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REVIEW ARTICLE

High-Intensity Interval Training, Solutions to the Programming Puzzle

Part II: Anaerobic Energy, Neuromuscular Load and Practical Applications

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Abstract High-intensity interval training (HIT) is a well-known, time-efficient training method for improving cardiorespiratory and metabolic function and, in turn, physical performance in athletes. HIT involves repeated short (<45 s) to long (2–4 min) bouts of rather high-intensity exercise interspersed with recovery periods (refer to the previously published first part of this review). While athletes have used 'classical' HIT formats for nearly a century (e.g. repetitions of 30 s of exercise interspersed with 30 s of rest, or 2–4-min interval repetitions ran at high but still submaximal intensities), there is today a surge of research interest focused on examining the effects of short sprints and all-out efforts, both in the field and in the laboratory. Prescription of HIT consists of the manipulation of at least nine variables (e.g. work interval intensity and duration, relief interval intensity and duration, exercise modality, number of repetitions, number of series, between-series recovery duration and intensity); any of which has a likely effect on the acute physiological response. Manipulating HIT appropriately is important, not only with respect to the expected middle- to long-term physiological and performance adaptations, but also to maximize daily and/or weekly training periodization. Cardiopulmonary responses are typically the first variables to consider when programming HIT (refer to Part I). However, anaerobic glycolytic energy contribution and neuromuscular load should also be considered to maximize the training outcome. Contrasting HIT formats that elicit similar (and maximal) cardiorespiratory responses have been associated with distinctly different anaerobic energy contributions. The high locomotor speed/power requirements of HIT (i.e. ≥95 % of the minimal velocity/power that elicits maximal oxygen uptake [v/p$\dot{V}O_{2max}$] to 100 % of maximal sprinting speed or power) and the accumulation of high-training volumes at high-exercise intensity (runners can cover up to 6–8 km at v$\dot{V}O_{2max}$ per session) can cause significant strain on the neuromuscular/musculoskeletal system. For athletes training twice a day, and/or in team sport players training a number of metabolic and neuromuscular systems within a weekly microcycle, this added physiological strain should be considered in light of the other physical and technical/tactical sessions, so as to avoid overload and optimize adaptation (i.e. maximize a given training stimulus and minimize musculoskeletal pain and/or injury risk). In this part of the review, the different aspects of HIT programming are discussed, from work/relief interval manipulation to HIT periodization, using different examples of training cycles from different sports, with continued reference to the cardiorespiratory adaptations outlined in Part I, as well as to anaerobic glycolytic contribution and neuromuscular/musculoskeletal load.

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1 Introduction

High-intensity interval training (HIT) is defined as either repeated short (<45 s) to long (2–4 min) bouts of rather high- but not maximal-intensity exercise, or short (≤10 s,

repeated-sprint sequences [RSS]) or long (>20–30 s, sprint interval session [SIT]) all-out sprints, interspersed with recovery periods. These varying-length efforts combine to create training sessions that last a total of ~5–40 min (including recovery intervals). The four distinct HIT formats these generate are typically thought to be important training components for including in the periodization of training programmes, for the development of middle- to long-term physiological adaptation, and to maximize performance (refer to Fig. 1 in Part I of this review [1]).

Any exercise training session will challenge, at different respective levels relative to the training content, both the metabolic and the neuromuscular/musculoskeletal systems [2, 3]. The metabolic system refers to three distinct yet closely related integrated processes, including (1) the splitting of the stored phosphagens (adenosine triphosphate and phosphocreatine [PCr]); (2) the nonaerobic breakdown of carbohydrate (anaerobic glycolytic energy production); and (3) the combustion of carbohydrates and fats in the presence of oxygen (oxidative metabolism, or aerobic system) [4]. It is therefore possible to precisely characterize the acute physiological responses of any HIT session, based on (a) the respective contribution of these three metabolic processes; (b) the neuromuscular load; and (c) the musculoskeletal strain (Fig. 1, Part I [1]). Under these assumptions, since HIT is in the first instance a tool to improve cardiorespiratory fitness, we consider the cardiorespiratory responses reviewed in Part I as the primary variable of interest when programming HIT sessions. By logic, anaerobic glycolytic energy contribution and neuromuscular load/musculoskeletal strain (e.g., see [2, 3, 5]) are therefore likely the more important secondary variables to consider. While quantification of the phosphagen-related metabolism during HIT is also of interest, there is today, unfortunately, no sound technique for measuring its contribution during field-based exercise; these variables have therefore been omitted from the present review.

To illustrate the importance of quantifying the different physiological responses to HIT, it is worth noting that contrasting HIT formats that have similar (and maximal) cardiorespiratory responses can be associated with distinctly different anaerobic energy contributions [6] and/or neuromuscular load [5]. Indeed, the high-exercise intensities (i.e. ≥90–95 % of the minimal velocity/power that elicits maximal oxygen uptake [v/p$\dot{V}O_{2max}$] to 100 % of maximal sprinting speed (MSS) or power; Part I [1]) and volumes (runners can cover up to 6–8 km at v$\dot{V}O_{2max}$ per session in runners) of HIT sessions lead naturally to high engagement of the neuromuscular/musculoskeletal system. For athletes training twice a day, and/or in team sport players typically taxing both metabolic and neuromuscular systems simultaneously [7], both the anaerobic energy contribution and physiological strain associated with HIT sessions should be considered in light of the demands of other physical and technical/tactical sessions so as to avoid overload and enable appropriate adaptation (i.e. maximize a given training stimulus and minimize musculoskeletal injury risk [8, 9]).

Controlling the level of anaerobic glycolytic energy contribution during HIT sessions may be an important programming consideration. In many sports, especially those where a high glycolytic energy contribution is required (e.g. track-and-field sprint athletes, some team sports), so-called 'lactate production training' is believed to be an important component [10]. In this training, the high anaerobic energy contribution of HIT quickly depletes glycogen stores [11]. Thus, the implementation of such sessions needs to be managed as a function of training strategy [12] and competition schedule [13]. For example, when implementing a 'training low' strategy (i.e. training twice a day with likely reduced glycogen stores during the second session) [12, 14], coaches must select the morning HIT sessions that are most effective for depleting glycogen stores. Conversely, under some circumstances, coaches prefer lower 'lactic' sessions, such as for distance runners aiming to complete larger volumes of HIT (Part I [1]), or in team sport players with little time to recover their glycogen stores before competition [15]. Finally, considering that training sessions associated with high blood lactate levels are generally perceived as 'hard' [16, 17], programming fewer 'lactic' sessions might help in maintaining perceived stress at a low level during heavy training cycles.

The acute neuromuscular load/musculoskeletal strain associated with HIT sessions should also be considered with respect to long-term performance development, the possible interference with other training content, as well as acute and chronic injury risk. In the context of this review, neuromuscular load refers to the various physical stressors an athlete's anatomy encounters during the HIT session, and the acute effects this has on the neuromuscular and musculoskeletal systems. These include, amongst others, the tension developed in locomotor muscles, tendons, joints and bone, the muscle fibre recruitment and associated changes in neuromuscular performance as a function of the potential neural adjustments and changes in force-generating capacity. In practice, endurance coaches often seek to increase the neuromuscular characteristics of the HIT sessions in an attempt to improve the athletes' locomotor function (i.e. running economy) and, hypothetically, the fatigue resistance of the lower limbs (neuromuscular learning effect [18]). Performing uphill HIT sessions, climbing stairs in a stadium, running in the sand [19–21], introducing jumps, lunges, sit-ups and performing short shuttle runs or lateral running between work intervals (so-called Oregon circuits) [22] are all different attempts distance-running coaches use to try to make gains in their

runners. Similarly, cyclists perform low cadence HIT, sometimes as strength-specific sessions [23]. Nevertheless, neuromuscular fatigue, if maintained for several hours/days after the HIT session, can have a direct effect on the 'quality' of subsequent training sessions [8, 9] (both neuromuscularly oriented as strength or speed sessions [possible interference phenomenon [24]]) and on technical and tactical sessions in team sports. Despite limited evidence [25], residual neuromuscular fatigue post-HIT may reduce force production capacity and rate of force application during the following (strength/speed) sessions, which can, in turn, attenuate training stimuli for optimal neuromuscular adaptations. Therefore, in contrast to endurance athletes, team sport players generally tend to perform low-volume HIT sessions with minimal acute neuromuscular load/fatigue [8, 9, 26]. In the final phase of team sport competition preparation, however, a high neuromuscular load during HIT might also be needed in players to replicate specific game demands [27, 28]. Finally, if we consider that a good athlete is first an injury-free athlete, neuromuscular load should also be considered within the context of musculoskeletal pain and injury risk management. Running speed, time/distance run at high intensity, as well as specific running patterns or ground surfaces should, therefore, also be considered when programming HIT.

The aim of the second part of this review is to explore the acute metabolic (restricted to anaerobic glycolytic energy contribution in this part) and neuromuscular responses to HIT, in order to offer practitioners and sport scientists insight towards maximizing their HIT programming. Numerous HIT variations exist in terms of work/relief interval manipulation and periodization, and these will be discussed using different examples of training cycles from different sports, with continued reference to time spent near $\dot{V}O_{2max}$ (T@$\dot{V}O_{2max}$, Table 1), anaerobic glycolytic energy contribution (Table 2), as well as acute neuromuscular load and injury risk (Table 3). As this was a narrative and not a systematic review, our methods included a selection of the papers we believed to be most relevant in the area. Additionally, since the literature on the anaerobic glycolytic energy contribution and neuromuscular responses to actual HIT sessions is limited, we used, where appropriate, responses to other forms of high-intensity exercises to offer a starting point towards understanding the possible responses to HIT sessions implemented in the field. Standardized differences (or effect sizes, ES [29]) have been calculated whenever possible to examine the respective effect of the manipulation of each HIT variable. ES are interpreted using Hopkins' categorization criteria where: 0.2, 0.6, 1.2 and >2 are considered small, medium-large and very-large effects, respectively [30].

2 Metabolic and Neuromuscular Responses to High-Intensity Inverval Training (HIT)

2.1 Anaerobic Glycolytic Energy Contribution to HIT

While a gold standard method of assessing anaerobic glycolytic energy contribution to high-intensity exercise has not been established [31], measurement of accumulated O_2 deficit [4] and muscle lactate concentration [13] tend to be the preferred methods. The accumulated O_2 deficit method, however, has a number of limitations [32] and such data have only been reported in a few HIT-related studies [3, 6]. While accumulated O_2 deficit has been mentioned in the present review where possible, blood lactate accumulation is also reported as a surrogate marker. The use of blood lactate concentration to assess anaerobic glycolytic energy contribution has a number of limitations, including large individual responses, prior nutritional substrate status [33], session timing in relation to prior exercise [11], timing of sampling post exercise [34], the possible variations between different analyzers and sampling sites (e.g. finger vs. ear lobe), the effect of aerobic fitness [35] and its poor association with muscle lactate [36], especially following high-intensity intermittent exercise [13]. Nevertheless, since all subjects would generally be expected to present with normal nutritional/substrate stores when involved in a study, the potential influence of these latter factors for the present review is likely to be low. Therefore, with the aforementioned limitations in mind, we have used blood lactate changes during exercise to estimate anaerobic energy contribution for a given exercise stimulus [37]. In an attempt to compare the anaerobic glycolytic energy contribution during different forms of HIT, the present review will focus on post-HIT values and on the initial rate of blood lactate accumulation in the first 5 min of exercise [38]. This latter measure was selected due to the fact that blood lactate values collected after prolonged HIT sessions do not permit discrimination between different HIT sessions (e.g. at exhaustion, participants have already reached a plateau in blood lactate accumulation [39, 40]). The 5-min duration was chosen since it corresponds to the average time to exhaustion shown for continuous exercise at v$\dot{V}O_{2max}$ [41, 42] (Part I [1]), is near the duration of long interval bouts used with typical HIT sessions, as well as being close to the duration of most RSS. Additionally, this duration approximates the time needed for blood lactate levels to normalize as a function of the metabolic demand [37, 43]. For studies where blood lactate values were not provided ~5 min following exercise onset, the rate of blood lactate accumulation has been linearly extrapolated to a predicted 5-min value using pre- and post- (immediate) exercise measures (only exercises lasting 2–6 min were

Table 1 Recommendations for the design of run-based high-intensity interval training protocols for optimizing time at maximal oxygen uptake

Format	Work duration	Work intensity[a]	Modality	Relief duration	Relief intensity	Reps and series [b]	Between-set recovery		Expected T@$\dot{V}O_{2max}$	Acute demands[c]
							Duration	Intensity		
HIT with long intervals	>2–3 min[d]	≥95 % v$\dot{V}O_{2max}$	Sport specific	≤2 min	Passive	6–10 × 2 min			>10 min	Central ++++
						5–8 × 3 min				Peripheral ++
				≥4–5 min	≤60–70 % v$\dot{V}O_{2max}$ [b]	4–6 × 4 min				
HIT with short intervals	≥15 s[d,e]	100–120 % v$\dot{V}O_{2max}$ (85–105 % V_{IFT})	Sport specific	<15 s	Passive	2–3 × ≥8-min series	≥4–5 min	≤60–70 % v$\dot{V}O_{2max}$ [b]	>10 min	Central +++
				≥15 s	≤60–70 % v$\dot{V}O_{2max}$ (45–55 % V_{IFT})					Peripheral ++
RST	>4 s (>30 m or 2 × 15 m)	All-out	COD jumps explosive efforts	<20 s	≈55 % v$\dot{V}O_{2max}$ / 40 % V_{IFT}	2–3 RSS (each >6 sprints)	≥6 min	≤60–70 % v$\dot{V}O_{2max}$ [b]	0–3 min	Central + Peripheral +++
SIT	>20 s	All-out	Sport specific	≥2 min	Passive	6–10			0–1 min	Peripheral ++++
Game-based training	>2–3 min	Self-selected RPE >7	Sport specific[f]	≤2 min	Passive	6–10 × 2 min			>8 min	Central ++
						5–8 × 3 min				Peripheral +++
						4–6 × 4 min				

[a] Intensities are provided as percentages of v$\dot{V}O_{2max}$, V_{IFT} [179] or RPE

[b] These can also be game-based (moderate intensity) in team sports

[c] The number of symbols '+' indicate the magnitude of the expected demands with respect to more central versus peripheral systems

[d] To be modulated with respect to exercise mode (longer for cycling vs. running for example), age and fitness status (shorter for younger and/or more trained athletes)

[e] To be modulated with respect to the sport, i.e. longer for endurance and highly trained athletes than team sport and less trained athletes

[f] To be modulated with respect to physiological training objectives (manipulating playing number, pitch area etc.) so that specific rules are added for the fittest players to compensate for the fitness-related responses, which will parallel the HIT sessions

COD changes of direction, *HIT* high-intensity interval training, *reps* repetitions, *RST* repeated-sprint training, *SIT* sprint-interval training, *SSG* small-sided games, T@$\dot{V}O_{2max}$ time at $\dot{V}O_{2max}$, V_{IFT} peak speed reached in the 30–15 Intermittent Fitness Test, *v* $\dot{V}O_{2max}$ lower speed associated with maximal oxygen uptake

Table 2 Recommendations for the design of run-based high-intensity interval training protocols with respect to blood lactate accumulation

Format	Work duration	Work intensity[a]	Modality	Relief duration	Relief intensity[a]	Expected initial rate of blood lactate accumulation (mmol/L/5 min)
HIT with short intervals	≥20 s	<100 % $v\dot{V}O_{2max}$ (<89 % V_{IFT})	Straight line	≥20 s	≈55 % $v\dot{V}O_{2max}$ (40 % V_{IFT})	<5
	<30 s			<30 s		
	<15 s	<120 % $v\dot{V}O_{2max}$ (<100 % V_{IFT})	Straight line	≥20 s	Passive	<5
Game-based training	3–4 min	Self-selected RPE >7	Sport specific	≤2 min	Passive 55 % $v\dot{V}O_{2max}$ (40 % V_{IFT})	≤5
				≥4–5 min		
HIT with long intervals	<2 min	<100 % $v\dot{V}O_{2max}$	Straight line	2 min	Passive	≈5
HIT with short intervals	>25 s	>110 % $v\dot{V}O_{2max}$ (>90 % V_{IFT})	COD	>15 s <30 s	60–70 % $v\dot{V}O_{2max}$ (45–55 % V_{IFT})	≈6–7
HIT with long intervals	>3 min	≥95 % $v\dot{V}O_{2max}$	Straight line, sand, hills	>3 min	Passive	≈5–7
RST	<3 s	All-out	45–90° COD	>20 s	Passive	≤10
RST	>4 s	All-out	Straight line + jump	<20 s	≈55 % $v\dot{V}O_{2max}$ (40 % V_{IFT})	>10
SIT	>20 s	All-out	Straight line	>2 min	Passive	>10

[a] Intensities are provided as percentages of $v\dot{V}O_{2max}$ and V_{IFT} [179]

COD changes of direction, *HIT* high-intensity interval training, *RPE* rating of perceived exertion, *RST* repeated-sprint training, *SIT* sprint-interval training, V_{IFT} peak speed reached in the 30–15 Intermittent Fitness Test, $v\dot{V}O_{2max}$ lower speed associated with maximal oxygen uptake

included in this analysis). Post-HIT blood lactate values were categorized as low <3 mmol/L, moderate >6 mmol/L, high >10 mmol/L and very high >14 mmol/L. HIT sessions were also categorized based on the initial rate of blood lactate accumulation as follows: strongly aerobic <3 mmol/L/5 min; aerobic >3 mmol/L/5 min; mildly anaerobic >4 mmol/L/5 min; anaerobic >5 mmol/L/5 min; and strongly anaerobic >6 mmol/L/5 min.

