rowing on the same day, *RL* rowing followed by lifting weights on the same day, *AD* participants undertaking resistance and endurance training on alternating days

* Significantly greater than pre ($p < 0.05$)

≠ Significantly greater than the other group ($p < 0.05$)

groups were compared between those that performed resistance training sets to failure to those without failure, a lesser improvement in rowing performance measures was observed for those that performed resistance training sets to failure. Robineau et al. [32] recently showed that 7 weeks of concurrent training induced greater improvement in running peak oxygen consumption (VO_{2peak}) for rugby players who had 24 h of recovery between resistance and endurance training sessions compared to groups that had 0–6 h of rest. Whilst VO_{2peak} does not necessarily equate to endurance performance measures [33], alterations in aerobic capacity tend to suggest that concurrent training methods appear to influence the level of endurance training adaptation. Collectively, these findings [30–32] suggest that training methods that minimise residual fatigue between each mode of training (e.g. performing endurance training prior to resistance training or extending periods of between-mode recovery) may further increase the benefits of resistance training on endurance development but by no means indicate that resistance training impairs endurance development (refer to Table 1). This concept is also attributed to cross-sectional studies that have examined various recovery strategies following a single bout of resistance training with the underlying conjecture that insufficient recovery from a strenuous training session may leave individuals with the inability to train at required levels during subsequent training sessions [34–36].

Interestingly, Halson and colleagues [37] reported greater improvement in a 4-min cycling time-trial for cyclists that received cold water immersion (CWI) interventions post 11 days of tapering after 21 days of intensified training compared to cyclists without a recovery intervention. Whilst Halson and colleagues [37] did not systematically examine the acute effects of CWI on cycling performance, a recent cross-sectional study reported a greater increase in cycling power output measures for a group that received CWI following a single bout of lower body resistance training compared to a group that received cold air therapy treatment [36]. Collectively, accelerating the recovery process and minimising fatigue between strenuous training sessions may enhance an athlete's 'readiness' for a subsequent training session and optimise training adaptation [28]. Consequently, the potential mechanisms of this phenomenon should be discussed to increase the awareness for and understanding of the implications of RT-SEP.

4 Consequences of Neuromuscular Fatigue

4.1 Neuromuscular Fatigue in Response to Resistance Training

Several studies have reported that a single bout of resistance training impairs muscle force generation capacity

(MFGC) for up to 72 h post-exercise in resistance-trained individuals [7, 38–41]. For example, Michaut et al. [41] examined the effects of five sets of ten maximal eccentric contractions of the biceps brachii. Twitch activation and maximal contraction was significantly reduced from 2 min to 48 h post-exercise. In addition, Stock et al. [38] reported impaired squatting performance from 24 to 72 h following six sets of squatting exercises at 75% of 1RM amongst resistance trained individuals. With repetitive maximal and submaximal contractions, the neural drive declines and the motor neurons become less responsive to synaptic stimulation [42]. This decrement of neural function in response to exercise can originate from peripheral and/or central sites of the nervous system. From a concurrent training standpoint, the attenuation of muscular function due to acute carry-over effects of neuromuscular fatigue between resistance and endurance training sessions may interfere with the training stimuli to optimise endurance development. In fact, the neuromuscular response following long distance running and cycling events has been studied extensively with results demonstrating reductions in maximal voluntary contraction (MVC) due to central and/or peripheral fatigue [43–45]. Romer et al. [46] reported that cycling time to exhaustion was less in hypoxia in comparison to normoxia with a concommitant reduction in potentiated twitch force. These findings indicate that cessation of endurance exercise appears to occur as a result of peripheral fatigue. However, it has also been suggested that termination of exercise occurs due to central fatigue as a protective mechanism from potentially harmful metabolic disturbances to preserve muscular function [47]. In light of this hypothesis, Ansley et al. [48] showed that power output and integrated EMG increased comparatively at the end of a 4-km cycling time trial exercise, indicating that fatigue was centrally controlled. Furthermore, Decorte et al. [49] reported that cycling exercise to failure was associated with central rather than peripheral fatigue. Subsequently, how the mechanisms of neuromuscular fatigue contribute to premature cessation of endurance exercises remains unclear. However, given that strength training has been shown to reduce MVC for several days post-exercise [38, 40, 41], neuromuscular fatigue generated by a strength training session may compromise the quality of a subsequent endurance training session.

