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Human Kinetics
BRIEF REVIEW

The Temporal Relationship Between Exercise, Recovery Processes, and Changes in Performance

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Physiological and psychological demands during training and competition generate fatigue and reduce an athlete's sport-specific performance capacity. The magnitude of this decrement depends on several characteristics of the exercise stimulus (eg, type, duration, and intensity), as well as on individual characteristics (eg, fitness, profile, and fatigue resistance). As such, the time required to fully recover is proportional to the level of fatigue, and the consequences of exercise-induced fatigue are manifold. Whatever the purpose of the ensuing exercise session (ie, training or competition), it is crucial to understand the importance of optimizing the period between exercise bouts in order to speed up the regenerative processes and facilitate recovery or set the next stimulus at the optimal time point. This implies having a fairly precise understanding of the fatigue mechanisms that contribute to the performance decrement. Failing to respect an athlete's recovery needs may lead to an excessive accumulation of fatigue and potentially "nonfunctional overreaching" or to maladaptive training. Although research in this area recently increased, considerations regarding the specific time frames for different physiological mechanisms in relation to exercise-induced fatigue are still missing. Furthermore, recommendations on the timing and dosing of recovery based on these time frames are limited. Therefore, the aim of this article is to describe time courses of recovery in relation to the exercise type and on different physiological levels. This summary supports coaches, athletes, and scientists in their decision-making process by considering the relationship of exercise type, physiology, and recovery.

Keywords: metabolism, sport physiology, training, exercise physiology, exercise performance

Each physical training session provides athletes with physiological stress, leading to disturbances in homeostasis of a number of physiological processes and biochemical pathways. In response to this stress, the body triggers postexercise adaptive reactions that counteract these physiological and biochemical changes, emphasizing the necessity for an adequate recovery period after the training bout. For the purposes of this commentary, fatigue is defined as a state in which physical and/or mental performance is reduced as a result of these disturbances.[1] Similarly, recovery is defined as the set of processes resulting in an athlete's renewed ability to meet or exceed previous performance levels, and the recovery period is defined as the time necessary for the various physiological parameters that were altered by exercise to return to resting values.[1]

A primary responsibility for scientists and coaches is to understand and manage the fatigue that athletes encounter in training and competition. To achieve this, a variety of data regarding the training load and the athlete's responses to these loads are often used to estimate the individual state of recovery and/or the need for recovery interventions. However, various physiological and biochemical systems require different recovery periods, which is important to consider when scheduling a specific type and timing of recovery interventions (eg, cold-water immersion [CWI], compression garments, or massage). Furthermore, the magnitude of fatigue in these systems depends on the type/mode, duration, and intensity of the previous exercise. It is noteworthy that available monitoring parameters are usually reflecting only one physiological aspect of fatigue and recovery, and therefore, their time course might deviate from each other. Therefore, an understanding of the mechanisms driving recovery is important to estimate if a specific intervention is adequate to enhance performance recovery.

Furthermore, throughout their preparation process, athletes periodize their training to maximize adaptations. This may include specific periods of (intended) insufficient recovery and/or periods of (functional) "overload" or "overreaching."[2] These alterations in the training stimulus highlight the need to adapt recovery time frames and interventions according to the specific period, the demands of training, and the specific needs of the athlete. Failing to meet an athlete's recovery needs may lead to an excessive accumulation of fatigue, resulting in reduced training tolerance and performance, increased risk of illness and injury, as well as cognitive and mood disturbances.

The aim of this overview is to provide a framework for coaches, athletes, and sport scientists to differentiate physiological load-adaptation pathways (cardiocirculatory, metabolic, neuromuscular, and central) and their different time course of action. Specifically, this review shall provide a physiology-based rationale for an appropriate use of recovery (interventions) in different training and competition scenarios.