2.1.1 Anaerobic Glycolytic Energy Contribution to Long-Bout Duration HIT Sessions

2.1.1.1 Effect of Work Interval Intensity In endurance-trained athletes performing constant-speed efforts at $v\dot{V}O_{2max}$, over interval durations longer than ~90 s, the initial rise in blood lactate ranges from 5 to 7 mmol/L/5min (Fig. 1a) [44–49]. For example, in elite French middle-distance runners ($v\dot{V}O_{2max}$ = 21.2 ± 0.6 km/h) performing repeated 600-m bouts (~1 min 40 s, work relief ratio 1), the initial rate of blood lactate increase was in the lower range of the values reported, i.e. ≈5 mmol/L/5 min [20]. While blood lactate accumulation will be related to training status [35] (Fig. 1a), it can likely be modulated acutely through the manipulation of HIT variables. Despite the lack of a direct examination in a similar group of athletes, study comparisons suggest that higher work intensities performed during long intervals likely create a higher rate of blood lactate increase and likely require a greater supply of anaerobic glycolytic energy [47–49].

2.1.1.2 Effect of Work Interval Duration Extending the interval duration of an HIT session without altering relief interval duration clearly increases anaerobic glycolytic energy contribution, as more work is completed in a given time period. In practice, however, coaches generally maintain the work/relief ratio when they manipulate HIT variables. In these latter conditions, an increase in work interval duration also likely increases anaerobic glycolytic energy contribution. For instance, doubling interval duration (2 vs. 1 min ran at $v\dot{V}O_{2max}$, work/relief ratio = 1) leads to substantial increases in anaerobic glycolytic energy release (Fig. 1a) [3]. Accumulated O_2 deficit (≈25 ± 2 vs. 21 ± 2 mL/kg, ES +2.3) and end-session blood lactate measurements (8.8 ± 3.6 vs. 4.8 ± 1.1 mmol/L, ES +1.7) were very largely and largely higher, respectively. Furthermore, in distance runners ($v\dot{V}O_{2max}$ 19.5 ± 0.7 km/h), increasing the interval duration ran at 100 % of $v\dot{V}O_{2max}$ from 2 min 10 s to 2 min 30 s (+15 %) with a work/relief ratio of 1:2 resulted in an almost twofold increase in the initial rate of blood lactate accumulation (from ≈6 to ≈10 mmol/L/5 min) [48]. It is worth noting, however, that a decrease in work intensity (from 93 to 84 % of $v\dot{V}O_{2max}$ with a 5 % grade on a

Table 3 Recommendations for the design of run-based high-intensity interval training protocols in reference to acute neuromuscular performance and potential injury risk

Format	Work duration	Work intensity[a]	Modality	Ground surface[b]	Relief duration	Relief intensity[a]	Acute change in muscular performance[c]	Injury risk level[d]
Game-based training	>2–3 min	Self-selected RPE >7	Sport specific	Sport specific	≤2 min	Passive 55 % $v\dot{V}O_{2max}$ (40 % V_{IFT})	SSG format-dependent	Traumatic ++ (contacts, joint sprain) overuse +
HIT with long intervals	>2–3 min[e]	≥95 % $v\dot{V}O_{2max}$	Straight line	Grass or treadmill	2 min	Passive	From improved ++ to impaired ++	Traumatic −; overuse +
	>2–3 min[e]	≥95 % $v\dot{V}O_{2max}$	Straight line	Track	4–5 min	60–70 % $v\dot{V}O_{2max}$ (45–55 % V_{IFT})	From improved + to impaired +	Traumatic ++ (tendons); overuse +++
	>2–3 min[e]	≥85 % $v\dot{V}O_{2max}$	Hill	Road	2 min	Passive	From improved + to impaired +	Traumatic −; overuse ++ (downhill = shocks)
HIT with short intervals	<15 s	<120 % $v\dot{V}O_{2max}$ (<100 % V_{IFT})	Straight line	Track, indoor	>15 s <30 s	60–70 % $v\dot{V}O_{2max}$ (45–55 % V_{IFT})	From improved + to impaired +	Traumatic−; overuse – (since short series)
	≤20 s	<110 % $v\dot{V}O_{2max}$ (>90 % V_{IFT})	Straight line	Track, indoor	≤20 s	Passive	From improved + to impaired ++	Traumatic −; overuse ++ (since long series)
	≤20 s	<110 % $v\dot{V}O_{2max}$ (>90 % V_{IFT})	COD	Track, indoor	≤20 s	Passive	From improved + to impaired ++	Traumatic ++ (ankle and knee sprain); overuse ++
	≤20 s	<110 % $v\dot{V}O_{2max}$ (>90 % V_{IFT})	COD	Grass	≤20 s	Passive	From improved − to impaired +++	Traumatic ++ (ankle and knee sprain + adductors); overuse +
RST	≤5 s	All-out	Distance <20 m, 45–90° COD	Sport specific	≤25 s	Passive	Impaired − to ++	Traumatic ++ (ankle and knee sprain)
	>3 s	All-out	Straight line >20 m	Sport specific	≤30 s	≈55 % $v\dot{V}O_{2max}$/40 % V_{IFT}	Impaired + to +++	Traumatic ++ (hamstring)
	≤3 s	All-out	Straight line <20 m + COD + jump	Sport specific	≤30 s	≈55 % $v\dot{V}O_{2max}$ / 40 % V_{IFT}	Impaired ++ to ++++	Traumatic − (hamstring), traumatic ++ (ankle and knee sprain)
SIT	>20 s	All-out	Straight line	Sport specific	>2 min	Passive	Impaired ++ to ++++	Traumatic ++++ (hamstring)

[a] Intensities are provided as percentages of $v\dot{V}O_{2max}$ and V_{IFT} [179]

[b] Comparisons between muscular fatigue following runs over different surfaces are adapted from the work of Sassi et al. [180] (with an energy cost of running lower for hard surface < sand < grass < treadmill) and Gains et al. [181]. While additional combinations between the different surfaces and exercise modes can be implemented (e.g. uphill running on a treadmill), these examples illustrate the main logic behind the selection of HIT variables

[c] Fatigue responses are likely athlete-dependent

[d] The level of injury rate is estimated based on the combination of running speed, total distance at high intensity, ground surface and specific running patterns, and expressed as a function of the number of '+' symbols

[e] To be modulated with respect to exercise mode (longer for cycling vs. running for example), age and fitness status (shorter for younger and/or more trained athletes)

COD changes of direction, *HIT* high intensity interval training, *RPE* rating of perceived exertion, *RST* repeated-sprint training, *SIT* sprint interval training, *SSG* small-sided games, V_{IFT} peak speed reached in the 30–15 Intermittent Fitness Test, $v\dot{V}O_{2max}$ lower speed associated with maximal oxygen uptake, + and − indicate the magnitude of the expected changes in neuromuscular performance and acute injury risk

treadmill) can compensate the effect of work interval extension from 1 to 6 min (work/relief ratio 1) and maintains blood lactate at 'acceptable' levels (i.e. 4–5 mmol/L post-exercise) [50].

2.1.1.3 Effect of Relief Interval Characteristics The influence of HIT recovery interval duration on the subsequent bout's anaerobic glycolytic energy contribution is not straightforward. While maintaining a work/relief ratio of 1, the accumulated O_2 deficit at the first min during 2 min/2 min intervals ran at $v\dot{V}O_{2max}$ was shown to be markedly greater than during a 1 min/1 min sequence (23.8 ± 1.6 vs. 20.5 ± 1.9 mlO_2/kg, ES 1.9) [3]. This is likely related to the lower oxygen uptake ($\dot{V}O_2$) attained

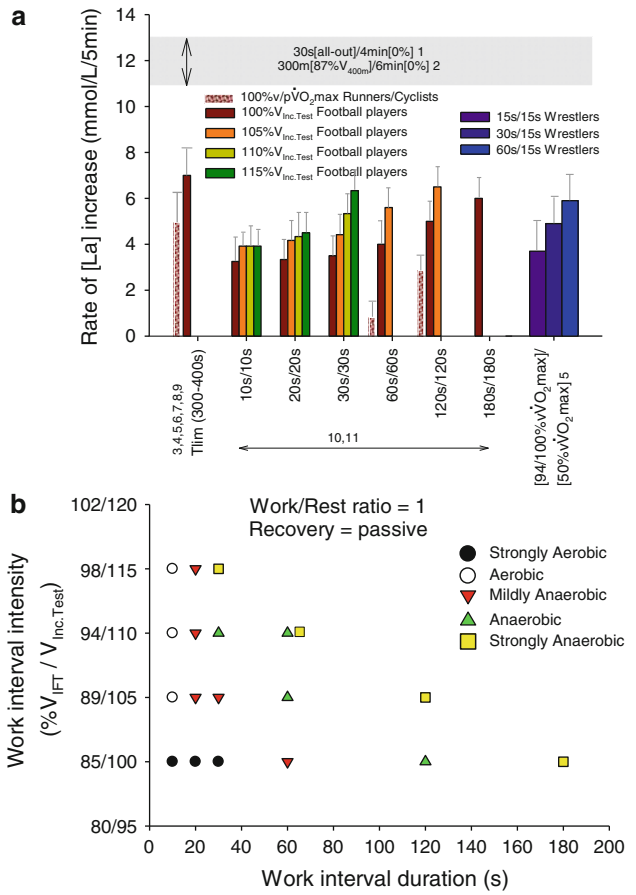

Fig. 1 Estimated anaerobic energy contribution to different short- and long high-intensity interval training (*HIT*) formats. **a** Anaerobic glycolytic energy contribution to different high-intensity interval training sessions as inferred from the initial rate of blood lactate accumulation (*[La]*). Values are mean ± standard deviation. High-intensity interval training sessions include either long or short intervals at different exercise intensities (% of the peak speed reached in the 30–15 Intermittent Fitness Test [V_{IFT}], speed or power associated with maximal O_2 uptake [v/p$\dot{V}O_{2max}$], or peak incremental test speed [$V_{Inc,Test}$] see review Part I [1]). The *grey rectangular box* represents the initial rate of blood lactate accumulation during 'lactate production training' i.e. all-out sprints or at the speed maintained during a 400-m run (V_{400m}) [10, 89]. **b** Schematic illustration of the energy system requirements for different forms of high-intensity interval training, with respect to blood lactate accumulation. (Strongly aerobic: <3 mmol/L/5 min; aerobic: >3 mmol/L/5 min; mildly anaerobic: >4 mmol/L/5 min; anaerobic: >5 mmol/L/5 min; strongly anaerobic: >6 mmol/L/5 min). 1 [10], 2 [89], 3 [45], 4 [61], 5 [62], 6 [44], 7 [46], 8 [48], 9 [47], 10 [63], 11 [3]. *Tlim* time to exhaustion, *v* $\dot{V}O_{2max}$ minimal velocity associated with maximal oxygen uptake

during longer (i.e. 2 min) rest periods, which may increase the O_2 deficit at the onset of the subsequent interval [51]. When the duration of the exercise interval is fixed, a shortening of the recovery interval is typically associated with higher anaerobic glycolytic energy contribution due to the increased exercise load (greater total work done in less time). However, following self-selected, 4-min work interval intensities ranging from 83 to 85 % v$\dot{V}O_{2max}$ (with a 5 % grade), reducing the relief interval from 4 to 1 min did not affect end-session blood lactate levels (6–7 mmol/L) in moderately trained runners (v$\dot{V}O_{2max}$ 17.6 ± 1 km/h) [47]. It is worth noting, however, that despite the inclined treadmill, the exercise intensity was submaximal in these studies [47, 50]. It is expected that for sessions ran at v$\dot{V}O_{2max}$, blood lactate responses would be higher and more responsive to relief interval manipulation [48]. It is also worth noting that blood lactate concentration, as a systemic measure with a certain inertia, is likely a less sensitive measure of anaerobic glycolytic energy contribution than accumulated O_2 deficit, which may explain the differences between Vuorimaa et al. [3] and Seiler and Hetlelid's [47] studies. The influence of the relief interval intensity on blood lactate and exercise capacity has been discussed in Part I of the review (section 3.1.1.3) [1]. When the relief interval duration is >3–4 min, active recovery (60–70 % v$\dot{V}O_{2max}$) can be used to accelerate blood lactate clearance compared with passive conditions [52, 53], leading to a lower accumulation throughout the session. While the effect of short (≤2 min) passive versus long (>3–4 min) active recovery on the anaerobic energy contribution to exercise has not been studied, it is worth noting that reductions in blood lactate may be indicative of effective 'lactate shuttling' and high-lactate consumption in working skeletal muscles [54].

2.1.1.4 Work Interval Modality Despite the common use of field-based HIT sessions involving hill repeats, sand running, stair climbing or plyometric work, there is limited data showing the anaerobic energy contribution of such exercise. In elite French middle-distance runners (v$\dot{V}O_{2max}$ = 21.2 ± 0.6 km/h, $\dot{V}O_{2max}$ 78 ± 4 ml/min/kg), blood lactate accumulation during a self-paced HIT hill session on the road (6 × 500 m [~1 min 40 s, work/relief ratio 1], slope: 4–5 %) was largely lower compared with a 'reference' self-paced track session (6 × 600 m) [blood lactate concentration ([La]) post-series: 8.5 ± 2.2 vs. 13.2 ± 4 mmol/L, ES −1.5] [20]. As discussed in Part I (Sect. 3.1.1.4) [1], this might be related to the lower absolute running speed attained during the inclined condition that did not compensate for an eventual higher mechanical muscle demand and potential change in muscle pattern activation [55]. Further studies are needed to compare the effect of different exercise modes (e.g. running vs. cycling vs. rowing) on anaerobic glycolytic energy contribution during actual HIT sessions in athletes.

In summary, while HIT with long intervals is likely the best format for adapting cardiopulmonary function (see Part I, i.e. 3–4 min intervals at >92–95 % v$\dot{V}O_{2max}$, interspersed with a passive ≤2-min recovery or ≥4- to 5-min active recovery) [1], blood lactate accumulation (and

likely anaerobic glycolytic energy contribution) will still reach high levels (rate of accumulation >5 mmol/L/5 min and end-session values >10 mmol/L) [48] (Fig. 1a). Therefore, for coaches striving to limit the anaerobic glycolytic contribution, the use of different forms of HIT, such as short intervals, may be warranted [19].

2.1.2 Anaerobic Glycolytic Energy Contribution to Short-Bout HIT Sessions

One of the interesting aspects of short-bout HIT, is the capacity to exercise at a high exercise intensity, yet with relatively low levels of blood lactate [19, 56–58]. From an evolutionary perspective, it is believed that such an intermittent locomotion strategy actually contributed to natural selection and survival of the species (i.e. "In nature, animals that stop and start win the race" [59]). The low lactate levels shown with brief intermittent exercise relate predominantly to stored oxygen sources. During the initial phase of short efforts, the O_2 bound to myoglobin supplies the majority of the O_2 requirements before the respiratory and circulatory systems are stimulated to meet the O_2 demand [57]. Thus, intense (>v/p$\dot{V}O_{2max}$) but short ($\leq \sim 20$ s) bouts of exercise can be repeated for up to 30 min before exhaustion when they are interspersed with passive pauses [57] or low-intensity recovery intervals. In the field, for example, endurance-trained runners (v$\dot{V}O_{2max}$ 18.5 ± 1.2 km/h) performing a 30 s/30 s HIT format [100 %/50 % v$\dot{V}O_{2max}$], managed to run at $\dot{V}O_{2max}$ from 1 to 3 min at a blood lactate level of 4 mmol/L [60]; a blood lactate level substantially lower than that obtained for the same exercise intensity (at both the cardiovascular and speed/power levels) during an incremental test (i.e., >10 mmol/L in the same study).

2.1.2.1 Work Interval Intensity and Duration When we consider short HIT sessions (i.e. 15 s/15 s) of a similar mean intensity (i.e. 85 % of v$\dot{V}O_{2max}$), we find that higher work interval intensities elicit greater blood lactate responses, and also shorten time to exhaustion [5]. For example, blood lactate at exhaustion was 9.2 ± 1.3 mmol/L for work/relief intensities of 90/80 % of v$\dot{V}O_{2max}$, 9.8 ± 1.4 mmol/L for work/relief intensities of 100/70 % (ES 0.4 vs. 90/80 %) and 11.3 ± 1.3 mmol/L for work/relief intensities of 110/60 % (ES 1.6 and 1.1 vs. 90/80 % and 100/70 %, respectively) [5]. The respective effects of work interval intensity and duration on blood lactate accumulation was also examined in semi-professional soccer players (peak incremental test running speed, $V_{Inc.Test}$ (see Part I [1]) 16.5 ± 2.3 km/h), during HIT that included intervals lasting 10 to 30 s (Fig. 1a [3, 44–48, 61–63]). Combined with data collected in other studies (e.g. see [5, 64]), this latter experiment [63] shows how the selection of the appropriate combinations of different HIT variables may be needed to reach specific blood lactate accumulation targets (Fig. 1b). For exercise at 100 % of $V_{Inc.Test}$, only work intervals longer than 1 min tend to be associated with high blood lactate levels. With work intensities of 110 % $V_{Inc.Test}$, however, anaerobic glycolytic energy contribution is already likely increased when exercise is longer than 30 s. At a fixed work interval intensity, increasing the work/relief ratio is associated, not surprisingly, with substantial increases in the initial rate of blood lactate accumulation (Fig. 1) [62]. Finally, at extremely high exercise intensities (i.e. 20 s @ 170 % of p$\dot{V}O_{2max}$ interspersed with 10-s rest periods for 2 min), the accumulated O_2 deficit may reach maximum levels [6] (i.e. similar to the anaerobic capacity of the subject, defined as the maximal accumulated O_2 deficit during 2–3 min of continuous high-intensity exercise to exhaustion) [31].