Whilst resistance training clearly impairs MFGC, it should be noted that the degree of resistance training-induced changes in neuromuscular performance could be influenced by resistance training variables and training background. For example, Paschalis et al. [50] reported significantly greater decrements in muscle performance for up to 72 h post-high intensity eccentric contractions compared to low intensity eccentric contractions when equated for training volume. However, both conditions impaired MFGC and there were similar effects on muscle damage. Similarly, Newton and colleagues [51] showed that eccentric contractions impaired MFGC to a greater extent in resistance-untrained individuals compared to their trained counterparts. These findings demonstrate that resistance training variables and training background impact on the magnitude of resistance training-induced fatigue following a single bout of resistance exercise. Nonetheless, MFGC is still impaired for several days following high intensity resistance training in resistance-trained individuals [50, 51]. Subsequently, irrespective of training variables and training background, prolonged neuromuscular fatigue generated by a bout resistance training may compromise the quality of a subsequent endurance training session.

4.2 Impact of Neuromuscular Fatigue on Running Gait Patterns

The effects of resistance training-induced fatigue have been reported to alter neural recruitment patterns during the performance of various exercises. Plattner et al. [52] recently showed that muscle activity of the biceps brachii decreased during maximal contractions although increased during sub-maximal contractions in response to resistance training-induced fatigue, indicating alterations in neural firing patterns and motor unit activity. Furthermore, eccentric contractions have been demonstrated to impair proprioceptive mechanisms by increasing the perception of force generation in comparison to the actual force recorded [53, 54]. Given that neuromuscular control is imperative for human locomotion [55], neuromuscular dysfunction in response to resistance training could alter kinematics of endurance performance, impair movement efficiency and decrease the economy of movement [56].

There is a growing body of evidence showing alterations in lower extremity running kinematics 24–48 h following resistive-type exercises, such as downhill running [23, 57–59] and lower extremity eccentric contractions [60, 61]. The majority of these studies have shown significant reductions in lower extremity joint range of motion during running [23, 57, 59, 61] with suggestions that neuromuscular fatigue, delayed-onset of muscle soreness (DOMS) and exercise-induced muscle damage (EIMD) may have contributed to such kinematic changes. Despite this consensus, however, only two studies have investigated the acute effects of traditional resistance training on running gait patterns. For example, Doma et al. reported reduction in hip range of motion in the saggital plane during sub-maximal running 6 h [7] and 24 h [14] following three sets of six repetitions of leg press, leg extension and leg curls at 6RM (Fig. 1). Interestingly, these findings are similar to those observed by others that have

implemented downhill running [23, 57, 59, 61] and eccentric contractions [60, 61], suggesting that resistive-type exercises appear to alter running kinematics during periods of neuromuscular fatigue, muscle damage and soreness.

Unfortunately, studies of the effects of exercise-induced fatigue on the mechanics of other modes of endurance exercises (e.g. cycling and rowing) are scarce. This may be due to the equipment restricting the movement to a particular plane of motion, hence causing the biomechanics to be predictable. For example, the pedals on a cycling ergometer predominantly execute lower extremity movement in the sagittal plane with limited variation in range of motion for each cadence. On the other hand, there is greater degree of freedom and larger emphasis on balance and proprioception during running, as there is limited equipment controlling gait. Subsequently, it is reasonable to assume that running gait may be more susceptible to movement variation as a result of fatigue in comparison to other modes of endurance exercises.