Time Course of Physiological Recovery

The overall aim of training is to elicit the desired physiological (and other) adaptations by targeted stimuli.[3] A training session generally results in stress and an acute disturbance of the homeostasis. This triggers adaptations that underpin athletic performance through

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increased endurance, speed, strength, and/or power. An acute consequence of the physical demand of training is fatigue, manifesting in negative physiological, functional, and perceptual outcomes, which can be present up to several days postexercise. Regardless of the purpose of the ensuing exercise session (ie, training or competition), it is crucial to understand the importance of optimizing the recovery between training bouts in order to expedite the regenerative processes and facilitate recovery. After a training session, these acute responses are reverted to (or beyond) their initial state. However, in the context of physiology, exercise-induced fatigue and subsequent recovery integrates alterations in many different systems, each following a different time course, depending on the exercise mode, duration, and intensity.[4] Specific resistance-training stimulus (eg, to a strength–power stimulus) fatigues the neuromuscular system specific to strength–power stimuli, whereas a specific endurance training (partly) exhausts the aerobic energy production system. However, it is important to understand that any exercise training will challenge both metabolic and neuromuscular systems at different levels depending on its specific content.[5] It seems logical that the type and amount of postexercise fatigue varies between the physiological systems, depending on the previously mentioned variables. For example, in endurance sports, an increase in external load (eg, speed, power) leads to an increase in metabolic cost, requiring augmented provision of fuel substrates and oxygen, challenging the cardiorespiratory and the metabolic systems.[6] In addition to these types of load stimuli, team-sport athletes are also challenged with accelerations and decelerations inducing a mechanical load component, which needs to be considered when evaluating the need for recovery.[7]

The physical demands of the exercise influence the magnitude of fatigue, the systems involved, and the need for (optimal) recovery. In addition, fatigue-induced changes seem to vary within and between individuals.[8–10] Hence, the definition of one specific time frame for postexercise recovery is difficult as recovery time courses most likely differ within and between the physiological systems of the human body.[8] This implies the need for an understanding of the fatigue mechanisms that contribute to the performance decrement as well as their time course to recover.[11]

Metabolic Recovery

The elevated energy expenditure caused by training and competition reduces substrate availability, which can decrease performance capacity. In high-intensity exercise, any decrease in substrate availability has the potential to decrease performance. For example, in brief, during high-intensity exercise, phosphocreatine stores decrease very rapidly reducing the force-generating capacity.[12] However, given the speed of its recovery kinetics,[12] phosphocreatine stores may be a limiting factor when short recovery periods are available (ie, the recovery between repetitions or sets within the same training session), but it is not considered as a (limiting) factor in recovery for competition or training bouts separated by hours or days. Conversely, muscle glycogen resynthesis is much slower and may continue for 2 to 3 days after high-intensity training.[11,13] While carbohydrate refueling can enhance immediate postexercise recovery, it can still take approximately 24 hours to normalize stores after substantial levels of depletion, irrespective of carbohydrate intake.[13,14] The repeated stimulus of anaerobic glycolysis during high-intensity exercise is associated with an accumulation of metabolic by-products such as lactic acid. One of the earliest hypotheses regarding peripheral fatigue purported a link between lactic acid, muscle/blood acidosis, and alterations in metabolic and muscle functions. This hypothesis is still incorrectly used to explain fatigue, despite evidence showing no causative effect in acute muscle fatigue.[15] It is not within the scope of this review to discuss this hypothesis extensively; however, the literature of the past decades clearly shows that (peripheral) fatigue is much more complex. While experiments on isolated muscle suggest that acidosis has little detrimental effect[15] and may even improve performance in some conditions,[16] whole-body experiments are less conclusive and suggest that acidosis may act as an exacerbator of other mechanisms of fatigue involving contractile and metabolic processes, or the brain and the central nervous system (CNS).[15,17] If some pieces of the puzzle still remain to be elucidated, the rate of postexercise lactic acid disappearance or pH normalization suggests that it may contribute to the mechanisms involved in immediate or short-term recovery (seconds to minutes), but not in long-term recovery (hours and days).