2.1.2.2 Relief Interval Intensity and Duration As most authors examining the impact of relief interval intensity during HIT on blood lactate accumulation have used runs to exhaustion, which are not typically completed in practice, the specific impact of recovery interval intensity on blood lactate accumulation during actual HIT sessions performed by athletes is not clear [39, 65, 66]. With increased recovery intensities (i.e. 50, 67 and 84 % of v$\dot{V}O_{2max}$) during a supramaximal 30 s [105 %v$\dot{V}O_{2max}$]/30 s effort model, a progressive increase in blood lactate is observed at exhaustion (estimated lactate from pH values: ≈6 ± 2, 10 ± 2, 11.5 ± 2 and 12.5 ± 2 mmol/L), despite progressive reductions in exercise time [65, 66]. In contrast, compared with passive recovery, active recovery (40–50 % v$\dot{V}O_{2max}$) during a repeated submaximal 15 s [102 %v$\dot{V}O_{2max}$]/15 s HIT model is consistently associated with slightly lower blood lactate values at exhaustion (10.7 ± 2.0 vs. 11.7 ± 2.1 mmol/L, ES −0.5 [39] and 12.6 ± 1.7 vs. 13.1 ± 2.7 mmol/L, ES −0.2 [40]). The exercise time is, however, shorter with active recovery, which prevents deciphering the respective effect of recovery intensity versus exercise time. In the only HIT study to examine recovery intensity over comparable durations (15 s [102 %v$\dot{V}O_{2max}$]/15 s), active recovery was shown to be associated with moderately higher post-HIT blood lactate values (10.7 ± 2.0 vs. 9.2 ± 1.4 mmol/L, ES +0.9 [67]). Thus, the lower muscle oxygenation level apparent with active recovery [40] will likely trigger for a greater anaerobic glycolytic energy system contribution [40, 68, 69]. But if we consider active recovery to be the preferred method for increasing T@$\dot{V}O_{2max}$ during HIT with short intervals (Part I, Sect. 3.1.2.3 [1]), then the programming of such HIT sessions with low blood lactate

levels may be difficult. In practice, reducing the work/relief ratio and using passive recovery, as with supramaximal HIT formats (e.g. 10 s [>100 %V_{IFT}]/20 s [0], holding V_{IFT} for the final speed reached at the end of the 30–15 Intermittent Fitness test, see Part I Sect. 2.7 [1]), provides an interesting alternative to achieve both a high T@$\dot{V}O_{2max}$ with moderate lactate production. In the latter case (i) the work interval duration prevents excessive anaerobic energy release (Fig. 1) yet is still great enough to reach a high $\dot{V}O_2$; and (ii) the passive recovery duration allows for partial PCr resynthesis [70], while limiting the drop in $\dot{V}O_2$. Another approach is to use submaximal work interval intensities (i.e. ≤100 % v$\dot{V}O_{2max}$, which is less likely to trigger anaerobic glycolytic energy contribution, Fig. 1) with active recovery periods (≥50 % v$\dot{V}O_{2max}$) [60]. It is worth noting, however, that all field-based HIT formats with short intervals shown in Fig. 1 are associated with low initial rates of blood lactate accumulation compared with long intervals [56, 58].

2.1.2.3 Work Interval Modality The introduction of changes of direction (COD) into HIT has been shown to moderately increase blood lactate accumulation, irrespective of the work intensity and duration (ES ≈ +1, for all short HIT models tested, i.e. 10 s/10 s, 15 s/15 s and 30 s/30 s) [71]. This is not surprising given the increased mechanical demands of the repeated accelerations inherent with consecutive COD [72] and the fact that, on average, the actual running speed is higher with COD (to compensate for the time lost while changing direction). In fact, COD running is likely to increase peripheral (particularly biarticular locomotor muscles) and, in turn, systemic $\dot{V}O_2$ demands (with a possibly greater upper body muscle participation). Therefore, while absolute anaerobic energy contribution is likely higher with COD [73, 74], the percentage contribution to total energy expenditure may be lower during high-intensity runs [75]. When the intensity of the work intervals during HIT was adjusted for the time lost with COD, blood lactate values were surprisingly similar or even lower compared with HIT without COD [76]. The acute blood lactate responses to different forms of HIT with short intervals (10 s/20 s), including either running, sprinting, hopping or squatting efforts every second work interval (Fig. 2a) were also compared in eight highly-trained adolescent handball players ($V_{Inc.Test}$ 16–17 km/h) [77]. Compared with the running-only condition, there were very large lower blood lactate concentrations following the sprinting (ES −3.5) and squatting (ES −2.9) conditions, but there was no clear difference with hopping (ES 0.0). While the cardiorespiratory responses to these specific sessions should also be considered (Part I [1]), these latter results provide important

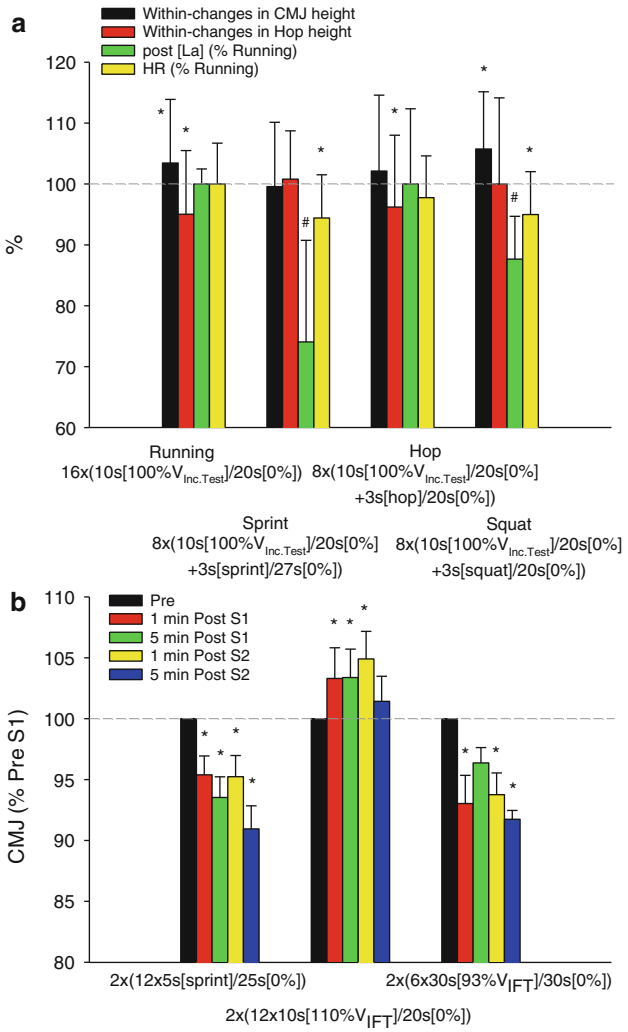

Fig. 2 Changes in physiological responses and neuromuscular performance following different high-intensity interval training formats. Values are mean ± standard deviation. **a** Changes in countermovement jump (*CMJ*) and hopping (*Hop*) height following four different high-intensity interval training sessions (short intervals [10 s/20 s] including running and, every second work interval, either running again or sprinting, hopping or squatting efforts) and differences in mean heart rate (*HR*) and post-exercise blood lactate (*[La]*) compared with the running-only condition [77]). **b** Changes in countermovement jump height following three different high-intensity interval training sessions (repeated-sprint sequence and high-intensity interval training of either 10 s/20 s or 30 s/30 s format) performed over two series [145]. *S1* first series, *S2* second series, V_{IFT} peak speed reached in the 30–15 Intermittent Fitness Test, $V_{Inc.Test}$ peak speed reached during an incremental test, * indicates moderate standardized difference, # indicates large standardized difference

information for coaches wishing to manipulate the anaerobic glycolytic contribution of their HIT sessions within their periodized programmes.

Finally, while there is limited evidence so far, running surface is another variable that likely affects anaerobic glycolytic energy release during HIT. When team-sport athletes (3-km running time 12 min 34 s) performed 3 sets

of 7 × 25 to 45-s intervals at the highest sustainable intensity (work/relief ratio 1/2–3) in the sand, their end-session blood lactate was largely greater (ES +1.0) than when they did the same session on grass [21].

2.1.3 *Anaerobic Glycolytic Energy Contribution During Repeated-Sprint Sequences (RSS)*

2.1.3.1 Work/Relief Ratio As illustrated in Fig. 3a, end-exercise blood lactate values reported for RSS can range from 6 to 18 mmol/L [78–88] and can reach very high levels, i.e. similar to those reached during specific 'lactate production training' (all-out 30-s efforts [10] or 300 m ran at the speed maintained during a 400-m run [V_{400m}] [89]). While it is clear that methodological inconsistencies can partly explain the differences between the protocols/studies (Sect. 2.1), these data show that manipulating both the sprint distance/duration and the recovery intensity/duration can have a substantial impact on the anaerobic glycolic contribution to exercise. When sprints are longer than 4 s (i.e. >25 m) and when the recovery interval is less than 20 s and generally active, the initial rate of blood lactate accumulation is consistently high (i.e. >10 mmol/L/5 min; Fig. 3b). In contrast, shorter sprints and/or longer recovery durations may be less taxing on the anaerobic energy system. The initial rate of blood lactate accumulation during an RSS is also largely correlated with its work/relief ratio, irrespective of the sprinting distance (Fig. 4a). Interestingly, extremely high initial rates of blood lactate accumulation (i.e. >than that observed for all-out 30-s efforts [10] or 300 m ran at V_{400m} [89]) can be reached when repeating 4-s sprints on a non-motorized treadmill [87] or when long sprints (>6 s) are separated by short recovery durations (≈17 s) [84]. These data are of particular interest for team-sport coaches wanting to implement 'lactate production training', since RSS are more team-sport specific and might improve player motivation in those reluctant to exercise on a track and/or for longer efforts (i.e. sprint interval training).

2.1.3.2 Work Modality In addition to the aforementioned variables, introducing COD and/or jumps into RSS have the potential to influence the blood lactate response. When 25-m sprints (departing every 25 s, relief intensity 2.0 m/s) are repeated over a shuttle (180° COD), post-test blood lactate (9.3 ± 2.4 vs. 10.0 ± 10.7 mmol/L, ES +0.3) can be slightly increased [90]. Interestingly, however, when RSS are matched for initial sprint time (requiring a reduction in the sprinting distance as COD angles increase), RSS with or without 90°-COD angles is recommended to either minimize or maximize blood lactate accumulation, respectively [81]. For RSS that involve 0°, 45°, 90°, or 135°-COD angles, [La] accumulation (post-

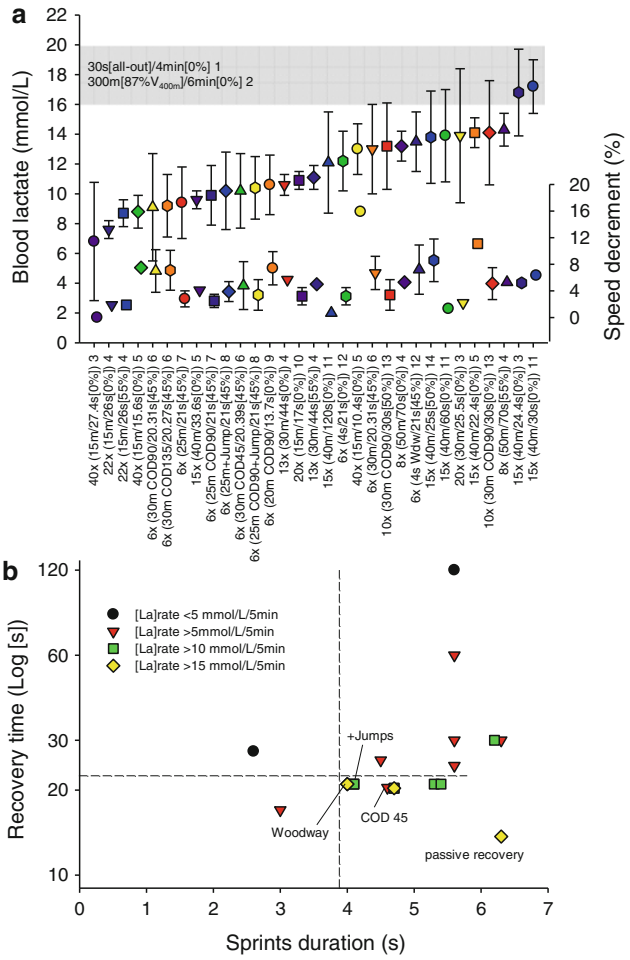

Fig. 3 Estimated anaerobic energy contribution to different repeated-sprint sequence formats. **a** Blood lactate measured following the different repeated-sprint sequences and their associated speed decrement (values are mean ± standard deviation when available). The *grey rectangular box* represents the initial rate of blood lactate (*[La]*) increase during 'lactate production training', i.e. all-out sprints or at the speed maintained during a 400-m run (V_{400m}) [10, 89]. **b** Rate of blood lactate increase during different repeated-sprint sequences as a function of recovery and sprint duration. The *dashed lines* represent the shortest sprint and recovery duration likely needed to achieve a rate of blood lactate increase >10 mmol/L/5 min (with the exception of one repeated-sprint sequence performed with 45°-changes of direction (*COD*): >5 mmol/L/5 min). All repeated-sprint sequences leading to a rate of blood lactate increase >10 mmol/L/5 min included an active recovery, except for one which passive recovery is mentioned. 1 [10], 2 [89], 3 [78]. 4 [79], 5 [80], 6 [81], 7 [82], 8 [83], 9 [84], 10 [85], 11 [86], 12 [87], 13 [88]. *COD* changes of direction, with associated angle, *Woodway* refers to sprints performed on a non-motorized treadmill [87]

resting values) of 10.1 ± 2.2, 8.0 ± 2.3, 6.1 ± 2.5 and 7.4 ± 2.3 mmol/L have been reported, with moderate-to-large differences between those COD angles shown (except 45° and 135°). While it is intuitive to think that muscle recruitment (and blood lactate) may increase with greater COD angles, absolute sprinting speed is actually lower for the larger angles in this particular setting (due to the shorter

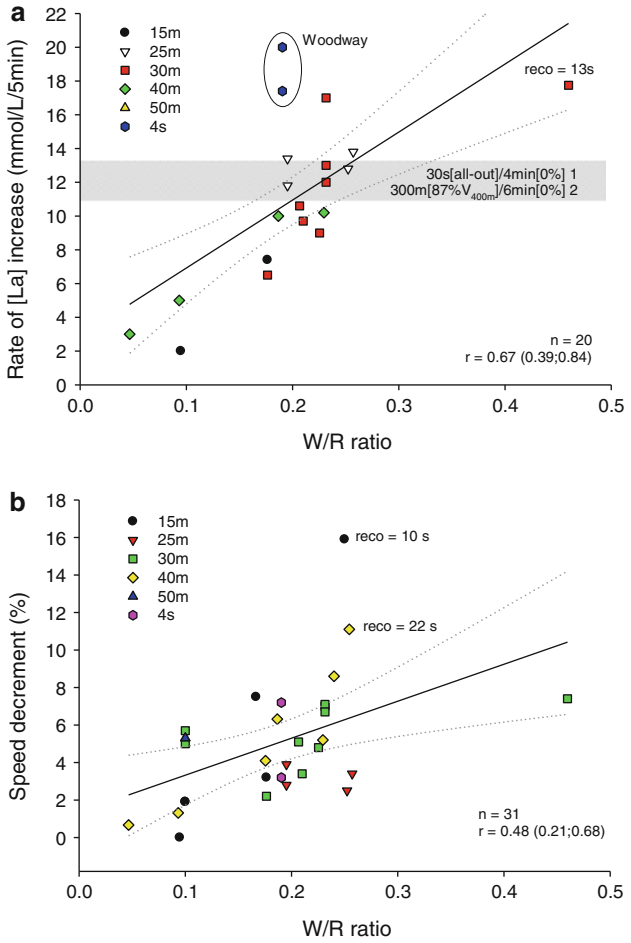

Fig. 4 Relationships (*r* [90 % confidence limits]) between the initial rate of blood lactate (*[La]*) increase (**a**) and speed decrement (**b**) during select repeated-sprint sequences and work/rest (*W/R*) ratios. Correlation coefficients are provided with 90 % confidence intervals. The *grey rectangular box* represents the initial rate of blood lactate increase during 'lactate production training', i.e. all-out sprints or at the speed maintained during a 400-m run (V_{400m}). The *black oval* highlights the two sprints performed on a non-motorized treadmill (i.e., *Woodway*) [87]. 1 [10], 2 [89]. *Reco* relief interval duration

distances) [81]. Finally, jumping following each sprint has also been shown to be associated with small increases in blood lactate accumulation (ES +0.2–0.3) [83], probably as a consequence of the increased total muscular work.