It has been hypothesised that lower extremity range of motion following strenuous exercises is restricted due to perception of muscle pain [58], altered motor unit activation pattern [62] and reduced reflex sensitivity and/or decreased ability to use the stretch-shortening cycle [59]. This compromise in lower extremity range of motion has been associated with reductions in stride length and an increase in stride frequency during running [62]. Such biomechanical modifications would elevate energy expenditure due to an increase in the number of muscular contractions for a given running velocity [16]. Furthermore, compromise of the stretch-shortening cycle would minimise energy transfer between segments, rendering the locomotion inefficient. Attenuation of movement efficiency will increase energy expenditure during endurance exercise thereby limiting the body's capability to produce sufficient cardiovascular training stimuli for adaptation [7, 14].

A strong correlation ($r = -0.8$) has been reported between reduction in stride length and increase in $\dot{V}O_2$ during a RE test following downhill running [62]. In addition, Bonacci et al. [63] showed that alterations in running kinematics following cycling were related to changes in RE. Subsequently, alterations in running kinematics as a result of exercise-induced fatigue appear to contribute to a decrement in RE. However, Doma and Deakin [7, 14] found no correlation ($r = 0.12$–0.41) between alterations in RE and running kinematics following a lower body resistance training bout. The authors postulated that the lack of relationship may have been due to differences in the way each participant altered their gait pattern to compensate for muscle fatigue given that running experience varied between each participant. Further, runners tend to choose the optimal stride length and frequency at which they are most economical for a given condition. Subsequently, strong correlations between RE and running gait patterns may not always exist as runners may purposefully change their running gait to be more economical. Nonetheless, given that alterations in both oxygen cost of running and lower extremity kinematics were found, Doma and Deakin [14] suggested that changes in running technique may in part have impaired RE 6 h post a resistance training bout.

4.3 Impact of Neuromuscular Fatigue on Indices of Endurance Performance

A few studies have examined the impact of resistance training on MFGC and subsequent endurance performance. Palmer et al. [64] examined the effects of a whole body resistance training session on MFGC and RE. The results showed that RE was significantly impaired for up to 8 h post-exercise, although torque production was only significantly less immediately following the resistance training session. Doma and colleagues [15] also reported attenuation in RE with a reduction in anaerobic power for up to 48 h following lower body resistance training session in resistance-untrained men. Studies have also reported attenuation of cycling efficiency [65], cycling-specific power output [36], rowing-specific power output [18], running time-to-exhaustion [14, 16], repeated sprints [6] and running time-trial performance [66] with a reduction in MFGC 24–72 h following lower body resistance exercises. These findings suggest that resistance training could compromise the quality of various modes of endurance training sessions at sub-maximal and maximal effort if impaired MFGC is sustained between training sessions during concurrent training.

4.4 Impact of the Repeated Bout Effect of Resistance Training and Training Background on Resistance Training-Induced Fatigue

To this point, the impact of resistance training-induced fatigue following a single bout of resistance training has been discussed with the aim of determining its implications on endurance performance. However, following the initial bout of muscle-damaging exercises, it has been shown that the level of EIMD is reduced following a subsequent identical bout of muscle damaging exercises, known as the repeated bout effect [67]. Subsequently, when considering the repeated bout effect, it could be speculated that repeated resistance training bouts during the course of concurrent training could minimise RT-SEP [9]. Furthermore, by periodising concurrent training, the magnitude of fatigue impacting upon endurance training sessions could be