Athletes are regularly exposed to oxidative stress during training and competition, which increases reactive oxygen and nitrogen species (RONS). Mitochondrial oxygen consumption, circulating catecholamines, eccentric muscle contraction, and inflammatory responses seem to influence RONS production during and after exercise.[18] Prior oxidative damage caused by intensive training periods might compromise recovery as well as exercise performance.[18] However, Ascensão et al[18] could not find a relationship between oxidative stress, markers of muscle damage, and performance after a soccer match,[18] questioning the link between oxidative stress and performance recovery. Indeed, a growing body of literature suggests that free radicals might act as signaling molecules, specifically activating redox-sensitive transcription factors, which are necessary for muscle regeneration and adaptation following damage.[19] For example, Close et al[19] reported delayed recovery of muscle function when RONS was suppressed by vitamin C supplementation following downhill running.[19] These authors suggest that RONS produced in the days following muscle-damaging exercise might enhance recovery and that redox-regulated transcription factors might be necessary for optimal adaptation.[19] Therefore, the determination of their blood concentrations might not indicate fatigue (or recovery).

Neuromuscular Recovery

Peripheral fatigue refers to decrements in neuromuscular function, for example, disruptions of the potential propagation, excitation–contraction coupling, or cross-bridging cycling, despite unchanged or increased neural drive.[20] Mechanical disruptions to the muscle fiber are task dependent and related to the volume of high-intensity contractions (eg, in particular decelerations/accelerations and/or direction changes).[21] The resulting delayed onset muscle soreness can be associated with elevated levels of membrane damage markers (eg, creatine kinase) and systematic inflammation indicators like C-reactive protein. This may be accompanied by decreases of both the maximal voluntary contraction force and the joint range of motion.[13] However, the time required to fully recover is related to the muscle mass involved and the intensity of the exercise and ranges from 24 to 96 hours.[11] Since the ability of the muscle to generate high levels of forces is involved in many facets of athletic performance, the consequences of exercise-induced neuromuscular fatigue are manifold.

Many recovery interventions focus on limiting postexercise disturbances and inflammatory events within the exercised muscle cell.[13] However, a dissociated time course of recovery has been reported between inflammation markers and muscular performance, with research showing neuromuscular force returning

to baseline even though blood-based markers of exercise-induced muscle damage are still elevated.[13] For example, inflammation markers (eg, interleukin-6, C-reactive protein, creatine kinase) were shown to peak between 24 and 48 hours following a soccer match [11,18,22] with a return to baseline between 48 and 120 hours following, depending on the magnitude of the peak. By contrast, neuromuscular function (eg, maximal voluntary contraction force, vertical jump height, and/or sprint speed) has been shown to return to baseline between 5 and 96 hours postmatch.[11,22,23] The most likely reason for this are the blood kinetics of these biochemical parameters, which are more dependent on their degradation following an exercise stimulus. In this context, it is notable that inflammation markers are not closely associated with perceived muscle soreness, whereas perceptual recovery is closely related to performance recovery.[13] It might be that the uncoupling of the inflammatory markers and perceptions of soreness that follows muscle damage is due to changes in the kinetics of the degradation of the blood-borne inflammatory markers.

Central Recovery

During exercise and fatigue, changes at all levels of the nervous system, including the brain, spinal cord, motor output, sensory input, and autonomic function occur. The mix of influences and the importance of their contribution vary with the type of exercise being performed.[24] The complexity of CNS functioning and the possible influence of the feedback mechanism from the muscle make it difficult to identify the exact locus of CNS fatigue, and thus how and in which site recovery takes place. Although the underlying mechanisms are not yet understood, it has been suggested that central fatigue might be related to a change in the synthesis and metabolism of brain monoamines, such as serotonin, dopamine, and noradrenaline.[25] These neurotransmitters play a key role in the modulation of various brain functions such as motivation, arousal, attention, stress responses, and motor control.[26] Microdialysis experiments in rodents observed that most neurotransmitter systems return to baseline levels within a few hours after intensive and/or exhaustive treadmill exercise.[27]