2.1.4 Anaerobic Glycolytic Energy Contribution During Sprint Interval Session

SIT, also termed 'speed endurance training', is another form of HIT that involves near to maximal (all-out) efforts [10]. Compared with the RSS format, efforts and recovery periods are typically longer (e.g. repeated 30-s sprints interspersed with 2–4 min of passive recovery [10]). When physical education students repeated 4 × 30-s sprints at 200 % of $p\dot{V}O_{2max}$ interspersed with 2-min rest periods, the accumulated O_2 deficit reached 67 % of their anaerobic capacity [6], defined as the maximal accumulated O_2 deficit during an exhaustive 2–3 min continuous exercise [31]. Total accumulated O_2 deficit over the 4 sprints however was 3 times higher than the anaerobic capacity. Blood lactate levels during SIT generally reach 16–22 mmol/L [2, 10, 91, 92], which corresponds to blood lactate accumulation rates >10–15 mmol/L/5 min (Figs. 3, 4). While SIT variables have rarely been manipulated for the specific purpose of examining anaerobic glycolytic energy contribution, it is likely that shorter sprints and/or lower intensities will lower the anaerobic glycolytic energy contribution. In addition, sprints >45 s are likely to engage a greater contribution from the aerobic system (i.e. >40 % [4]), which will lower the anaerobic glycolytic energy demand during subsequent sprints. In contrast, if the goal is to produce a large amount of lactate, recovery periods should be long enough (i.e. >1.5–2 min) to allow for the aerobic system to return to resting levels, to enable an O_2 deficit to occur at the onset of the following exercise [51]. Since high intramuscular H^+ concentrations may inhibit glycolysis and PCr recovery [93, 94], extending the recovery period might also allow for the following sprint to be achieved with a (partially) recovered acid/base status and greater PCr stores, allowing, in turn, a greater mechanical power production and a greater anaerobic glycolytic energy contribution. For instance, post-exercise blood lactate levels were shown to moderately increase from 13.3 ± 2.2 to 15.1 ± 1.7 mmol/L (ES +0.9) in well trained team sport athletes (V_{IFT} 18.9 ± 1.5 km/h) when the recovery between 30-s all-out shuttle-sprints was increased from 30 to 80 s [95]. Maximal blood lactate values during 30-s sprints have also been shown to be greater when interspersed by 4 (≈17 [96] and 22 [92] mmol/L) versus 2 min (15.3 ± 0.7 mmol/L [97]). These results highlight the effect that SIT variable manipulation has on the blood lactate response, which has important implications for HIT programming.

2.1.5 Summary

In this section we have shown how anaerobic glycolytic energy contribution during an HIT session is HIT–parameter dependent. SIT- and RSS-type HIT formats are typically associated with elevated rates of blood lactate accumulation. During RSS sessions, sprint durations greater than 4 s with work relief intervals less than 20 s lead to the highest blood lactate accumulation. In contrast, SIT sessions require relief interval durations equal or longer than 4 min to maximize anaerobic glycolytic energy contribution. As shown, the manipulation of HIT variables with short intervals may allow practitioners to vary the level of anaerobic glycolytic energy contribution to a given

session. In general, longer interval durations and higher interval intensities (when $>v\dot{V}O_{2max}$) will elicit higher blood lactate levels. While the evidence is limited, increasing the intensity of exercise during the recovery interval in short HIT bouts may also increase blood lactate accumulation. Interestingly, anaerobic glycolytic energy release can also be manipulated during game-based HIT (i.e. small-sided games, as detailed in Part I, Sect. 2.2 [1]) via changes in rules and/or player number and pitch dimension [27], so that blood lactate accumulation can be maintained at low levels despite a prolonged $T@\dot{V}O_{2max}$ [98]. Practical examples of HIT sessions associated with varying levels of anaerobic glycolytic energy contribution are shown in Table 2.

2.2 Neuromuscular Responses to HIT

Quantifying the neuromuscular load of an HIT session is important, since it (1) affects the HIT performance, and subsequently, the $T@\dot{V}O_{2max}$ (see Part I [1]); (2) might have potential carry-over effects for subsequent training sessions [99–101]; (3) might modulate long-term neuromuscular adaptations [18, 24, 26, 101]; and (4) may influence injury risk during (i.e. traumatic-type injuries) [102–104] and following (i.e. overuse-type injuries both in runners [105] and team sport athletes [104]) the HIT sessions. Therefore, understanding how to manipulate HIT variables to modulate neuromuscular load during HIT is important to maximize a given training stimulus and minimize musculoskeletal pain and/or injury risk. However, until only recently, there has been limited data quantifying the effect of HIT variable manipulation on neuromuscular function.

Data on neural and muscular adjustments using force trace measures and motor nerve stimulation [106–109] following high-intensity exercise suggests that fatigue induced by HIT including either very short (<20 s) to short ($\leq$1 min) and/or non-maximal efforts ($\leq\sim$120 % $v\dot{V}O_{2max}$) tends to be predominantly peripheral in origin [106, 107, 110, 111] (i.e. alterations to muscle excitability and excitation-contraction coupling, related to intramuscular potassium concentration disturbance and accumulation of metabolic by-products including inorganic phosphate and hydrogen ions, respectively [112]). Interestingly however, performance impairment during repeated long (i.e. $\geq$30 s) and all-out sprints may be more essentially related, in addition to the usually reported peripheral mechanisms, to central fatigue [113]. However, few authors, if any, have investigated the neuromuscular responses to HIT sessions using sport- and training-specific tasks, with maximal isometric voluntary contractions of the active musculature (MVC) generally chosen as the laboratory-based task for assessment of neuromuscular changes [106–110]. This is problematic, since (muscle) fatigue is task-specific [114]. Alternate and perhaps more specific field-based measurements might include the assessment of changes in musculoskeletal stiffness regulation [22, 115], stride parameters [112], countermovement jump (CMJ) height and sprint speed [51, 83, 116]. While CMJ height reflects the efficiency of both muscle activation and muscle contractile properties [51], sprint speed can be a less precise measurement of neuromuscular fatigue, as changes in inter- and intramuscular coordination factors, motor control and/or stride parameters may limit the speed decrement during maximal sprints, so that fatigue appears to be less pronounced. For example, jump performance shows a much greater performance decrement (i.e. 3 times) than sprint performance during repeated-sprint and jump sequences [83, 117]. Another limitation of these latter performance measures is that they are generally of maximal nature, while in practice, HIT with short and long intervals are often not performed at maximal intensity (i.e. less than MSS or peak power output). It is also worth noting that the acute effects of high-intensity running on leg muscle performance depends on the physiological characteristics and training history of the athlete [89, 116, 118]. For example, while explosive athletes involved with speed events (i.e. track and field) or team sports generally show impairments in muscular performance following high-intensity exercises [89, 116, 119], endurance-trained athletes tend to show less impairment [89], no changes [116, 119] or even improvements [120] (possible post-activation potentiation [121]). During repeated-sprint exercises, similar findings have been reported, with endurance-trained athletes showing less fatigue than team sport athletes [122]. Finally, with the exception of a very limited number of studies [21], post-exercise neuromuscular tests are generally performed either immediately after or 10–30 min following the HIT sessions. Since residual fatigue is likely to be extended following such sessions, investigating the time course of muscular and neural responses to HIT over a longer time course (i.e. hours/days [123–125]) is needed to assess potential carry-over effect of HIT on the subsequent training sessions [8, 9].

2.2.1 Neuromuscular Responses to Long-Bout HIT

The effect of interval duration on changes in CMJ height during an HIT session was examined in national level runners ($v\dot{V}O_{2max}$ 19.1 $\pm$ 1 km/h) [120]. There was no within- or between-HIT differences in CMJ height throughout 1 min/1 min versus 2 min/2 min interval sessions ran at $v\dot{V}O_{2max}$ [120]. Similarly, there was no effect on stride length throughout successive intervals [120]. These findings may relate to the particular profile of the

endurance-trained athletes, the fact that running speed was actually similar between the two protocols and that blood lactate levels remained moderate, even during the 2 min/2 min intervals (8.8 ± 3.6 mmol/L). While not implying cause and effect, impairment in muscle function during high-intensity exercise is generally accompanied by high blood lactate levels, i.e. >10–12 mmol/L [89, 126].

The effect of running speed on neuromuscular load may be indirectly inferred from recent results reported in highly-trained young runners ($v\dot{V}O_{2max}$ 18.6 ± 0.9 km/h) [2]. CMJ height and 20-m sprint times were examined before and after long-duration HIT (5 × 3 min) in normoxia (90 % $v\dot{V}O_{2max}$) or normobaric hypoxia (inspired O_2 fraction 15.4 %, simulated altitude of 2400 m, 84 % $v\dot{V}O_{2max}$) [2]. Since neuromuscular performance tests were performed in normoxia for both running conditions, and considering similar constraints with the exception of the actual running speed (e.g., same air resistance, treadmill), the specific effect of a 16 % difference in running speed on neuromuscular load could be indirectly examined. For both HIT protocols, no changes in CMJ height were observed, either immediately or 20-min post. Interestingly, however, while there was no change in sprint times following the runs completed at 84 % $v\dot{V}O_{2max}$, sprint times were improved after the runs completed at 90 % $v\dot{V}O_{2max}$ immediately after the session (ES for difference in changes 0.2–0.5). This possible immediate post-activation potentiation effect [121] was no longer evident 4-h post.

Finally, compared with a 'reference' track session (6 × 600 m), stride frequency (2.98 vs. 3.1 strides/s) and amplitude (185 vs. 203 cm) tended to be lower during a road-based HIT hill session (6 × 500, 4–5 % incline) [20], suggesting a lower loading of the hamstring muscles. In agreement with this, inclined (5 %) 250-m sprints have been associated with reduced stride length (−14 %) and rate (−7 %), and a +27 % increase in push-off time, compared with 300-m sprints on the track [55]. Importantly, despite no changes in the activation of quadriceps muscles, hamstring muscles were also less activated. Finally, in middle-distance runners ($v\dot{V}O_{2max}$ 21.8 ± 1.8 km/h), performance measures reflecting muscle power have also been shown to be lower for uphill versus horizontal incremental running performance [127]. Taken together, these data [20, 55, 127], and others [128], suggest that incline running lowers hamstring strain in runners, which could be beneficial to prevent injuries during maximal and/or high volume sessions [129]. However, since athletes might be required to run downhill to prepare for the next interval, care should be taken with respect to potential acute muscle damage arising from the downhill phase [130]. Therefore, if coaches can find strategies for avoiding the downhill running, HIT hill sessions could represent a useful alternative to reducing overall acute hamstring strain and appears well suited for high-volume training cycles. It is also worth noting that over longer time frames (once the acute muscle damage is recovered), downhill running can have a prophylactic effect [130] (i.e. repeated-bout effect) and can therefore be effective training for preventing future musculoskeletal injuries.

2.2.2 *Neuromuscular Responses to Short-Bout HIT*

The acute neuromuscular responses to 5 × 300-m runs (77 % of MSS, ≈120–130 % $v\dot{V}O_{2max}$) interspersed by 1-min recovery periods (100-m walk-jog) was examined in well-trained middle- and long-distance runners [109]. Despite non-significant changes in maximal torque during a knee extensor MVC (−5 %, with ES ≈ −1.2, which still shows a large effect), the HIT session caused severe acute peripheral fatigue, as evidenced by reduced efficiency of excitation-contraction coupling (e.g. −28 % for twitch torque, ES > −5). Muscle contractile function was recovered and even improved within 10 min following the session (e.g. +11 % for twitch torque, ES > 1), while maximal torque during the MVC remained depressed for at least 120 min (≈−5–6 %, ES < −1.2). Due to the task-dependency of acute muscular fatigue [114], the potential carry-over effects of HIT-related fatigue on subsequent training sessions involving sport-specific movement patterns (e.g. sprints, squats) is not straightforward and requires further research. In the only study to date to assess neuromuscular performance after an HIT session (3 sets of 7 × 25 to 45-s intervals performed at the highest sustainable intensity, work/relief ratio 1/2–3), most measures showed a return to baseline within 24 h [21]. As well, the recovery of some measures was slightly better when the session was performed on sand versus grass. It is possible, therefore, that longer recovery periods may be required when sessions are performed on a harder surfaces (e.g. track, road). In addition, consideration for the run surface and its influence on the neuromuscular load should be considered when programming run training (i.e. Table 3).

While direct comparisons between long- and short-bout HIT have yet to be documented, the acute neuromuscular load may be greater with short intervals for the following reasons. First, work intensity is generally higher with shorter intervals (Part I, Fig. 3 [1]). While the majority of muscle fibres might already be recruited during long intervals (considering a minimal recruitment threshold at >75–85 % $v\dot{V}O_{2max}$ for both type I and II fibres [131, 132]), the firing rate and relative force development per fibre is likely greater during short intervals [133]. Second, short intervals require frequent accelerations, decelerations and re-accelerations (for which occurrences are increased if

the intervals are performed over shuttles). In addition to the increased metabolic and muscle force demands during acceleration phases of high-intensity exercise [72, 134], the completion of short intervals requires achievement of a greater absolute speed. For example, for a run completed at 120 % of $v\dot{V}O_{2max}$ in the field, a small portion (i.e. 1−3 s) of such a run may, in fact, need to be run at 135 % to compensate for the time lost during both the acceleration and deceleration phases [67]. When this high-speed portion of HIT is considered in relation to the percentage of MSS and anaerobic speed reserve achieved (>80 % and >50 %, respectively), the level of neuromuscular engagement is high [133] and should be considered within the context of training load and injury management (especially hamstring muscles [129]; traumatic-type injuries [102–104]). Finally, for unique athletes, such as extremely tall or heavy basketball or rugby players, the musculoskeletal load of long intervals might actually be higher than during short intervals, due, potentially, to poor running technique and economy. Therefore, for these particular athletes, long run-based intervals should be avoided, or implemented on soft surfaces (e.g. sand, grass [21]), bikes or rowing ergometers to prevent injuries.

While the specific impact of COD on the neuromuscular system during field-based HIT sessions has yet to be examined in detail, it may exacerbate lower limb muscular fatigue compared with straight-line running due to the additional accelerations and decelerations required [135]. The impact of a strength-oriented HIT session on the neuromuscular system was also examined in eight high-level endurance runners (peak incremental test speed [$V_{Inc.Test}$] 20.7 ± 1.7 km/h) [22, 77]. Interestingly, when repeating six 200-m runs at 90–95 % of $v\dot{V}O_{2max}$ (36–38 s), with alternate 30-s dynamic or explosive strength exercises, the runners avoided impairment in leg stiffness during running (8.08 ± 1.49 vs. 7.87 ± 1.31 kN/m, ES < −0.2). While this might be related to the specific population of athletes (Sect. 2.2), this finding suggests that such training is well tolerated by distance runners, and might have limited carry-over effects on subsequent sessions. Whether injury risk is increased from such training is not known, but the increased load on the musculoskeletal system should be considered [22]. Finally, the impact of different running pattern/exercise modes on acute neuromuscular responses during HIT with short intervals was also examined in adolescent handball players (detailed in Sect. 2.1.2.3) [77]. The acute neuromuscular responses to the four different HIT sessions were protocol specific, with improvements in CMJ height shown following the running and squatting formats (possible post-activation potentiation [121]), and reductions in hopping height shown following the running and hopping conditions (possible neuromuscular fatigue related to localized overload on these muscle groups). While the effect of these sessions on neuromuscular fatigue over longer durations is unknown (i.e. 24–48 h), these data suggest that in team sport players, running pattern/exercise modes have important implications for programming, which should be considered with respect to other training sessions to avoid overload/injury and maximize adaptation.

2.2.3 *Neuromuscular Responses to RSS*

During run-based RSS, the reduction in running speed observed throughout the successive sprint repetitions reflects the progressive increase in overall locomotor stress, as evidenced by an impaired force production capacity, changes in stride patterns, musculoskeletal stiffness regulation [115], and both neuromuscular adjustments and metabolic disturbances at the muscle level [112]. Despite its poor reliability [136], the percentage of speed decrement (%Dec) is still the more commonly reported index used to assess acute fatigue during RSS; a marker that varies from 1 to 12 %, depending on the (run-based) RSS format (Fig. 3a). In practice, %Dec values should be interpreted with caution with respect to neuromuscular load. While a high %Dec is likely associated with an increased (muscle) fatigue in the acute setting, the actual musculoskeletal strain of the sequence (with respect to possible muscle damage and/or injury risk) is more likely related to running patterns and the running speed maintained during each repetition. For instance, an RSS allowing a higher running speed to be maintained (i.e. low %Dec) may induce a greater musculoskeletal strain. When data from the studies shown in Fig. 4 were pooled, %Dec was moderately and positively correlated with work/relief ratio ($r = 0.48$; 90 % confidence limits 0.21, 0.68, Fig. 3b). A higher work/relief ratio is generally associated with reduced PCr resynthesis and an accumulation of blood lactate (Fig. 4a) and metabolites in the muscle, which may partially explain the greater impairment of repeated sprinting capacity [112, 114]. It is, however, worth noting that for extremely short recovery periods (i.e. 10 s [80]), the percentage of speed decrement is dramatically increased (see 'outlier' data point, Fig. 4b). Since this latter format might impose a substantial load on the musculoskeletal system, it should only be implemented with consideration for the timing of the other neuromuscular-oriented training sessions so as to avoid overload.