minimised [68]. Indeed, in a study examining the repeated bout effect of lower body resistance training sessions on running performance measures, Doma et al. [19] reported that the magnitude of increase in muscle damage markers (i.e. creatine kinase and DOMS) was attenuated following the second bout of lower body resistance training in resistance-untrained runners. However, no differences were found in RE measures between the first and second lower body resistance training bout, suggesting that the initial bout of resistance training did not provide any protection against the second bout of resistance training for sub-maximal running performance. In a similar cohort, Doma et al. [69] showed that lower extremity resistance training impaired running time-to-exhaustion (i.e. above 110% of anaerobic threshold) for 48 h post-exercise across three lower extremity resistance training bouts, indicating minimal protection from the initial exposure to resistance training. Furthermore, Doma et al. [14] reported impaired running time-to-exhaustion measures 24 h following lower body resistance training in runners despite their having undergone a flush-out period (i.e. performing a number of resistance training sessions to minimise the possibility of a repeated bout effect) prior to the experimental intervention. Similarly, for individuals who completed a flush-out period, Doma and Deakin [15] showed that three lower body resistance training sessions on alternating-days still impaired running time-to-exhaustion for 48 h following each resistance training session. Accordingly, whilst the levels of muscle-damage markers are attenuated following the second bout of muscle-damaging exercises, this protection does not always appear to occur for running performance measures. This phenomenon has been observed by several studies [15, 23, 59], where the trend over time in muscle damage markers (i.e. CK, DOMS and MVC) following muscle-damaging exercises was different to changes in running performance measures. This discrepancy appears to exist as the cause of impairment in running performance may not directly be associated with changes in the indirect markers of muscle damage given the complex neurophysiological factors required for execution of running. Subsequently, resistance training-induced fatigue may still be present irrespective of the number of times runners are exposed to resistance training.

With respect to training background, studies have reported that the magnitude of muscle damage and attenuation in muscle function is greater for resistance-untrained compared to resistance-trained individuals [70]. However, despite the discrepancy in the magnitude of muscle damage markers amongst different resistance training backgrounds, several studies have reported performance decrements in 1RM bench press performance [71], resistance training performance [39, 72] and elbow-flexor torque [73] 24–48 h following resistance training in resistance-trained individuals. Whilst these findings were not based on endurance performance measures, it appears that resistance training-induced fatigue can impair neuromuscular performance measures even in resistance-trained individuals. Furthermore, attenuated neuromuscular properties as a result of resistance training have also been associated with impaired running, cycling and rowing time-trial performance measures [6, 18, 36].

Whilst a number of studies have systematically compared the acute effects of muscle damaging exercises between resistance-trained and resistance-untrained individuals, such investigations between endurance-trained and endurance-untrained individuals are scarce. One study by Snieckus and colleagues [74] did compare the acute effects of eccentric contractions in long distance runners, cyclists and healthy untrained men and did find that the magnitude of muscle damage markers were less in endurance trained than untrained men. However, they reported that the endurance-trained men had ceased their resistance training program for only one month prior to study commencement, indicating that the lesser susceptibility to muscle damage may not have been due to differences in endurance training background, but as a result of their previous resistance training exposure given that protection against muscle damage can be preserved for as long as 6 months Nosaka et al. [75]. Further studies are warranted to systematically compare the susceptibility to muscle damage amongst different endurance training backgrounds with similar previous exposure to resistance training. Nonetheless, previous studies have reported increased muscle damage markers and impaired neuromuscular performance and running performance measures for at least 48 h post-muscle-damaging exercises in both endurance-trained [7, 15, 20] and endurance-untrained individuals [23, 59]. Accordingly, given that impaired physical performance measures are present 24–48 h post-muscle-damaging exercises for both resistance-trained and endurance-trained individuals, coaches should therefore be cautious of resistance training-induced fatigue for individuals of all training backgrounds.