When there is an insufficient output from the motor cortex, this deficit is usually called supraspinal or cortical fatigue.[28] It is not fully understood how recovery of supraspinal fatigue occurs, but it is known that the excitability of the sarcolemma is altered during recovery from training, suggesting a sustained inability of the CNS to optimally drive skeletal muscles, and thus a role of the nervous system in neuromuscular recovery.[13] Rampinini et al[23] observed reductions in voluntary activation, using motor nerve stimulation for up to 48 hours after a soccer match. In addition, reductions in voluntary activation after repeated-sprint exercises have been reported,[29] suggesting that recovery of the CNS is likely contributing to the overall recovery of physical performance. The slower recovery of peripheral fatigue indicates that recovery of muscle function after high-intensity exercise is primarily explained by peripheral processes.[30,31]

However, longer exercise at lower intensity rather amplifies central fatigue.[31] Despite these suggestions, the role of a central regulation in recovery processes should be interpreted with caution, as evidence from direct measures is widely lacking. Recently, measuring brain activity during exercise and recovery became possible using electroencephalography.[32] To investigate which brain areas could be involved in exercise-induced fatigue and recovery, De Pauw et al[32] determined the effect of prolonged intensive cycling and postexercise recovery in the heat on brain sources of altered brain oscillations using electroencephalography. The beta (β) frequency range (between 12 and 30 Hz) extracted from electroencephalography is of interest when human movement is involved. Generally, more complex movements imply more brain activity and thus higher β activity. However, a prolonged intensive cycling performance in the heat has been shown to reduce β activity across the whole brain, probably due to inhibitory signals between brain areas involved in sensory-motor information processing. Postexercise active and passive recovery did not change brain activity, whereas CWI significantly increased β3 (between 21.5 and 30 Hz) activity in brain areas involved in somatosensory information processing.[32] Some authors state that recent literature has focused too much on the reduction of descending motor drive or muscle power generated in the CNS to understand brain fatigue.[33] However, as brain fatigue is complex and involves many interactions, future research should also integrate psychobiological measures (eg, concentration, attention, decision making) as well as physiological parameters.[33] More studies are necessary to determine the exact contribution of the CNS on the recovery from exercise. Furthermore, the optimal timing, dose, and combination of interventions for brain recovery remain unclear.[33]

Cardiocirculatory Recovery

A negative fluid balance is common after prolonged and/or intense training sessions. The level of dehydration, which depends on environmental conditions (ie, temperature and humidity), can reach up to 4% of body mass.[11,13] It is not known whether such a deficit has an impact on anaerobic performance[11,13]; however, it undoubtedly alters endurance performance. In fact, the main consequence of the exercise-induced hypovolemia is a decrease in stroke volume and a tightly coupled increase in heart rate to maintain cardiac output. This alteration in turn not only decreases maximal oxygen uptake (due to the reduced remaining capacity to increase heart rate) and related fitness components such as repeated-sprint ability, but also the capacity to maintain core temperature, which is considered as an important determinant for the perception of fatigue.[34] The time required to return to euhydration is difficult to establish, as it depends on a number of factors including the level of postexercise dehydration, the volume of ingested fluid, its temperature, and the composition as well as the speed of gastric emptying. However, it can be relatively short (~6 h) provided all recommendations are fulfilled.[35]

Another parameter of the cardiovascular system that can be altered after training or competition is the maximal excitability of the autonomic nervous system. A maximal graded exercise test,[36] a long duration race,[37] or a high-intensity interval exercise[38] alter cardiac autonomic control immediately after exercise. The duration required to return to baseline is on average 48 hours. In addition, heart rate was still reduced during a 40-km cycling time trial after 72 hours of recovery after a 6-day intense training camp.[39] This might be due to the influence of peripheral locomotor muscle fatigue leading to reduced power output achieved with maximal effort. Alternatively, this could be the result of an altered/impaired maximal responsiveness of the adrenergic system in general. Therefore, it might be difficult to cope with exercise of near-maximal intensity and thus reach high heart rate values despite sufficient recovery of the cardiovascular system.