In addition to variations in sprint duration and work/relief ratio, the introduction of COD to RSS can also affect the fatigue profile, and hence acute neuromuscular load. Compared with straight line sprinting for example, RSS with 180°-COD were associated with a slightly lower

%Dec (ES −0.4) [90]. When comparing the effect of COD-angles per se (on sprints adjusted for initial sprint duration), the fatigue response to the RSS were angle-dependent [81]. Values for %Dec were 6.7 ± 2.5, 4.8 ± 3.6, 7.0 ± 3.2 and 7.1 ± 3.0 % for straight line, 45°, 90° and 135°, respectively (with %Dec for 45° being substantially lower than for the three other conditions) [81]. Running with 45° COD required variation in muscle activity compared with straight-line running (slowing down slightly to turn), without the need to apply large lateral forces, such as with the greater angles (135°) [135]. This finding suggests that repeated sprints involving 45° COD may be an effective alternative to reducing acute neuromuscular load during RSS. Preliminary results from our laboratory during repeated high-intensity 16.5-m runs with 90°-COD (performed within 4 s, departing every 20 s) have shown that a stabilization time after a single-leg drop jump is increased compared with straight-line HIT (Hader, Mendez-Villanueva and Buchheit, unpublished results). In the same study, a fatigue-induced modification in lower limb control was observed with CODs (i.e., a selective reduction of electromyography activity in hamstring muscles), which may induce, in turn, a potential mechanical loss of knee stability (Hader, Mendez-Villanueva and Buchheit, unpublished results). Therefore, RSS including sharp COD might expose athletes to a higher acute risk of both ankle sprain and knee injuries. This risk may be exacerbated in athletes not used to performing such movement patterns at high speed (e.g. martial art athletes, gymnasts).

The specific impact of deceleration during RSS has the potential to increase acute muscle fatigue (i.e. impaired repeated-sprinting performance), but only when a large number of sprints are performed, i.e. >11 sprints [137]. Finally, adding jumps after each sprint during RSS is also likely to increase neuromuscular load. This was shown by a moderately greater %Dec for sprints, with (ES +0.7) and without (ES +0.8) shuttles (180°) [83]. Finally, when jumps were added to RSS that involved shuttles, the %Dec for jump performance was highest (12 ± 4 vs. 8 ± 4 % for shuttle vs. straight sprints, ES +0.8), which suggests that this latter RSS format is likely more demanding on lower limbs. With respect to traumatic injuries at the muscle level, the introduction of COD during RSS has the advantage of restricting sprinting distance and stride length, which might help prevent hamstring overload/acute injuries.

Finally, the time needed to recover from acute neuromuscular fatigue following RSS is important for RSS programming [99]. In cycling studies, it has been shown that when a second RSS was performed, 6 min after the first, fatigue was exacerbated (i.e. %Dec of 17 vs. 13 % for the second vs. the first set) [138], suggesting that a certain level of neuromuscular fatigue remained. When interspersing run-based RSS with 5 min rest, followed by 6-min active recovery and then a short specific warm-up (total time ≈15 min), repeated-sprint performance was not impaired during the second set [83]. Explosive strength, however, had not fully recovered, since CMJ height was 5 % lower (ES −0.4) than before the first set [83]. Compared with cycling, however, changes in neuromuscular coordination and stride adjustments appear to compensate for the acute fatigue during sprints to maintain running performance [51]. This data suggest that when RSS are repeated within a short time period (i.e. within 2–5 min [139–141]), neuromuscular fatigue, as evaluated from post-exercise jump tests, is accentuated. To implement 'quality' RSS sessions (if this is indeed possible [26]), a prolonged and likely active recovery period should be implemented between sets (possibly >15–20 min) to maximize muscle recovery.

2.2.4 Neuromuscular Responses to SIT

The large speed or power decrement score generally observed (20 % for repeated 30-s cycling sprints [92, 97], 5–20 % for track sessions involving repeated 300-m runs [89]) suggests that neuromuscular function is largely impaired following a SIT session. Recent data suggests also that, in contrast to other forms of HIT (e.g. short-duration and/or non-maximal efforts [106, 107]), central mechanisms may be the primary origin of the impairment to MVC performance following the repetition of long sprints [113]. There is, however, little data available examining the neuromuscular response to SIT variable manipulation. Neuromuscular fatigue (as assessed by jump height) following an HIT session in elite 400-m runners (personal best 92 % of world record) is likely protocol-dependent [89]. The magnitude of the SIT-induced reduction in CMJ height was shown to be positively correlated with initial CMJ height and is affected by between-run recovery duration, with longer recovery durations (and hence the greater the anaerobic glycolytic energy contribution, see Sect. 2.1) showing greater impairments in jump height [89]. When examining the neuromuscular responses in team sport athletes to single maximal sprints lasting 15 (100 m), 31 (200 m) and 72 s (400 m), Tomazin et al. [142] showed that knee extensor MVC reduction was only apparent immediately and 5 min after the 72-s sprint, and was fully recovered 30-min post-sprint.

In run-based sports, SIT is often implemented in a straight-line fashion or over an arcing shape of >200 m (running over half of a football pitch for example). While this setting can allow players to reach very high running speeds (close to their MSS for the first intervals), it can dramatically increase hamstring injury risk [129]. SIT sessions should therefore be considered using a cost/benefit

approach. It is clear that SIT is unlikely to develop MSS [26, 143]. However, when an appropriate warm-up is used and when distance is built-up over the sessions, such SIT formats can, in addition to triggering the well-known metabolic adaptations in the muscle [144], serve as a prophylactic intervention (i.e. preparing lower limb muscles and tendons to tolerate the extreme tensions associated with maximal sprinting). For a safer option (or for the initial sessions of a SIT programme), coaches can implement SIT sessions using 40-m shuttles [143].

2.2.5 Neuromuscular Responses to Different Forms of HIT

There is limited data on the direct comparison of different forms of HIT on neuromuscular responses in a field setting. In one such study, 14 highly trained young handball players (V_{IFT} 19.2 ± 1.1 km/h) performed three alternate HIT sessions: two series of either RSS (12 × 5 s [sprints]/25 s [0]), short HIT (10 s [110 %V_{IFT}]/20 s [0]) or short HIT (30 s [93 %V_{IFT}]/30 s [0]) [145] [Fig. 2b]. CMJ height was substantially increased following the 10 s/20 s exercise, whereas it was substantially impaired after both RSS and 30 s/30 s HIT. The 10 s/20 s format, as prescribed here, was unlikely associated with a substantial anaerobic energy release (as discussed in Sect. 2.1), and given the relatively high (but not maximal) running speed, a possible post-activation potentiation [121] could have enhanced jumping performance (Fig. 5). In contrast, speed was maximal during RSS, and anaerobic glycolytic energy contribution was likely high for both such RSS and 30 s/30 s formats (Figs. 1, 3). While the impairment of CMJ following RSS is not surprising (see Sect. 2.2.3), the decreased CMJ after the 30 s/30 s format shows that a high-work intensity per se is not required to induce muscular fatigue, and that metabolic perturbations at the muscle level may be enough to induce neural and muscular adjustments [112].

2.2.6 Summary

In summary, variance in subject training status between studies renders it difficult to be certain as to the effects that different HIT formats have on musculoskeletal and neuromuscular load (i.e. endurance-trained athletes generally perform long-bout HIT while team sports players usually perform short-interval HIT and RSS). Nonetheless, there is likely a bell-shaped relationship between the intensity of an HIT session and the acute neuromuscular performance, with too low and too high (all-out) intensities having not enough and acute detrimental effects, respectively (Fig. 5). Work intensities >80–85 % v$\dot{V}O_{2max}$ require recruitment of fast twitch fibres [131, 132], induce post-activation potentiation and possibly lead to long-term structural

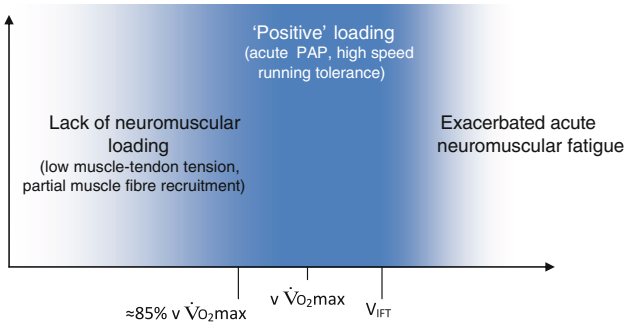

Fig. 5 Neuromuscular load and associated performance outcomes as a function of running speed during typical high-intensity interval training sessions. *PAP* post-activation potentiation, V_{IFT} peak speed reached at the end of the 30–15 Intermittent Fitness Test, $v\,\dot{V}O_{2max}$ lowest running speed required to elicit maximal oxygen uptake

adaptations that allow fatigue-resistance to high-speed running [146]. In contrast, supramaximal-to-maximal (≥120 % v$\dot{V}O_{2max}$, ≥100 % V_{IFT}) intensity exercises are likely associated with acute impairments in muscular performance. The residual fatigue from HIT sessions that persists over time may have large implications for subsequent training (carry-over effect), but there is limited data documenting the recovery course of neuromuscular function following HIT. Finally, in addition to mechanical work intensity, the associated metabolic responses and potentially accumulated metabolites within muscle should also be taken into account when examining the acute neuromuscular load of a given session (Sect. 2.2.5).

HIT volume should also be considered with respect to potential musculoskeletal pain and/or injury risk [104, 105]. Since impact forces imposed on the lower limbs increase with running speed [147], choosing slower running intensities (e.g. at the speed midway between maximal lactate steady state and v$\dot{V}O_{2max}$, i.e., vΔ50, which can allow the attainment of $\dot{V}O_{2max}$ in less trained athletes [148], see Part I, Sect. 3.1.1.1 [1]) or softer ground surfaces (e.g. grass or synthetic track vs. road, Table 3) might be a safer option to accomplish high-volume training in distance runners [149]. Similarly, because of the possible association between high-intensity running volume and soft-tissue injuries in team sports [104], coaches might strive for time-efficient HIT sessions with short running distances completed at high speed [104]. Finally, running pattern (e.g. COD, introduction of jumps), exercise mode (e.g. cycling, running), ground surface (e.g. pavement, synthetic track, grass, sand) and terrain (uphill, downhill), all have direct implications on injury risk, and should be selected in programming based on a risk/benefit analysis. Practical examples of HIT sessions associated with varying levels of neuromuscular/musculoskeletal load are offered in Table 3.

3 Programming HIT

3.1 Programming with the Thibault Model

As discussed in Part I (Sect. 2.4) [1], many coaches prescribe exercise intensity based on the athletes' perceived exertion during training. The Thibault model [150] (Fig. 6) is an empirical tool that practitioners can use to programme HIT sessions, yet with unique work interval durations and intensities. With respect to these independent variables, the model describes the appropriate number of repetitions and series that achieve similar ratings of perceived exertion. While this is of particular interest when there is the desire to vary HIT formats (e.g. to enhance athlete motivation/interest), HIT sessions of a similar perceived level of difficulty are unlikely associated with the same acute physiological responses, which has important implications for programming (see Part I). Therefore, to select the most appropriate HIT sessions within the model, each parameter needs to be considered in light of the aim of the session (Part I, Fig. 1 [1], and Part II, Tables 1, 2 and 3).

3.2 Using Specific HIT Sessions to Target Selected Physiological Responses in the Microcycle

Choosing an appropriate HIT session for a given training cycle is no simple task, and this may be the art of good programming [8, 9]. The general rules that guide selection include (1) the session's likely acute metabolic and neuromuscular responses (targeting long-term adaptations); and (2) the time needed to recover from the session (Part I, Fig. 1, to 'fit into the puzzle' [1]). Based on content presented throughout Parts I and II of this review, the following section offers the practitioner various examples of HIT sessions that could be considered when the aims are to optimize T@$\dot{V}O_{2max}$ (Table 1), manipulate anaerobic glycolytic energy contribution (Table 2) and alter neuromuscular and musculoskeletal load (Table 3).

3.2.1 Time Required Between HIT Sessions and Following Training Sequence(s)

As detailed in Sects. 2.1 and 2.2, manipulation of HIT parameters elicits different metabolic consequences (e.g. glycogen depletion, metabolite accumulation in muscle) and acute neuromuscular load. Additionally, there is limited data on the time course of post-HIT muscle metabolite clearance, glycogen store repletion and neuromuscular recovery [21]. Finally, the individual nature of these responses to training status and training history makes the challenge of offering recommendations to the practitioner difficult. Nevertheless, on a neuromuscular basis,

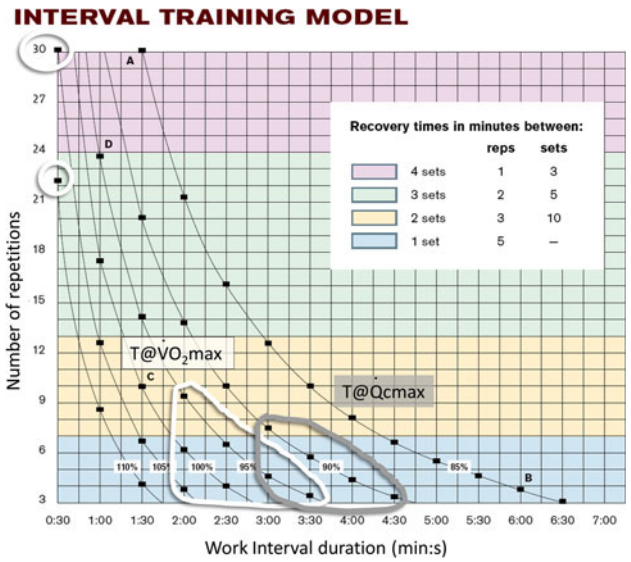

Fig. 6 'Thibault' graphical model [150]. All particular high-intensity interval training sessions (*black dots*) are obtained while manipulating work interval duration (*x axis*) and the number of repetitions (*y axis*). All sessions are (empirically) believed to represent a similar overall exercise strain, based on empirical observations with respect to the rating of perceived exertion responses (see Part I, Sect. 2.4 [1]). The *black lines* join the different sessions performed at a similar percentage of the minimal velocity/power associated with maximal oxygen uptake (*v/p* $\dot{V}O_{2max}$). For a work interval of 95 % of p$\dot{V}O_{2max}$ for example, if 3 intervals are performed, the suggested interval duration of each is 3 min 30 s, if 4–5 intervals are performed, then 3 min each, if 6–7 intervals are performed, then 2 min 30 s each, etc. The number of series needed to perform a given number of repetitions is provided in the table, together with the between-series recovery duration. As an example, the two *small white circles* on the *y axis* illustrate two high-intensity interval training sessions (i.e. 22 × 1 min 30 s at 110 % and 30 × 2 min at 105 % of v/p$\dot{V}O_{2max}$) believed to be identical with respect to RPE. The *large white-circled zone* illustrates the maximized high-intensity interval training formats with respect to time spent at maximal oxygen uptake (*T@* $\dot{V}O_{2max}$) and the *grey-circled zone* illustrates the maximized high-intensity interval training formats with respect to time spent at maximal cardiac output (*T@* $\dot{Q}c_{max}$) (Part I [1]). Reproduced from Thibault [150], with permission

endurance-trained athletes may require less time between successive sessions than team sport athletes (Sect. 2.2). In addition, within a given athlete, the recovery time course of different biological systems is also likely different following high-intensity exercise (e.g. muscle performance [21, 124] appears to recover faster than muscle glycogen repletion [15]), so that an accurate measure of recovery for the practitioner is currently lacking. In the absence of a gold standard measure of an athlete's overall metabolic and neuromuscular recovery following HIT, assessment of cardiac autonomic function (ANS) via heart rate variability (HRV) has emerged as a promising alternative [151–155]. Indeed, it is thought that such data may be used practically to individualize the programming of HIT sessions [156, 157]. While the rationale for this practice has yet to be

Table 4 Example of high-intensity interval training programming over two different weekly microcycles for an elite female Olympic distance triathlete (International Triathlon Union points ranking <100)

Microcycle	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Preparation phase							
AM	Swim (90 min, 5 km, including 30 min long HIT[a])	Rest	Swim (90 min, 5 km, including 30 min short HIT[b])	Swim (60 min, 3 km, technique)	Swim (90 min, 5 km, including 30 min long HIT[a])	Bike (3–5 h; <VT_1HR)	Run off-road, hilly, LSD (2 h, <VT_1HR)
PM	Bike (3 h, <VT_1HR steady), followed by run (30 min easy, grass)	Rest	Run (90 min steady)	Bike (90 min trainer session, including single leg HIT [182], 3–4 × 3 min [p$\dot{V}O_{2max}$]/3 min [50 W])	Run (fartlek, 60 min variation in terrain)	Rest	
Note: this microcycle is characterized by a progressive increase in training volume, with weekly training durations ranging from 15 to 25 h/wk (small, medium, large, small, etc.)							
Competition phase							
AM	Swim (90 min, 5 km, including 30 min long HIT[a])	Swim (open water; 4 km)	Swim (90 min, 5 km, including 30 min short HIT)	Swim (60 min, 3 km, technique)	Swim (90 min, 5 km, including 30 min long HIT[a])	Bike (3–5 h; <VT_1HR)	Swim (long, <VT_1HR, 2 h, 8 km)
PM	Bike (3 h, <VT_1HR steady), followed by run (30 min easy, grass)	Run, HIT, 4–8 × 3 min [v$\dot{V}O_{2max}$]/4 min [50 % v$\dot{V}O_{2max}$]	Rest	Bike, HIT, 4–6 × 5 min [p$\dot{V}O_{2max}$]/3 min [100 W])	Run (60 min, steady)		Run off-road, hilly, LSD (2 h, <VT_1HR)
Note. Recovery (i.e. day-off) inserted and subsequent programme adjusted based on athlete perceptions, soreness, and morning monitoring of cardiac autonomic status [183]. Microcycle is characterized by similar weekly training durations (15–30 h/week), but higher training intensities and therefore higher training loads. Note that longer HIT intervals are performed on the bike to compensate for the likely slower $\dot{V}O_2$ kinetics [184]							

[a] Due to the specificity of swim HIT programming, these sessions are not discussed in the present review. For the reader's information, swim long HIT sessions include repetitions of 100-m intense bouts at various speed (1 min 13–1 min 07) interspersed with 10–20 s passive recovery bouts for a total volume of ≈15 repetitions

[b] Short HIT sessions include repetitions of 25–50 m at various speeds (13–31 s) interspersed with 10–25 s passive recovery bouts for a total volume of ≈30 repetitions

AM morning, *HIT* high-intensity interval training; *LSD*, long slow distance, *PM* afternoon, *p or v* $\dot{V}O_{2max}$, power or velocity associated with maximal oxygen uptake, *VT_1HR* heart rate associated with the first ventilatory (aerobic) threshold

clearly demonstrated, it has been suggested that to maximize adaptations, HIT sessions should be performed when vagal-related indices of HRV are high [156, 157] (i.e. when, after the initial post-exercise decrease, cardiac ANS activity has recovered or even rebounded above pre-existing levels). Both the acute and long-term ANS responses to HIT sessions are likely related to the nature of the session (e.g. exercise intensity and anaerobic glycolytic energy release [85, 158]) and an athlete's training background (with fitter athletes recovering faster [159, 160]). While acknowledging the limitations of HRV to track the recovery of some biological variables (e.g. glycogen stores and neuromuscular function [161]) the combined analysis of these aforementioned studies [151–155] using moderately trained athletes (e.g. physical education students) confirm previous training recommendations [8], i.e. that an average of ≈48 h should separate HIT sessions to enable the majority of athletes to perform and train maximally. This premise is supported by research in runners showing that when HIT sessions are separated by less than 2 days, a progressive overload is likely to occur [162]. While this occurrence could be part of an athlete's normal training cycle (i.e. shock microcycle [163, 164] generally followed by reduced training or tapering), this can lead to non-functional overreaching when repeated over several days or weeks [162].