5 Effects of Exercise-induced Muscle Damage and Delayed-Onset of Muscle Soreness on Indices of Endurance Performance

A common acute response following typical resistance training sessions is EIMD, particularly from the repetitive eccentric components of the exercises [67, 76]. One major consequence of EIMD is DOMS that occurs immediately post [77] and peaks over the next 72 h [57, 62]. The production of prostaglandin E_2 in response to muscle damage stimulates type III and IV pain afferents whereas

neutrophils permeate through the vascular wall to the damaged site due to leukotriene synthesis. These neutrophils cause further muscle damage via production of free radicals [77] and attract an influx of protein-rich fluid into the muscle (i.e. oedema swelling). Consequently, repetitive eccentric contractions cause inflammatory responses [78], increase intramuscular osmotic pressure and generate pain by the activation of group III and IV afferents [79]. The symptoms of EIMD have also been shown to cause marked reduction in MVC [52, 80, 81] and increased muscle stiffness [82] and swelling [83]. Given that indices of endurance performance are highly dependent on the mechanical properties of the musculature [84], symptoms associated with EIMD may need to be considered within the context of the RT-SEP phenomenon.

5.1 Consequences of Exercise-Induced Muscle Damage and Delayed-Onset of Muscle Soreness on Endurance Performance

Attenuation in endurance performance measures has been reported as a result of EIMD [23, 59, 62]. For example, Braun et al. [62] examined the effects of downhill running on RE 48 h post, and demonstrated an increase in $\dot{V}O_2$ and muscle soreness. The authors speculated that the attenuation of RE may have been attributed to muscle damage as indicated by DOMS. Similarly, Chen et al. [23, 59] examined RE 1–5 days following downhill running. The results showed that $\dot{V}O_2$ was significantly greater in conjunction with an increase in CK and a reduction in MVC over the course of the 5-day period. Given the increase in myofibre proteins and reduction in MVC, the authors postulated that the increased physiological cost of running following downhill running was associated with EIMD.

In contrast, Paschalis et al. [85] reported significant changes in muscle damage indicators (i.e. CK, DOMS and range-of-motion and MVC) 24–96 h following 120 maximal eccentric contractions although RE parameters were not affected. Other studies have also shown no alterations in RE 6–48 h post-resistance training despite significant changes in muscle damage indicators (i.e. CK, MVC and DOMS) [16, 66]. However, according to studies by Marcora and colleagues [66] and Doma and Deakin [16], running time-trial and time-to-exhaustion performances were impaired as a result of a single bout of resistance training. Furthermore, Byrne and Eston [86] reported a significant reduction in 30-second Wingate performance as a result of EIMD. Collectively, attenuation of endurance performance due to EIMD appears to be more susceptible at higher intensities. In fact, it has been suggested that fast-twitch muscle fibres are more prone to EIMD than slow twitch muscle fibres [6]. Given that fast-twitch muscle fibres are predominantly used above anaerobic threshold (AT), EIMD may have a greater impact on subsequent endurance performance at higher intensities [59].

Whilst the impact that EIMD has on endurance performance is dependent on the intensity of the endurance exercise following a single bout of resistance training, little is known of the effects of EIMD on endurance performance at various intensities following multiple bouts of resistance training. A more recent study by Doma and Deakin [15] examined the acute effects of concurrent training on DOMS and running performance measures across 6 days. Specifically, following a flush out period of three resistance training sessions, one group undertook resistance training on alternating days combined with running training on consecutive days (CCT) versus another group with resistance training on alternating days only (RT). The results showed that DOMS was significantly elevated during the 6-day period for both groups. Furthermore, RPE was significantly increased when running at 90–110% of AT with reduced running time-to-exhaustion during the running sessions although RPE was unaffected at 70% of running AT for the CCT group. When compared between groups, DOMS was significantly greater for the CCT group compared to the RT group. These findings demonstrate that DOMS is consistently elevated across multiple resistance training sessions, and that a combined resistance and endurance training micro-cycle induces greater DOMS. In addition, the concomitant increase in RPE and DOMS with reduced running time-to-exhaustion suggests that DOMS may have caused participants to perceive running to be harder at higher intensities thereby impairing running performance. Studies have also shown impaired running velocity [87], cycling time-trial performance [35] and rowing work rate [88] with increased RPE. Increased perceived exertion may be caused by a number of sensory cues [89] that may be governed by muscular pain, feelings of breathlessness during endurance exercise and increased motor unit recruitment necessary to produce equivalent sub-maximal force production as baseline prior to muscle-damaging exercises [90–92].