Performance Recovery

Irrespective of the physiological underpinnings of recovery, it is clearly more critical for coaches and athletes to understand its impact on physical performance. In team-sport athletes,

maximal strength,[18,23] vertical jump ability,[23,30] and sprint performance[18,22,23] are significantly impaired immediately postexercise with a time to fully recover of 12 to >72 hours. The duration and magnitude of the performance decrement vary between studies, as these effects obviously depend on the exercise type as well as intensity and individual characteristics.[23] For example, in endurance athletes, Skorski et al[39] did not observe a meaningful reduction in cycling performance (40-km time trial) after a 6-day intensive training camp in 10 out of the 23 cyclists involved. Even though it has been stated that the assessment of a decrement in sports-specific performance represents the gold standard for measuring short-term fatigue in athletes, it might be speculated that fatigue-related performance decrements may persist despite measurable physiological changes.

The mechanisms involved in training- or competition-induced fatigue (and the recovery kinetics) are numerous. While many of these physiological factors are classically linked to fatigue and recovery, corresponding rates of recovery markers often do not follow the time courses of performance recovery. Indeed, the time course of recovery in various physiological and biochemical systems appear to depend on the type, duration, and intensity of the exercise. Figure 1 displays hypothetical and schematic time courses (based on reviews by Bessa et al[40] and Peake et al[41]) of carbohydrate resynthesis and inflammation after an endurance (A) and strength (B) training stimuli as an example. Carbohydrate resynthesis and inflammation have been specifically chosen as these markers show very different responses depending on the exercise type. The figure shall support the described dependence of different physiological markers as well as their time courses for recovery based on the exercise type/stimulus. Finally, external factors influencing the time course of the measurable parameters (eg, the degradation rate of blood-borne molecules, recovery interventions, diet, or lifestyle behavior) may further impair simple and linear interpretations. Thus, even though studies investigating recovery interventions often focus on changes in these markers, no single parameter has yet shown a direct and causative relationship with performance recovery.[13] It appears that factors such as an athlete's gender, age, training experience, level of performance, environmental conditions, psychological profile, and the characteristics of the fatiguing exercise also influence the recovery profile of many of these markers. This further challenges the use of a single surrogate parameter to monitor fatigue and recovery and