Table 5 Example of HIT programming over two different weekly microcycles for an elite male rower during the competition phase

Week	Time	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
1.Competition phase: build race pace speed and anaerobic capacity	AM1	Row 20 × 500 m (100–120 s) [≥LT2]/250 m (90–120 s) [<LT1]	Row (90 min) [<LT1]	Row 10 × 1 km (~3 min) [≥LT2]/1.5 min [passive recovery or <LT1]	Row (60–90 min), including HIT 5 × 250 m (50–60 s) [>LT2 to all out]/3–4 min [passive recovery or <LT1]	Row (90 min) [<LT1]	Row (80 min), including HIT 5 × 3 min [>LT2]/3 min [passive recovery or <LT1]	Rest
	AM2	Rest	Rest	2–2.5 h ride	Rest	Rest	2.5–3 h ride [<LT1]	
	PM	Row (60–90 min) [<LT1]	Row (60 min) including 5 × 10–15 stroke starts (max 15–20 s)/open recovery [<LT1]	Rest	Row (60–75 min) [<LT1]	Row (90 min) [<LT1], 5 × 10 stroke starts with bungee[a] (max 15–20 s)/open recovery [<LT1]	Rest	
2. Competition phase: build race pace speed and anaerobic capacity	AM1	Row 40 × 250 m (50–60 s) [≥LT2]/250 m (90–120 s) [passive recovery or <LT1]	Row (90 min) [<LT1]	Row 5 × 2 km (~6–7 min) [≥LT2]/3 min [passive recovery or <LT1]	Row (60–90 min), including HIT 6 × 250 m [>LT2 to all out]/3–4 min [<LT1]	Row (90 min) [<LT1]	Row (80 min), including HIT 6 × 3 min [>LT2]/3 min [<LT1]	Rest
	AM2	Rest	Rest	2–2.5 h ride	Rest	Rest	2.5–3 h ride [<LT1]	
	PM	Row (60–90 min) [<LT1]	Row (60 min) including 6 × 10–15 stroke starts (max 15–20 s)/open recovery [<LT1]	Rest	Row (60–75 min) [<LT1]	Row (90 min) [<LT1], 8 × 10 stroke starts with bungee[a] (max 15–20 s)/open recovery [<LT1]	Rest	

a Bungee sessions simulate long HIT sessions (i.e. cardiopulmonary intervals, Part I [1])

AM1 first morning session, *AM2* second morning session, *HIT* high intensity interval training, *LT1* the intensity associated with the first lactate (aerobic) threshold, *LT2* the intensity associated with the second lactate (anaerobic) threshold, *max* maximum, *PM* afternoon

When endurance-trained athletes ($v\dot{V}O_{2max}$ 20.5 ± 1 km/h) repeated three HIT sessions (5 × 3 min [100 % $v\dot{V}O_{2max}$]/3 min [50 % $v\dot{V}O_{2max}$]) per week for 4 weeks, they showed early signs of overtraining, with increased subjective ratings of fatigue, muscle soreness and poor sleep quality [162]. When 22 elite junior alpine skiers (peak power output 347 ± 67 W) performed an 11-day 'shock' microcycle, including 15 HIT sessions (4 × 4 min [90–95 % of maximal HR]/3 min [active]), their CMJ performance remained slightly impaired by 5 % (ES −0.5) up to 7 days following the end of the microcycle. This highlights the high neuromuscular demand of such training when HIT sessions are performed with limited recovery.

3.2.2 HIT Selection During a Microcycle

Despite the lack of scientific data related to the effects of HIT sessions on the 'quality' of subsequent training and potential overload and injury risk [8], relevant insight can be gained through discussions with expert coaches who resolve the programming puzzle on a daily basis. Typical examples of HIT programming during different training cycles in international elite athletes are provided for triathlon (Table 4), rowing (Table 5) and team sports (Tables 6, 7). Here, the HIT format is chosen with the other training sessions in mind. For example, depending on the expected metabolic and neuromuscular load, HIT sessions

Table 6 Example of high-intensity interval training programming over three different weekly microcycles for elite soccer (male adults)

Microcycle (physical emphasis)	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Off-season $\dot{V}O_2$ development (generic)							
AM	Injury prevention	Speed (technique)	Soccer	Injury prevention	Power/ speed	Soccer	Rest
	HIT[a]: 2 × 5 min, 10 s [95 % V_{IFT}]/ 10 s [0]	Soccer	HIT[a]: 2 × 4 min 10 s [100 % V_{IFT}]/ 10 s [0]	Soccer	Soccer	HIT[a]: 5 × 3 min, [90 % $V_{Inc.Test}$]/ 2 min [0]	
PM	Soccer	Rest	Soccer	Rest	Soccer	Rest	
	HIT: 2 × 6 min [SSG][b]		HIT: 2 × 4–5 min [SSG][b] 4 versus 4 RPE 7				
	5 versus 5 RPE 6						
Note that during SSG, the number of players is modified with respect to exercise duration (the shorter the SSG, the higher the expected intensity)							
Off-season $\dot{V}O_2$ development (specific)							
AM	Active recovery/ regeneration/injury prevention	Speed (technique)	Soccer	Injury prevention	Power/ speed	Soccer	Rest
	HIT: 2 × 5 min, technical circuit[c] RPE 6	Soccer	HIT: 2 × 4 min technical circuit[c] RPE 7	Soccer	Soccer	HIT: 3–4 × 4 min [SSG][b] 3 versus 3 RPE 8	
PM	Soccer	Rest	Soccer	Rest	Soccer	Rest	
	HIT: 2 × 6 min [SSG][b] 5 versus 5		HIT: 2 × 4–5 min [SSG][b]				
	RPE 6		4 versus 4, RPE 8				
Note that compared with the previous phase, run-based HIT with short intervals (10 s/10 s) is replaced by an intermittent technical circuit with the ball with similar a W/R ratio. The run-based 3-min intervals are replaced by SSG over similar durations							
Competitive (in season)							
AM	Recovery/injury prevention	Soccer[d]	Power/speed	Speed	Agility	Rest	Rest
				Soccer	Soccer		
PM	Rest	Soccer[d]	Rest	Rest	Rest	Game	
		HIT[d]: 3–4 × 3 min [SSG][(b)] 4 versus 4, RPE 8					
The type of SSG can be manipulated based on the content of the Tuesday and Thursday morning sessions (more or less neuromuscular load—based on player numbers; the higher the number, the lower the neuromuscular load)							

[a] All run-based HIT sessions are performed in a straight line and on grass to minimize musculoskeletal load

[b] In addition to player number (which are indicated as # versus #, with the lower the player number, the greater the cardiorespiratory demands), pitch size and playing rules can be altered to manipulate anaerobic glycolytic energy contribution and neuromuscular load (the greater the pitch size, the longer the sprints) [27]

[c] High-intensity running with the ball (e.g. slaloms, repeated passes and shots [28])

[d] The sessions on Tuesday are often performed as one unique session in the morning, especially during the late stages of the competitive season

AM morning, *HIT* high-intensity interval training, *PM* afternoon, *RPE* rating of perceived exertion, *SSG* small-sided games [27], V_{IFT} peak speed reached in the 30–15 Intermittent Fitness Test [179], $V_{Inc.Test}$ peak speed reached during an incremental track test, $\dot{V}O_2$ maximal oxygen uptake, *W/R* work/rest

are separated by 48 (Sect. 3.2.2, Tables 4, 6, 7) to 72 h (Tables 5, 7), and are generally followed by an easy session the following day (i.e. rest or a swim session in triathlon), which might accelerate post-HIT metabolic and neuromuscular recovery [165]. In the case of team handball for example (Table 7), while 2 'recovery' days are scheduled following HIT with long intervals, there is only 1 day of recovery after HIT with short intervals (expected to be less 'lactic', see Sect. 2.1). During the preseason especially, HIT sessions are performed at least 48 h before strength or speed-oriented training sessions, to ensure optimal player freshness in these sessions. During the competitive season, when the strength/power sessions are not programmed in a particular week, RSS sessions are preferred over other HIT formats to compensate for the lack of neuromuscular load (Table 7). During the

Table 7 Example of high-intensity interval training (HIT) programming over four different weekly microcycles for elite handball (male adults)

Physical	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Exclusive $\dot{V}O_2$ development							
AM	Rest	Handball	Generic strength/ injury prevention	Handball	Rest	Handball	Rest
PM	Handball	Handball	Handball	Handball	Handball	Handball	
	HIT (grass)[a]: 5 × 3 min, [90 % $V_{Inc.Test}$]/2 min [0] or HIT (grass)[a]: 2 × 12 min 30 s [85 %V_{IFT}]/30 s [40 % V_{IFT}]			HIT (grass)[a]: 2 × 12 min 15 s [95 % V_{IFT}]/15 s [40 % V_{IFT}]		HIT (grass)[a]: 5 × 3 min 30 s [90 % $V_{Inc.Test}$]/ 2 min [0] HIT	
Note that 2 days are allowed after the 'lactic' session (30 s/30 s), and that the long-bout HIT is programmed before the day off							
$\dot{V}O_2$ and strength/speed development (generic)							
AM	Generic strength/speed/ injury prevention	Rest	Generic power/ speed	Rest	Generic strength/ speed	Handball	Rest
						HIT (grass)[a]: 2 × 10 min	
						20 s [95 % V_{IFT}]/20 s [0]	
PM	Handball	Handball	Handball	Handball	Handball	Rest	
		HIT (grass)[a]: 2 × 10 min 10 s [110 % V_{IFT}]/20 s [0]					
Note the de-emphasis of 'lactic' work on Tuesday's HIT session							
$\dot{V}O_2$ and strength/speed development (specific)							
AM	Specific strength/speed/ injury prevention	Rest	HIT: 10 min 10 s [110 % V_{IFT} shuttle]/20 s[0] + 6 min 10 s [100 % V_{IFT} shuttle]/10 s [0]	Rest	Specific power/ speed	Handball	Rest
PM	Handball	Handball	Handball	Handball	Rest	Handball	
						SIT: 6 × 30 s [all-out shuttle]/ 4 min [0]	
Note that the 'lactic' session is programmed before the day off							
Competitive (in season)							
AM	Recovery/strength/injury prevention	Rest	Rest	Strength/ power/speed	Rest	Rest	Rest
PM	Handball	Handball	Handball	Handball	Handball	Game	
		HIT: 1 × 10 min 15 s [100 % V_{IFT} shuttle]/15 s [0] or (if the power/speed session of Thursday is cancelled): 2 × RSS 6 × 20 m-90° COD/ 25 s [0] with 15 min [handball] between RSS	HIT: 2 × 3–4 min [SSG] 4 versus 4 RPE 7				
Note that the RSS session is performed with 90°COD to reduce glycolytic anaerobic energy release, and is performed only when the speed/power session is missed (to partially compensate for the neuromuscular work at high intensity). SSG can also be selected for their specificity, to lower the anaerobic glycolytic energy contribution 3 days before the game							

[a] When not specified, the running pattern is 'straight-line' and performed indoors

AM morning, *COD* changes of direction, *HIT* high-intensity interval training (running), *PM* afternoon, *RPE* rating of perceived exertion, *RSS* repeated-sprint sequences, *SIT* sprint interval training, *SSG* small-sided games [98], V_{IFT} peak speed reached at the 30–15 Intermittent Fitness Test [179], $V_{Inc.Test}$ peak speed reached during an incremental track test

competitive season, the HIT format selected also includes likely lower anaerobic energy contributions than during the preseason, so as not to compromise muscle glycogen recovery for critical competition play (Tables 6, 7). Comparison of elite soccer (Table 6) and handball (Table 7) is interesting with respect to both training volume and HIT format. While soccer coaches typically put an emphasis on integrated HIT formats (i.e. circuits or small-sided games), handball training content is usually more 'isolated'; an approach comparable to that of Australian football and rugby for example. Note also, that in most team sports, long-interval HIT is typically used during the preseason for its likely cardiopulmonary benefits (Part I [1]), despite its lack of specificity.

Finally, as musculoskeletal injury can unintentionally occur during an HIT session, it is important to consider the total distance ran (at high intensity), the ground surface used and the likely neuromuscular load [18, 105]. As alluded to throughout these reviews, distance ran at high intensity in training is a key determinant of neuromuscular adaptation and performance in endurance-trained athletes [18, 166–168]. To achieve this, endurance coaches tend to opt for high-volume HIT sessions (Tables 4, 5), requiring moderate levels of anaerobic glycolytic energy contribution. The addition of active recovery between intervals and sets can also increase the total distance ran during the session (i.e. Fig. 6, Part I [1]). Thus, Billat et al. [5] suggest the use of 15 s/15 s HIT runs at 100/70 % compared with 90/80 % $v\dot{V}O_{2max}$, despite the fact that both are associated with a similar $T@\dot{V}O_{2max}$. While increasing total weekly running distance can have a protective effect against some injuries (i.e. knees), a high training volume background and a history of previous injuries is another important risk factor [105]. In team sports, where HIT sessions have an essential cardiorespiratory and metabolic objective, coaches tend to favour highly efficient sessions (with a high $T@\dot{V}O_{2max}$/exercise time ratio, see Part I [1]) performed on grass or sand [21] to minimize leg muscle load (Table 7), so that athletes can gain freshness for upcoming tactical, strength and speed sessions (e.g. Part I, Fig. 6 [1]). Further, Gabbett et al. [104] recently reported that greater amounts of high-velocity running during training were associated with an increased risk of lower body soft-tissue injury in team sports; this further supports the use of time-efficient and short HIT sessions (Table 3).

4 Conclusions

Manipulating the acute responses to HIT is important, not only in regard to the middle- to long-term physiological and performance adaptations expected, but also to maximize daily and/or weekly training periodization (Part I, Fig. 1 [1]). With respect to training prescription for any athlete, there is, of course, always 'more than one way to skin the cat' [169]. Our recommendation, however, is that the integration of all training contents within a given microcycle is viewed with the overall 'performance puzzle solution' in mind. Choosing the best solution to such a puzzle might be analogous to how an Admiral goes about deciding which operation is best suited to take out a given military target. While mass destruction weapons might hit all targets at once (i.e., "running hard for a few minutes will do the job"), collateral damage often occurs (analogous to extra fatigue, more injury/illness). Sometimes then, the best solution might involve specific 'US Navy SEAL-(Sea, Air, Land Teams) type' operations, for more specific targeting of the required physiological capacities, but with less risk of collateral damage. Along these lines, HIT sessions should first be specific to the physiological adaptations desired (Part I, Fig. 1 [1]), and not necessarily compulsory to the sport itself. With respect to HIT, following $\dot{V}O_2$ data (e.g. [2, 3, 5]), anaerobic energy contribution and neuromuscular/musculoskeletal load are likely the main secondary variables of interest. In practice, most forms of HIT are effective at stressing the aerobic energy system (both central and peripheral aspects, Part I [1]), and some are associated with a large anaerobic glycolytic energy contribution. It is, however, possible to minimize the anaerobic system participation by using certain forms of HIT, including short intervals (Table 2), and possibly some types of small-sided games, at least for certain sports [27] (not detailed in the present review). When appropriately manipulated, HIT sessions (especially RSS or SIT) can, in contrast, be a powerful stimulus for producing high levels of lactic acid in the blood (Table 2).