Of particular interest in the study by Doma and colleagues [15] was elevated DOMS across multiple resistance training sessions despite participants completing a flush-out period. These findings are consistent with those of Hassan et al. [93] who reported significantly elevated DOMS for 4 weeks across four bouts of eccentric knee extensor exercises. Collectively, whilst DOMS is attenuated following the initial bout of resistance training in resistance-untrained individuals, given that this measure is still present in individuals with previous exposure to resistance training, DOMS should be monitored following resistance training irrespective of training background or level of previous resistance training exposure.

The morphological disruption causing EIMD may also contribute to attenuation of endurance performance. A number of studies using magnetic resonance imaging have shown an increase in inorganic-phosphate:phosphocreatine (PCr) ratio as a result of EIMD following resisted eccentric contractions [94–96]. This metabolic alteration may be attributed to disruption of the sarcolemma or an increase in metabolic rate [96]. Furthermore, Davies et al. [97] showed an increase in indirect markers of muscle damage (i.e. CK, DOMS and MVC), inorganic phosphate concentration and inorganic-phosphate:PCr ratio with a concomitant reduction in time-to-exhaustion of knee extensors 24 h following knee extensor eccentric contractions. Walsh et al. [98] suggested that metabolic alterations in response to EIMD may result in reduced oxygen diffusion due to restricted blood flow, sub-optimal mitochondrial respiration and adenosine diphosphate desensitization of mitochondrial respiration. Furthermore, EIMD has been shown to inhibit insulin sensitivity which may resist the uptake of glucose into damaged muscles [99, 100]. Studies have also shown a compromise in glycogen synthesis following eccentric contractions that caused EIMD [101–103].

6 Effects of Exercise-Induced Muscle Glycogen Depletion on Endurance Performance

The literature suggests that the pathways that impair the neuromuscular properties are strongly dependent on the level of endogenous glycogen [104] and that inadequate energy supply may lead to failure of transformation from the neuromuscular event to mechanical force production [105]. Muscle glycogen has been shown to be the dominant fuel source at workloads above 65–70% of maximal oxygen consumption (VO_{2max}) [106, 107] during endurance exercise. The association between muscle glycogen content and endurance performance has been most apparent during cycling and running exercises performed at intensities from 65–85% of VO_{2max} [108–110]. Impaired performance in prolonged and high intensity endurance exercise has been attributed to energy substrate depletion, which includes muscle and liver glycogen [111, 112], blood glucose [113] and PCr [114, 115]. Accordingly, resistance training-induced muscle glycogen depletion may cause RT-SEP with inadequate recovery and nutrition.

6.1 Muscle Glycogen Content Following Resistance Training

There has been limited research examining glycogenolytic effects following resistance training. A study conducted by MacDougall et al. [116] showed that muscle glycogen depleted by 25% in the biceps brachii following three sets of 10 repetitions of elbow flexion/extension exercises. A more recent study by MacDougall and colleagues [117] reported similar findings where muscle glycogen content of the biceps brachii was reduced by approximately 20% following elbow flexion/extension exercises at 80% of 1RM to failure. In addition, Tesch et al. [118] reported that muscle glycogen content of the vastus lateralis decreased by 26% following repeated sets of resistive exercises of the lower extremity.