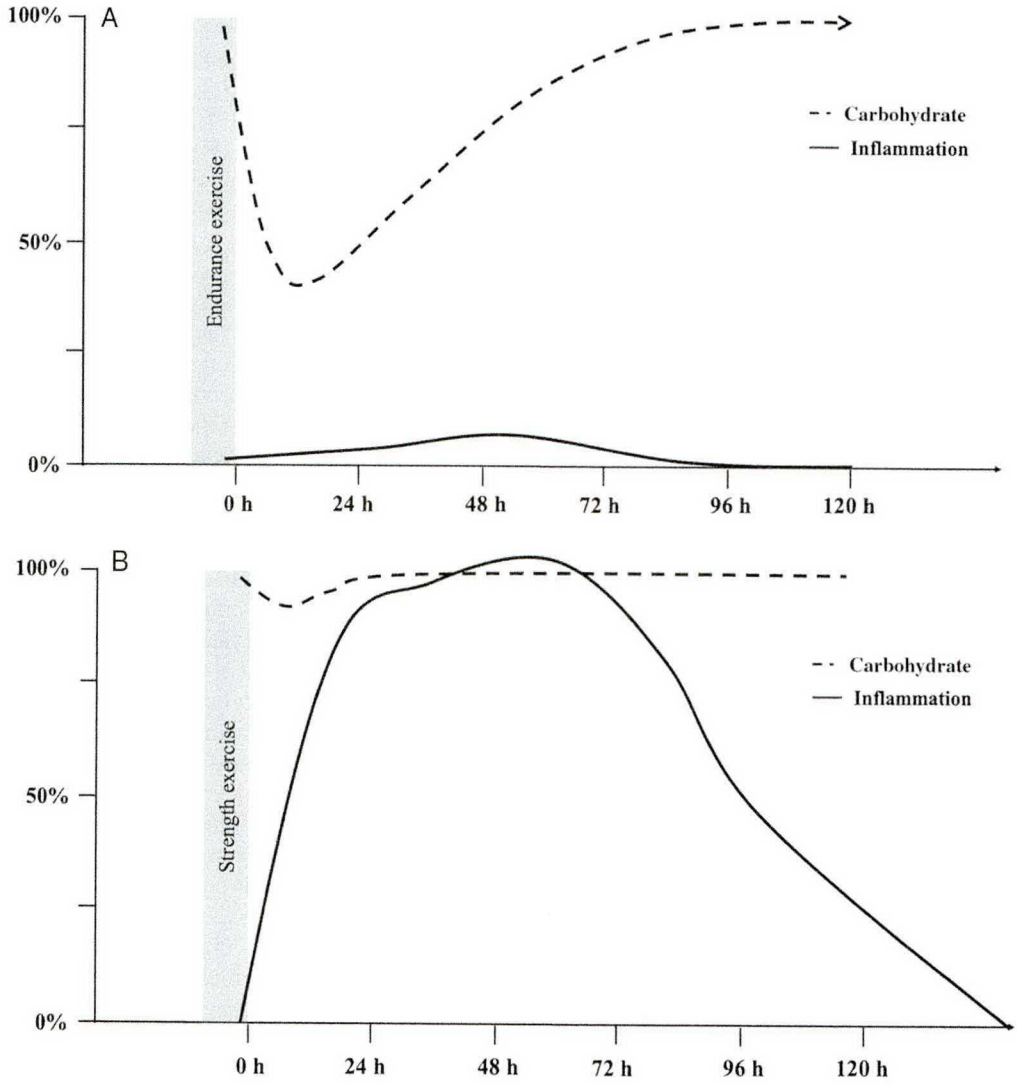


Figure 1 — Schematic example for time courses of recovery of carbohydrate resynthesis and inflammation after (A) endurance and (B) strength-training stimuli. The gray bar displays the training stimulus, the solid line the time course of inflammation, and the dotted line the time course of carbohydrate synthesis. (Time course for inflammation in strength training is based on reviews by Bessa et al[40] and Peake et al.[41])

emphasizes the importance of a multivariate approach to monitoring. Furthermore, it seems plausible to suggest that recovery interventions need to be periodized throughout a season (depending on training load) and administered based on training mode, intensity, and duration as well as individual responses. The different time courses of peripheral disturbances alongside central regulation should definitely be considered when recovery interventions and/or session are implemented.[13]

Planning Recovery in Training and Competition

Because insufficient recovery prevents athletes from training/competing at the required intensity or completing the required load in the upcoming training session, often active recovery interventions are undertaken after training and competition to enhance the recovery process. Such interventions are reported to shift the stress-recovery balance toward recovery. Thus, strategies that minimize fatigue and/or accelerate recovery after training are considered beneficial, as they can facilitate an athlete's readiness for further training or competition.

Planning and implementation of specific recovery interventions to improve sport-specific performance recovery can be complex. As outlined previously, a myriad of physiological systems are involved during exercise with differing time courses of postexercise recovery, which further depend on the type and the load of the exercise. Figure 2 describes hypothetical and schematic time courses of these physiological pathways after team-sport play to illustrate these variations. The figure shall support and summarize the previously described differences in the recovery time course of several physiological systems. In addition, the time courses of recovery in physiological measures rarely align with performance recovery. As such, the effectiveness of a recovery intervention seems to be related to the nature and extent of the induced fatigue. This challenges coaches and scientists to successfully plan and structure appropriate evidence-based recovery interventions (see review by Mujika et al[42] on periodizing recovery in individual and team sports).