In practice, while the magnitude of neuromuscular load during HIT can be modulated through the manipulation of HIT variables (e.g. work intensity or duration, exercise mode/pattern), the responses are highly athlete profile-dependent, with endurance-type athletes showing low levels of acute fatigue and speed decrement, and team sport athletes typically showing high levels of neuromuscular fatigue following HIT. There is likely a bell-shaped relationship between exercise intensity and acute neuromuscular performance responses, with too low ($\leq$85 % $v\dot{V}O_{2max}$) and too high (all-out) an intensity having not enough and acute detrimental effects, respectively (Fig. 5). Using data from the present review, coaches and supporting scientists can choose and balance the level of neuromuscular engagement associated with a given HIT format, based on both the expected training-induced adaptations (either through the HIT session itself or the associated sessions and possible additive effects) and the acute

changes in neuromuscular performance (Table 3). Running pattern (e.g. COD, introduction of jumps during the recovery periods), exercise mode (e.g. cycling, running, bouncing) or ground surfaces (e.g. pavement, synthetic track, grass, sand, treadmill) and terrain (uphill, downhill) also may have direct implications on traumatic and overuse injury risk, and should be chosen for programming based on a risk/benefit approach. Similarly, before programming an HIT training cycle, coaches should also consider that in team sports, physical fitness is unlikely to have the same impact on match running performance for all players, as playing position, systems of play and individual playing styles directly affect the relationship between physical fitness and match running performance [170–173].

5 Future Research

Future research should examine the timecourse of neuromuscular recovery following different forms of HIT over several hours and days, so that practitioners can understand better how to maximize training periodization. The various physiological responses to open (the format that has been the more investigated by researchers) versus closed (the format more used in the field) loop HIT designs should also be examined with respect to programming considerations. Strategies to individualize HIT variables in athletes presenting different neuromuscular profiles should also be considered, with respect to both acute neuromuscular fatigue and long-term performance changes and injury rates [18]. Additionally, although our understanding of the long-term effects of classical HIT formats (i.e. running and cycling based) on cardiopulmonary function and performance is progressing [19, 149, 174, 175], very little is known on the specific effect of neuromuscular-oriented HIT formats (as 'Oregon circuits' performed by endurance athletes [22] or strength-endurance HIT and RSS [26, 176] as performed in team sports) on changes in neuromuscular function during prolonged efforts (e.g. preventing the impairment in running speed during the last portion of an 800-m race or the decreased occurrence of sprints at the end of a game [177, 178]). Whether the repetition of a high neuromuscular load leading to either post-activation potentiation or acute muscle fatigue during consecutive HIT/RSS/SIT sessions can be additive or counterproductive to the potential strength/speed gains reached via isolated sessions is also unclear [26, 121] and warrants further investigation (interference phenomenon and concurrent training [24]).

To further improve the programming of HIT, the impact of time of day, timing within a session and external training contents needs to be considered, as typically most studies are conducted with 'fresh' participants in controlled environments, while in practice, HIT sessions are often performed in a state of accumulated fatigue (end of a team sport session or in the afternoon following an exhaustive morning training session). Understanding the physiological responses to technical/tactical training sessions is also an important aspect of successful training in team sport athletes, so that the most appropriate HIT sessions can be programmed as supplemental sessions, i.e. how does one "best solve the programming puzzle", while adding what is 'missed' during the technical/tactical sessions.

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References

1. Buchheit M, Laursen PB. High-intensity internal training, solutions to the programming puzzle. Part I: cardiopulmonary emphasis. Sports Med. 2013;43(5):313–38.
2. Buchheit M, Kuitunen S, Voss SC, et al. Physiological strain associated with high-intensity hypoxic intervals in highly trained young runners. J Strength Cond Res. 2012;26:94–105.
3. Vuorimaa T, Vasankari T, Rusko H. Comparison of physiological strain and muscular performance of athletes during two intermittent running exercises at the velocity associated with VO_{2max}. Int J Sports Med. 2000;21:96–101.
4. Gastin PB. Energy system interaction and relative contribution during maximal exercise. Sports Med. 2001;31:725–41.
5. Billat LV, Slawinksi J, Bocquet V, et al. Very short (15s–15s) interval-training around the critical velocity allows middle-aged runners to maintain VO_{2max} for 14 minutes. Int J Sports Med. 2001;22:201–8.
6. Tabata I, Irisawa K, Kouzaki M, et al. Metabolic profile of high intensity intermittent exercises. Med Sci Sports Exerc. 1997;29:390–5.
7. Hoff J, Helgerud J. Endurance and strength training for soccer players: physiological considerations. Sports Med. 2004;3:165–80.
8. Bompa TO, Haff GG. Periodization: theory and methodology of training. 5th ed. Champaign: Human Kinetics; 2009.
9. Francis C. Training for speed. Canberra (ACT): Faccioni Speed & Conditioning Consultants; 1997. p. 206.
10. Iaia FM, Bangsbo J. Speed endurance training is a powerful stimulus for physiological adaptations and performance improvements of athletes. Scand J Med Sci Sports. 2010;20 (Suppl. 2):11–23.
11. Jacobs I. Lactate, muscle glycogen and exercise performance in man. Acta Physiol Scand Suppl. 1981;495:1–35.
12. Yeo WK, Paton CD, Garnham AP, et al. Skeletal muscle adaptation and performance responses to once a day versus twice every second day endurance training regimens. J Appl Physiol. 2008;105:1462–70.
13. Krustrup P, Mohr M, Steensberg A, et al. Muscle and blood metabolites during a soccer game: implications for sprint performance. Med Sci Sports Exerc. 2006;38:1165–74.
14. Yeo WK, McGee SL, Carey AL, et al. Acute signalling responses to intense endurance training commenced with low or normal muscle glycogen. Exp Physiol. 2010;95:351–8.
15. Krustrup P, Ortenblad N, Nielsen J, et al. Maximal voluntary contraction force, SR function and glycogen resynthesis during

the first 72 h after a high-level competitive soccer game. Eur J Appl Physiol. 2011;111:2987–95.
16. Stoudemire NM, Wideman L, Pass KA, et al. The validity of regulating blood lactate concentration during running by ratings of perceived exertion. Med Sci Sports Exerc. 1996;28:490–5.
17. Steed J, Gaesser GA, Weltman A. Rating of perceived exertion and blood lactate concentration during submaximal running. Med Sci Sports Exerc. 1994;26:797–803.
18. Bonacci J, Chapman A, Blanch P, et al. Neuromuscular adaptations to training, injury and passive interventions: implications for running economy. Sports Med. 2009;39:903–21.
19. Billat LV. Interval training for performance: a scientific and empirical practice. Special recommendations for middle- and long-distance running. Part I: aerobic interval training. Sports Med. 2001;1:13–31.
20. Hanon C, Lehénaff D, Gajer B. A comparative analysis of two intermittent training sessions aiming at VO_{2max} development in top elite athletes. Proceeding of the 8th European Congress of Sport Science, 9–12 July 2003, Salzburg.
21. Binnie MJ, Peeling P, Pinnington H, et al. Effect of training surface on acute physiological responses following interval training. J Strength Cond Res. 2013; 27:1047–56.
22. Di Michele R, Del Curto L, Merni F. Mechanical and metabolic responses during a high-intensity circuit training workout in competitive runners. J Sports Med Phys Fitness. 2012;52:33–9.
23. Paton CD, Hopkins WG, Cook C. Effects of low- vs. high-cadence interval training on cycling performance. J Strength Cond Res. 2009;23:1758–63.
24. Docherty D, Sporer B. A proposed model for examining the interference phenomenon between concurrent aerobic and strength training. Sports Med. 2000;30:385–94.
25. Blazevich A. Are training velocity and movement pattern important determinants of muscular rate of force development enhancement? Eur J Appl Physiol. 2012;112:3689–91.
26. Buchheit M. Should we be recommending repeated sprints to improve repeated-sprint performance? Sports Med. 2012;42: 169–72.
27. Hill-Haas SV, Dawson B, Impellizzeri FM, et al. Physiology of small-sided games training in football: a systematic review. Sports Med. 2011;41:199–220.
28. Hoff J, Wisloff U, Engen LC, et al. Soccer specific aerobic endurance training. Br J Sports Med. 2002;36:218–21.
29. Cohen J. Statistical power analysis for the behavioral sciences. Hillsdale: Lawrence Erlbaum; 1988. p. 599.
30. Hopkins WG, Marshall SW, Batterham AM, et al. Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc. 2009;41:3–13.
31. Medbo JI, Mohn AC, Tabata I, et al. Anaerobic capacity determined by maximal accumulated O_2 deficit. J Appl Physiol. 1988;64:50–60.
32. Bangsbo J. Quantification of anaerobic energy production during intense exercise. Med Sci Sports Exerc. 1998;30:47–52.
33. Yoshida T. Effect of dietary modifications on lactate threshold and onset of blood lactate accumulation during incremental exercise. Eur J Appl Physiol Occup Physiol. 1984;53: 200–5.
34. Margaria R, Edwards H, Dill DB. The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. Am J Physiol. 1933;106:689–715.
35. Bergman BC, Wolfel EE, Butterfield GE, et al. Active muscle and whole body lactate kinetics after endurance training in men. J Appl Physiol. 1999;87:1684–96.
36. Jacobs I, Kaiser P. Lactate in blood, mixed skeletal muscle, and FT or ST fibres during cycle exercise in man. Acta Physiol Scand. 1982;114:461–6.
37. Beneke R, Leithauser RM, Ochentel O. Blood lactate diagnostics in exercise testing and training. Int J Sports Physiol Perform. 2011;6:8–24.
38. Rampinini E, Sassi A, Azzalin A, et al. Physiological determinants of Yo–Yo intermittent recovery tests in male soccer players. Eur J Appl Physiol. 2008;108:401–9.
39. Dupont G, Berthoin S. Time spent at a high percentage of VO_{2max} for short intermittent runs: active versus passive recovery. Can J Appl Physiol. 2004; 29 Suppl.: S3–S16.
40. Dupont G, Moalla W, Guinhouya C, et al. Passive versus active recovery during high-intensity intermittent exercises. Med Sci Sports Exerc. 2004;36:302–8.
41. Billat LV, Koralsztein JP. Significance of the velocity at VO_{2max} and time to exhaustion at this velocity. Sports Med. 1996;22: 90–108.
42. Hill DW, Rowell AL. Significance of time to exhaustion during exercise at the velocity associated with VO_{2max}. Eur J Appl Physiol Occup Physiol. 1996;72:383–6.
43. Heck H, Mader A, Hess G, et al. Justification of the 4-mmol/l lactate threshold. Int J Sports Med. 1985;6:117–30.
44. Billat LV, Renoux J, Pinoteau J, et al. Validation d'une épreuve maximale de temps limiteà VMA (vitesse maximale aérobie) et à $\cdot VO_{2max}$. Sci Sports. 1994;9:3–12.
45. Demarie S, Koralsztein JP, Billat V. Time limit and time at VO_{2max}, during a continuous and an intermittent run. J Sports Med Phys Fitness. 2000;40:96–102.
46. Midgley AW, McNaughton LR, Carroll S. Physiological determinants of time to exhaustion during intermittent treadmill running at vV(.-)O(2max). Int J Sports Med. 2007;28:273–80.
47. Seiler S, Hetlelid KJ. The impact of rest duration on work intensity and RPE during interval training. Med Sci Sports Exerc. 2005;37:1601–7.
48. Smith TP, Coombes JS, Geraghty DP. Optimising high-intensity treadmill training using the running speed at maximal O(2) uptake and the time for which this can be maintained. Eur J Appl Physiol. 2003;89:337–43.
49. Stepto NK, Martin DT, Fallon KE, et al. Metabolic demands of intense aerobic interval training in competitive cyclists. Med Sci Sports Exerc. 2001;33:303–10.
50. Seiler S, Sjursen JE. Effect of work duration on physiological and rating scale of perceived exertion responses during self-paced interval training. Scand J Med Sci Sports. 2004;14:318–25.
51. Rusko H, Nummela A, Mero A. A new method for the evaluation of anaerobic running power in athletes. Eur J Appl Physiol. 1993;66:97–101.
52. Belcastro AN, Bonen A. Lactic acid removal rates during controlled and uncontrolled recovery exercise. J Appl Physiol. 1975;39:932–6.
53. Ahmaidi S, Granier P, Taoutaou Z, et al. Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. Med Sci Sports Exerc. 1996;28: 450–6.
54. Brooks GA. Current concepts in lactate exchange. Med Sci Sports Exerc. 1991;23:895–906.
55. Slawinski J, Dorel S, Hug F, et al. Elite long sprint running: a comparison between incline and level training sessions. Med Sci Sports Exerc. 2008;40:1155–62.
56. Astrand I, Astrand PO, Christensen EH, et al. Intermittent muscular work. Acta Physiol Scand. 1960;48:448–53.
57. Astrand I, Astrand PO, Christensen EH, et al. Myohemoglobin as an oxygen-store in man. Acta Physiol Scand. 1960;48: 454–60.
58. Christensen EH, Hedman R, Saltin B. Intermittent and continuous running. (A further contribution to the physiology of intermittent work). Acta Physiol Scand. 1960;50:269–86.

59. Pennisi E. In nature, animals that stop and start win the race. Science. 2000;288:83–5.
60. Billat VL, Slawinski J, Bocquet V, et al. Intermittent runs at the velocity associated with maximal oxygen uptake enables subjects to remain at maximal oxygen uptake for a longer time than intense but submaximal runs. Eur J Appl Physiol. 2000;81:188–96.
61. Dupont G, Blondel N, Lensel G, et al. Critical velocity and time spent at a high level of VO_2 for short intermittent runs at supramaximal velocities. Can J Appl Physiol. 2002;27:103–15.
62. Rozenek R, Funato K, Kubo J, et al. Physiological responses to interval training sessions at velocities associated with VO_{2max}. J Strength Cond Res. 2007;21:188–92.
63. Bisciotti GN. L'incidenza fisiologica dei parametri di durata, intensità e recupero nell'ambito dell'allenamento intermittente. Sienza di Sport. 2004: 90–6.
64. Buchheit M, Laursen PB, Millet GP, et al. Predicting intermittent running performance: critical velocity versus endurance index. Int J Sports Med. 2007;29:307–15.
65. Thevenet D, Leclair E, Tardieu-Berger M, et al. Influence of recovery intensity on time spent at maximal oxygen uptake during an intermittent session in young, endurance-trained athletes. J Sports Sci. 2008;26:1313–21.
66. Thevenet D, Tardieu-Berger M, Berthoin S, et al. Influence of recovery mode (passive vs. active) on time spent at maximal oxygen uptake during an intermittent session in young and endurance-trained athletes. Eur J Appl Physiol. 2007;99:133–42.
67. Dupont G, Blondel N, Berthoin S. Performance for short intermittent runs: active recovery vs. passive recovery. Eur J Appl Physiol. 2003;89:548–54.
68. Christmass MA, Dawson B, Arthur PG. Effect of work and recovery duration on skeletal muscle oxygenation and fuel use during sustained intermittent exercise. Eur J Appl Physiol Occup Physiol. 1999;80:436–47.
69. Christmass MA, Dawson B, Passeretto P, et al. A comparison of skeletal muscle oxygenation and fuel use in sustained continuous and intermittent exercise. Eur J Appl Physiol. 1999;80:423–35.
70. Harris RC, Edwards RH, Hultman E, et al. The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. Pflugers Arch. 1976;367:137–42.
71. Dellal A, Keller D, Carling C, et al. Physiologic effects of directional changes in intermittent exercise in soccer players. J Strength Cond Res. 2010;24:3219–26.
72. Osgnach C, Poser S, Bernardini R, et al. Energy cost and metabolic power in elite soccer: a new match analysis approach. Med Sci Sports Exerc. 2010;42:170–8.
73. Haydar B. Al Haddad H, Buchheit M. Assessing inter-efforts recovery and change of direction abilities with the 30–15 Intermittent Fitness Test. J Sports Sci Med. 2011;10:346–54.
74. Ahmaidi S, Collomp K, Prefaut C. The effect of shuttle test protocol and the resulting lactacidaemia on maximal velocity and maximal oxygen uptake during the shuttle exercise test. Eur J Appl Physiol. 1992;65:475–9.
75. Buchheit M, Haydar B, Hader K, et al. Assessing running economy during field running with changes of direction: application to 20-m shuttle-runs. Int J Sports Physiol Perform. 2011;6:380–95.
76. Buchheit M. Individualizing high-intensity interval training in intermittent sport athletes with the 30-15 Intermittent Fitness Test. NSCA Hot Topic Series [online]. Available from URL: www.nsca-lift.org. 2011; November. Accessed 2013 May 21.
77. Cometti G, Jaffiol T, Chalopin C, et al. Etude des effets de différentes séquences de travail de type intermittent sur la fréquence cardiaque, la lactatémie, et la détente [online]. Available from URL: http://expertise-performance.u-bourgogne.fr/pdf/lactate.pdf. Accessed 2002 Jan 19.
78. Balsom PD, Seger JY, Sjodin B, et al. Physiological responses to maximal intensity intermittent exercise. Eur J Appl Physiol Occup Physiol. 1992;65:144–9.
79. Abt G, Siegler JC, Akubat I, et al. The effects of a constant sprint-to-rest ratio and recovery mode on repeated sprint performance. J Strength Cond Res. 2011;25:1695–702.
80. Little T, Williams AG. Effects of sprint duration and exercise: rest ratio on repeated sprint performance and physiological responses in professional soccer players. J Strength Cond Res. 2007;21:646–8.
81. Buchheit M, Haydar B, Ahmaidi S. Repeated sprints with directional changes: do angles matter? J Sports Sci. 2012;30:555–62.
82. Dupont G, Millet GP, Guinhouya C, et al. Relationship between oxygen uptake kinetics and performance in repeated running sprints. Eur J Appl Physiol. 2005;95:27–34.
83. Buchheit M. Performance and physiological responses to repeated- sprint and jump sequences. Eur J Appl Physiol. 2010;101:1007–18.
84. Nakamura FY, Soares-Caldeira LF, Laursen PB, et al. Cardiac autonomic responses to repeated shuttle sprints. Int J Sports Med. 2009;30:808–13.
85. Buchheit M, Laursen PB, Ahmaidi S. Parasympathetic reactivation after repeated sprint exercise. Am J Physiol Heart Circ Physiol. 2007;293:H133–41.
86. Balsom PD, Seger JY, Sjodin B, et al. Maximal-intensity intermittent exercise: effect of recovery duration. Int J Sports Med. 1992;13:528–33.
87. Buchheit M, Cormie P, Abbiss CR, et al. Muscle deoxygenation during repeated sprint running: effect of active vs. passive recovery. Int J Sports Med. 2009;30:418–25.
88. Castagna C, Abt G, Manzi V, et al. Effect of recovery mode on repeated sprint ability in young basketball players. J Strength Cond Res. 2008;22:923–9.
89. Gorostiaga EM, Asiain X, Izquierdo M, et al. Vertical jump performance and blood ammonia and lactate levels during typical training sessions in elite 400-m runners. J Strength Cond Res. 2010;24:1138–49.
90. Buchheit M, Bishop D, Haydar B, et al. Physiological responses to shuttle repeated-sprint running. Int J Sport Med. 2010;31:402–9.
91. Bogdanis GC, Nevill ME, Boobis LH, et al. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. J Physiol. 1995;482(Pt 2):467–80.
92. McCartney N, Spriet LL, Heigenhauser GJ, et al. Muscle power and metabolism in maximal intermittent exercise. J Appl Physiol. 1986;60:1164–9.
93. Gaitanos GC, Williams C, Boobis LH, et al. Human muscle metabolism during intermittent maximal exercise. J Appl Physiol. 1993;75:712–9.
94. Putman CT, Jones NL, Lands LC, et al. Skeletal muscle pyruvate dehydrogenase activity during maximal exercise in humans. Am J Physiol. 1995;269:E458–68.
95. Buchheit M, Duthie G, Ahmaidi S. Increasing passive recovery duration leads to greater performance despite higher blood lactate accumulation and physiological strain during repeated shuttle 30-s sprints. Proceeding of the 14th European Congress of Sport Science. 24–27Jun 2009, Olso, Norway.
96. Bogdanis GC, Nevill ME, Lakomy HK, et al. Effects of active recovery on power output during repeated maximal sprint cycling. Eur J Appl Physiol Occup Physiol. 1996;74:461–9.
97. Buchheit M, Abbiss C, Peiffer JJ, et al. Performance and physiological responses during a sprint interval training session:

relationships with muscle oxygenation and pulmonary oxygen uptake kinetics. Eur J Appl Physiol. 2012;111:767–79.
98. Buchheit M, Lepretre PM, Behaegel AL, et al. Cardiorespiratory responses during running and sport-specific exercises in handball players. J Sci Med Sport. 2009;12:399–405.
99. Ross A, Leveritt M, Riek S. Neural influences on sprint running: training adaptations and acute responses. Sports Med. 2001;31:409–25.
100. Bishop PA, Jones E, Woods AK. Recovery from training: a brief review. J Strength Cond Res. 2008;22:1015–24.
101. Coffey V, Hawley J. The molecular bases of training adaptation. Sports Med. 2007;37:737–63.
102. Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Med. 2012;42:209–26.
103. Small K, McNaughton LR, Greig M, et al. Soccer fatigue, sprinting and hamstring injury risk. Int J Sports Med. 2009;30:573–8.
104. Gabbett TJ, Ullah S. Relationship between running loads and soft-tissue injury in elite team sport athletes. J Strength Cond Res. 2012;26:953–60.
105. van Gent RN, Siem D, van Middelkoop M, et al. Incidence and determinants of lower extremity running injuries in long distance runners: a systematic review. Br J Sports Med. 2007;41:469–80.
106. Perrey S, Racinais S, Saimouaa K, et al. Neural and muscular adjustments following repeated running sprints. Eur J Appl Physiol. 2010; 109:1027–36.
107. Lattier G, Millet GY, Martin A, et al. Fatigue and recovery after high-intensity exercise part I: neuromuscular fatigue. Int J Sports Med. 2004;25:450–6.
108. Vuorimaa T, Virlander R, Kurkilahti P, et al. Acute changes in muscle activation and leg extension performance after different running exercises in elite long distance runners. Eur J Appl Physiol. 2006;96:282–91.
109. Skof B, Strojnik V. Neuro-muscular fatigue and recovery dynamics following anaerobic interval workload. Int J Sports Med. 2006;27:220–5.
110. Girard O, Bishop DJ, Racinais S. Neuromuscular adjustments of the quadriceps muscle after repeated cycling sprints. PLoS One. 2013;8:e61793.
111. Mendez-Villanueva A, Edge J, Suriano R, et al. The recovery of repeated-sprint exercise is associated with PCr resynthesis, while muscle pH and EMG amplitude remain depressed. PLoS One. 2012;7:e51977.
112. Girard O, Mendez-Villanueva A, Bishop D. Repeated-sprint ability—part I: factors contributing to fatigue. Sports Med. 2011;41:673–94.
113. Fernandez-Del-Olmo M, Rodriguez FA, Marquez G, et al. Isometric knee extensor fatigue following a Wingate test: peripheral and central mechanisms. Scand J Med Sci Sports. 2013;23:57–65.
114. Enoka RM, Stuart DG. Neurobiology of muscle fatigue. J Appl Physiol. 1992; 72:1631–48.
115. Girard O, Micallef JP, Millet GP. Changes in spring-mass model characteristics during repeated running sprints. Eur J Appl Physiol. 2011;111:125–34.
116. Paavolainen L, Hakkinen K, Nummela A, et al. Neuromuscular characteristics and fatigue in endurance and sprint athletes during a new anaerobic power test. Eur J Appl Physiol Occup Physiol. 1994;69:119–26.
117. Buchheit M, Spencer M, Ahmaidi S. Reliability, usefulness and validity of a repeated sprint and jump ability test. Int J Sports Physiol Perform. 2010;5:3–17.
118. Bosco C, Komi PV, Tihanyi J, et al. Mechanical power test and fiber composition of human leg extensor muscles. Eur J Appl Physiol Occup Physiol. 1983;51:129–35.
119. Garrandes F, Colson SS, Pensini M, et al. Neuromuscular fatigue profile in endurance-trained and power-trained athletes. Med Sci Sports Exerc. 2007;39:149–58.
120. Vuorimaa T, Hakkinen K, Vahasoyrinki P, et al. Comparison of three maximal anaerobic running test protocols in marathon runners, middle-distance runners and sprinters. Int J Sports Med. 1996;17(Suppl 2):S109–13.
121. Hodgson M, Docherty D, Robbins D. Post-activation potentiation: underlying physiology and implications for motor performance. Sports Med. 2005;35:585–95.
122. Bishop D, Spencer M. Determinants of repeated-sprint ability in well-trained team-sport athletes and endurance-trained athletes. J Sports Med Phys Fitness. 2004;44:1–7.
123. Ispirlidis I, Fatouros IG, Jamurtas AZ, et al. Time-course of changes in inflammatory and performance responses following a soccer game. Clin J Sport Med. 2008;18:423–31.
124. Andersson H, Raastad T, Nilsson J, et al. Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. Med Sci Sports Exerc. 2008;40:372–80.
125. Palmer CD, Sleivert GG. Running economy is impaired following a single bout of resistance exercise. J Sci Med Sport. 2001;4:447–59.
126. Nummela A, Vuorimaa T, Rusko H. Changes in force production, blood lactate and EMG activity in the 400-m sprint. J Sports Sci. 1992;10:217–28.
127. Paavolainen L, Nummela A, Rusko H. Muscle power factors and VO_{2max} as determinants of horizontal and uphill running performance. Scand J Med Sci Sports. 2000;10:286–91.
128. Gottschall JS, Kram R. Ground reaction forces during downhill and uphill running. J Biomech. 2005;38:445–52.
129. van Beijsterveldt AM, van de Port IG, Vereijken AJ, et al. Risk Factors for Hamstring injuries in male soccer players: a systematic review of prospective studies. Scand J Med Sci Sports. 2013;23:253–62.
130. Byrnes WC, Clarkson PM, White JS, et al. Delayed onset muscle soreness following repeated bouts of downhill running. J Appl Physiol. 1985;59:710–5.
131. Gollnick PD, Piehl K, Saltin B. Selective glycogen depletion pattern in human muscle fibres after exercise of varying intensity and at varying pedalling rates. J Physiol. 1974;241:45–57.
132. Altenburg TM, Degens H, van Mechelen W, et al. Recruitment of single muscle fibers during submaximal cycling exercise. J Appl Physiol. 2007;103:1752–6.
133. Higashihara A, Ono T, Kubota J, et al. Functional differences in the activity of the hamstring muscles with increasing running speed. J Sports Sci. 2010;28:1085–92.
134. di Prampero PE, Fusi S, Sepulcri L, et al. Sprint running: a new energetic approach. J Exp Biol. 2005;208:2809–16.
135. Brughelli M, Cronin J, Levin G, et al. Understanding change of direction ability in sport: a review of resistance training studies. Sports Med. 2008;38:1045–63.
136. Oliver JL. Is a fatigue index a worthwhile measure of repeated sprint ability? J Sci Med Sport. 2009;12:20–3.
137. Lakomy J, Haydon DT. The effects of enforced, rapid deceleration on performance in a multiple sprint test. J Strength Cond Res. 2004;18:579–83.
138. Mendez-Villanueva A, Hamer P, Bishop D. Fatigue responses during repeated sprints matched for initial mechanical output. Med Sci Sports Exerc. 2007;39:2219–25.
139. Bravo DF, Impellizzeri FM, Rampinini E, et al. Sprint vs. interval training in football. Int J Sports Med. 2008;29:668–74.

140. Buchheit M, Millet GP, Parisy A, et al. Supramaximal training and post-exercise parasympathetic reactivation in adolescents. Med Sci Sports Exerc. 2008;40:362–71.
141. Buchheit M, Mendez-Villanueva A, Delhomel G, et al. Improving repeated sprint ability in young elite soccer players: repeated sprints vs. explosive strength training. J Strength Cond Res. 2010;24:2715–22.
142. Tomazin K, Morin JB, Strojnik V, et al. Fatigue after short (100-m), medium (200-m) and long (400-m) treadmill sprints. Eur J Appl Physiol. 2012;112:1027–36.
143. Buchheit M, Mendez-Villanueva A, Quod MJ, et al. Improving acceleration and repeated sprint ability in well-trained adolescent handball players: speed vs. sprint interval training. Int J Sports Physiol Perform. 2010;5:152–64.
144. Gibala MJ, McGee SL. Metabolic adaptations to short-term high-intensity interval training: a little pain for a lot of gain? Exerc Sport Sci Rev. 2008;36:58–63.
145. Omeyer C, Buchheit M. Vertical jump performance in response to different high-intensity running sessions [Master Thesis]. Faculté des sciences du sport (STAPS), Strasbourg, 2002.
146. Rusko HK, Tikkanen HO, Peltonen JE. Altitude and endurance training. J Sports Sci. 2004; 22: 928–44; discussion 45.
147. Hreljac A. Impact and overuse injuries in runners. Med Sci Sports Exerc. 2004;36:845–9.
148. Billat V, Binsse V, Petit B, et al. High level runners are able to maintain a VO_2 steady-state below VO_{2max} in an all-out run over their critical velocity. Arch Physiol Biochem. 1998;106:38–45.
149. Midgley AW, McNaughton LR, Wilkinson M. Is there an optimal training intensity for enhancing the maximal oxygen uptake of distance runners?: empirical research findings, current opinions, physiological rationale and practical recommendations. Sports Med. 2006;36:117–32.
150. Thibault G. A graphical model for interval training. IAAF New Studies in Athletics. 2003;18:49–55.
151. Hautala AJ, Kiviniemi AM, Tulppo MP. Individual responses to aerobic exercise: the role of the autonomic nervous system. Neurosci Biobehav Rev. 2009;33:107–15.
152. James DV, Barnes AJ, Lopes P, et al. Heart rate variability: response following a single bout of interval training. Int J Sports Med. 2002;23:247–51.
153. Mourot L, Bouhaddi M, Perrey S, et al. Decrease in heart rate variability with overtraining: assessment by the Poincare plot analysis. Clin Physiol Funct Imag. 2004;24:10–8.
154. Niewiadomski W, Gasiorowska A, Krauss B, et al. Suppression of heart rate variability after supramaximal exertion. Clin Physiol Funct Imag. 2007;27:309–19.
155. Buchheit M, Laursen PB, Al Haddad H, et al. Exercise-induced plasma volume expansion and post-exercise parasympathetic reactivation. Eur J Appl Physiol. 2009; 105:471–81.
156. Kiviniemi AM, Hautala AJ, Kinnunen H, et al. Daily exercise prescription based on heart rate variability among men and women. Med Sci Sports Exerc. 2009.
157. Kiviniemi AM, Hautala AJ, Kinnunen H, et al. Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol. 2007;101:743–51.
158. Al Haddad H, Laursen PB, Ahmaidi S, et al. Nocturnal heart rate variability following supramaximal intermittent exercise. Int J Sports Physiol Perform. 2009;4:435–47.
159. Seiler S, Haugen O, Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. Med Sci Sports Exerc. 2007;39:1366–73.
160. Hautala AJ, Tulppo MP, Makikallio TH, et al. Changes in cardiac autonomic regulation after prolonged maximal exercise. Clin Physiol. 2001;21:238–45.
161. Buchheit M, Voss SC, Nybo L, et al. Physiological and performance adaptations to an in-season soccer camp in the heat: associations with heart rate and heart rate variability. Scand J Med Sci Sports. 2011;21:e477–85.
162. Billat VL, Flechet B, Petit B, et al. Interval training at VO_{2max}: effects on aerobic performance and overtraining markers. Med Sci Sports Exerc. 1999;31:156–63.
163. Breil FA, Weber SN, Koller S, et al. Block training periodization in alpine skiing: effects of 11-day HIT on VO_{2max} and performance. Eur J Appl Physiol. 2010;109:1077–86.
164. Issurin V. Block periodization versus traditional training theory: a review. J Sports Med Phys Fitness. 2008;48:65–75.
165. Lum D, Landers G, Peeling P. Effects of a recovery swim on subsequent running performance. Int J Sports Med. 2010;31:26–30.
166. Noakes T. Lore of running. Oxford University Press Southern African ed. Oxford. Champaign (IL): Leisure Press; 1991. p. 450.
167. Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the VO_{2max} test predicts running performance. J Sports Sci. 1990;8:35–45.
168. Paavolainen LM, Nummela AT, Rusko HK. Neuromuscular characteristics and muscle power as determinants of 5-km running performance. Med Sci Sports Exerc. 1999;31:124–30.
169. Laursen PB. Training for intense exercise performance: high-intensity or high-volume training? Scand J Med Sci Sports. 2010;20(Suppl 2):1–10.
170. Buchheit M, Mendez-Villanueva A, Simpson BM, et al. Match running performance and fitness in youth soccer. Int J Sports Med. 2010;31:818–25.
171. Mendez-Villanueva A, Buchheit M, Simpson BM, et al. Match play intensity distribution in youth soccer. Int J sport Med. 2013;34:101–10.
172. Mooney M, O'Brien B, Cormack S, et al. The relationship between physical capacity and match performance in elite Australian football: a mediation approach. J Sci Med Sport. 2011;14:447–52.
173. Buchheit M, Simpson BM, Mendez-Villaneuva A. Repeated high-speed activities during youth soccer games in relation to changes in maximal sprinting and aerobic speeds. Int J sport Med. 2013; 34(1):40–8.
174. Laursen PB, Shing CM, Peake JM, et al. Interval training program optimization in highly trained endurance cyclists. Med Sci Sports Exerc. 2002;11:1801–7.
175. Midgley AW, McNaughton LR, Jones AM. Training to enhance the physiological determinants of long-distance running performance: can valid recommendations be given to runners and coaches based on current scientific knowledge? Sports Med. 2007;37:857–80.
176. Bishop D, Girard O, Mendez-Villanueva A. Repeated-sprint ability. Part II: recommendations for training. Sports Med. 2011;41:741–56.
177. Buchheit M, Mendez-Villanueva A, Simpson BM, et al. Repeated-sprint sequences during youth soccer matches. Int J Sport Med. 2010;31:709–16.
178. Carling C, Le Gall F, Dupont G. Analysis of repeated high-intensity running performance in professional soccer. J Sports Sci. 2012;30:325–36.
179. Buchheit M. The 30–15 Intermittent Fitness Test: accuracy for individualizing interval training of young intermittent sport players. J Strength Cond Res. 2008;22:365–74.
180. Sassi A, Stefanescu A, Menaspa P, et al. The cost of running on natural grass and artificial turf surfaces. J Strength Cond Res. 2011;25:606–11.
181. Gains GL, Swedenhjelm AN, Mayhew JL, et al. Comparison of speed and agility performance of college football players on field turf and natural grass. J Strength Cond Res. 2010;24:2613–7.

182. Abbiss CR, Karagounis LG, Laursen PB, et al. Single-leg cycle training is superior to double-leg cycling in improving the oxidative potential and metabolic profile of trained skeletal muscle. J Appl Physiol. 2011;110:1248–55.
183. Plews DJ, Laursen PB, Kilding AE, et al. Heart rate variability in elite triathletes, is variation in variability the key to effective training? A case comparison. Epub: Eur J Appl Physiol; 2012.
184. Carter H, Jones AM, Barstow TJ, et al. Oxygen uptake kinetics in treadmill running and cycle ergometry: a comparison. J Appl Physiol. 2000;89:899–907.