A study conducted by Robergs et al. [119] examined high and low intensity (i.e. 70 and 35% of 1RM, respectively) resistance training on muscle glycogen content by performing 6 sets of leg extension exercises whilst matching work between the two intensities. The results showed that muscle glycogen content reduced by 38 and 39% following low and high intensity resistance training protocols, respectively. These results show that muscle glycogen content is substantially reduced following resistance training regardless of resistance training intensity provided that work is matched between high and low training intensities. Pascoe et al. [120] reported that muscle glycogen content remained significantly depleted (by approximately 25%) 6 h following leg extension exercises at 70% of 1RM to failure. These findings suggest that muscle glycogen depletion can be sustained for several hours following resistance training, although it should be acknowledged that the degree to which glycogen depletion is sustained or not following RT may depend on post-exercise nutritional intake and activity levels following the session [121, 122]. Overall, the glycogenolytic effect elicited by resistance training could impair endurance performance within the hours following resistance training and which may impact on optimising endurance development during concurrent training.

6.2 Impact of Muscle Glycogen Depletion on Endurance Performance

A number of studies have examined the impact of muscle glycogen depletion on endurance performance [123–125]. For example, Suriano et al. [125] examined the effects of cycling-induced muscle glycogen depletion on RE. Following 60 min of cycling, the results showed a significant reduction in muscle glycogen content with a concomitant increase in oxygen consumption (VO_2) during the RE test. In addition, a significant relationship ($r = 0.57$) was found between changes in VO_2 and the magnitude of total glycogen depletion. Furthermore, the reduction in muscle glycogen content that claimed to have impaired RE was approximately 20%, which is less than previous reports that have examined muscle glycogen content immediately post to 6 h following resistance training (i.e. 25–39% loss of muscle glycogen content) [119, 126]. This relationship

between alterations in muscle glycogen content and VO_2 kinetics reported by Suriano et al. [125] demonstrated that muscle glycogen depletion as a result of strenuous exercises (e.g. resistance training) may contribute to attenuation of subsequent endurance performance. However, there is still limited evidence about the relationship between alterations in muscle glycogen content and endurance performance following resistance training which warrants investigation.

Despite the possibility of RT-SEP from reduction in glycogen content, it should also be noted that energy restriction and glycogen depletion enhances AMPK activity both under basal conditions and following acute exercise [127, 128], suggesting that exercising in glycogen-depleted conditions may produce a positive metabolic environment for endurance adaptation. However, as Knuiman and colleagues [122] recently mentioned in their review, training sessions repetitively commencing in a low glycogen state may increase the risk of an overtraining syndrome [129], and as a result, impair training capacity [130]. Accordingly, the role of glycogen status in chronic training adaptation remains a matter of debate warranting further research, particularly from a concurrent training perspective.

7 Conclusion

In summary, resistance training impairs muscular contractility via numerous physiological mechanisms which may have profound detrimental effects on the quality of subsequent endurance training sessions. Overall, RT-SEP may be a complex phenomenon that may occur from multiple neuropsychophysiological factors, including neuromuscular fatigue, alterations in running gait patterns, DOMS, and muscle glycogen depletion, all of which can be induced following a single bout of resistance training and acutely impair endurance performance. However, it should be acknowledged that the acute RT-induced attenuation on subsequent endurance performance is a short-term effect following a single RT session. In addition, the quality of endurance training may only be compromised if any RT-induced residual fatigue is not attenuated by aspects of program design (e.g., exercise order, length of between-mode recovery, etc.), particularly when endurance training is performed following resistance training. It is also critical to understand that the purpose of this review was to discuss the possibility that resistance training-induced fatigue may limit optimisation of endurance development. This is by no means to suggest that concurrent training impairs endurance development, but rather, even greater benefits of resistance training may be experienced by endurance athletes if potential fatigue between each mode of training session is accounted for, particularly with respect to resistance training-induced fatigue on subsequent endurance training sessions during a concurrent training program. Subsequently, resistance training should be encouraged for endurance athletes given the plethora of evidence indicating the benefits of resistance training for endurance development. Furthermore, whilst the level of interference that fatigue may have on the quality of endurance training session may be dictated by the nature of the training program, discussing training variables was beyond the scope of this review. However, we hope that the review encourages practitioners to become more aware of resistance training-induced fatigue for endurance athletes undertaking concurrent training.

Compliance with Ethical Standards

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