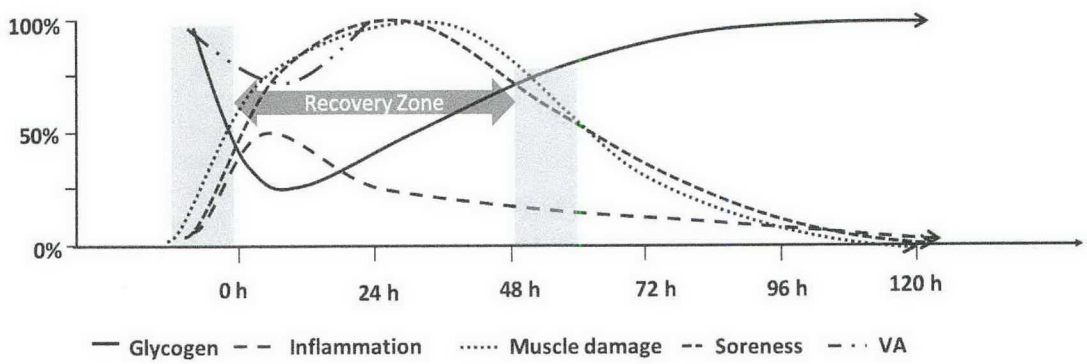
The most obvious factor that dictates recovery requirements is the nature and extent of the exercise stimulus (ie, the specific training load). Fatigue as a consequence of training may influence training quality and performance over subsequent days.[42] Thus, recovery interventions are often seen as an important tool to help the body to return to the previous state. However, too much recovery may lead to an insufficient overall training stress and reduce or hinder adaptation. For example, some studies observed detrimental effects of regular CWI on hypertrophy adaptations during strength training periods, questioning its use during specific training periods.[43] These detrimental effects might be attributed to CWI-related blunted activation of key proteins and satellite cells in the skeletal muscle after exercise.[43] By contrast, Halson et al[44] observed a greater increase in repeated high-intensity cycling performance when CWI was regularly administered during an intensified training phase. Ihsan et al[45] reported greater improvements in maximum oxygen consumption (VO_2max) as well as maximum running velocity (V_{max}) when CWI was regularly used after intense running bouts. The authors speculated that regular CWI applications might enhance p38 and AMPK activation and thus possibly augment mitochondrial biogenesis.[45] However, Broatch et al[46] observed only limited effects on exercise-induced mitochondrial biogenesis, changes in mitochondrial content or function, and VO_2max when administering regular CWI during a 6-week cycling sprint interval training period. As current results are still controversial, further research on long-term effects of CWI and other recovery interventions on performance adaptation is warranted.

It should further be considered that as most recovery interventions seem to positively influence subjective parameters, a placebo effect can hardly be eliminated. In this regard, it should be noted that due to contamination effects of different treatments, scientifically evaluating a specific recovery intervention usually prohibits any other recovery treatment. Moreover, as psychological aspects are important in elite sport, withholding an intervention the athlete is accustomed to could "disrupt" his or her habits and negatively influence their perception toward the new strategies. Therefore, recovery interventions should be individualized, sport-specific, and periodized according to the specific training context.[47]

Practical Applications

Although several recovery interventions such as postexercise CWI, whole-body cryotherapy, massage, or compression garments can have a positive impact on athletes' acute recovery, the efficacy of these specific recovery interventions should be determined in the context of the specific training and competition stimuli and the short- and long-term training goals. As training goals and objectives shift in accordance with a periodized approach throughout the season, recovery strategies should be adjusted to meet each athlete's specific needs.

In this regard, 2 adaptation theories have been proposed regarding the implementation of recovery interventions[44]: (1) they might allow athletes to perform subsequent training sessions with a greater load and/or quality, resulting in an enhanced adaptation and (2) they



might decrease training adaptations as they minimize training-induced fatigue; therefore, withholding recovery during specific phases (eg, general preparation phase at the beginning of the season) may be important to maximize training adaptations. In phases involving a high-skill component or when a high quality of the training session is required (eg, technique skills), utilizing recovery interventions might improve the athletes' ability to prepare for these sessions. During the competition phases where decreasing acute fatigue is paramount for optimal performance, incorporating adequate interventions might improve perceptual and performance recovery (eg, during multiday events; for a detailed review on periodizing recovery, see Mujika et al[42]; detailed reviews regarding the general effects of specific recovery interventions, such as sleep[14,48–51] and CWI[47,52,53] have been published elsewhere); however, more research is still needed evaluating the effects of recovery interventions in different training phases.

Conclusion

The effects of fatigue and recovery on performance in training and competition are complex. The fatigue resulting from a training session depends on the specific training stimulus and the extent of the stress in the various physiological systems. These systems can require different recovery periods for complete restoration of homeostasis. In addition, despite a vast amount of research evaluating the efficacy of various recovery interventions, a "one-size-fits-all" approach does not appear to be appropriate. Indeed, recovery strategies should be tailored to the need of the individual athlete and carefully periodized throughout a season, given that too much recovery and/or specific interventions might blunt training adaptation. Further research is needed to evaluate the effectiveness of recovery interventions based on the type of exercise, as well as the training load. Moreover, as responses seem to be highly individual, coaches are advised to administer different recovery interventions in a variety of situations to evaluate the optimal procedure for each athlete in different training and competition settings.

References

1. Hausswirth C, Mujika I, eds. *Recovery for Performance in Sport.* Champaign, IL: Human Kinetics; 2013. ISBN 9781450434348.
2. Meeusen R, Duclos M, Foster C, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Med Sci Sports Exerc.* 2013;45(1):186–205. PubMed ID: 23247672 doi:10.1249/MSS.0b013e318279a10a
3. Vanrenterghem J, Nedergaard NJ, Robinson MA, Drust B. Training load monitoring in team sports: a novel framework separating physiological and biomechanical load-adaptation pathways. *Sports Med.* 2017;47(11):2135–2142. PubMed ID: 28283992 doi:10.1007/s40279-017-0714-2
4. Lambert MI, Mujika I. Physiology of exercise training. In: Hausswirth C, Mujika I, eds. *Recovery for Performance in Sport.* Champaign, IL: Human Kinetics; 2013.
5. Buchheit M, Laursen PB. High-intensity interval training, solutions to the programming puzzle: part I: cardiopulmonary emphasis. *Sports Med.* 2013;43(5):313–338. PubMed ID: 23539308 doi:10.1007/s40279-013-0029-x
6. di Prampero PE, Atchou G, Bruckner JC, Moia C. The energetics of endurance running. *Eur J Appl Physiol Occup Physiol.* 1986;55(3):259–266. PubMed ID: 3732253 doi:10.1007/BF02343797
7. Gaudino P, Alberti G, Iaia FM. Estimated metabolic and mechanical demands during different small-sided games in elite soccer players. *Hum Mov Sci.* 2014;36:123–133. PubMed ID: 24968370 doi:10.1016/j.humov.2014.05.006
8. Kellmann M, Bertollo M, Bosquet L, et al. Recovery and performance in sport: consensus statement. *Int J Sports Physiol Perform.* 2018;13(2):240–245. PubMed ID: 29345524 doi:10.1123/ijspp.2017-0759
9. Julian R, Meyer T, Fullagar HH, et al. Individual patterns in blood-borne indicators of fatigue—trait or chance. *J Strength Cond Res.* 2017;31:608–619. doi:10.1519/JSC.0000000000001390
10. Hecksteden A, Pitsch W, Julian R, et al. A new method to individualize monitoring of muscle recovery in athletes. *Int J Sports Physiol Perform.* 2017;12(9):1137–1142. PubMed ID: 27967274 doi:10.1123/ijspp.2016-0120
11. Nedelec M, McCall A, Carling C, Legall F, Berthoin S, Dupont G. Recovery in soccer: part I—post-match fatigue and time course of recovery. *Sports Med.* 2012;42(12):997–1015. PubMed ID: 23046224
12. McMahon S, Jenkins D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. *Sports Med.* 2002;32(12):761–784. PubMed ID: 12238940 doi:10.2165/00007256-200232120-00002
13. Minett GM, Duffield R. Is recovery driven by central or peripheral factors? A role for the brain in recovery following intermittent-sprint exercise. *Front Physiol.* 2014;5:24. PubMed ID: 24550837 doi:10.3389/fphys.2014.00024
14. Burke LM, Mujika I. Nutrition for recovery in aquatic sports. *Int J Sport Nutr Exerc Metab.* 2014;24(4):425–436. PubMed ID: 24901517 doi:10.1123/ijsnem.2014-0022
15. Cairns SP. Lactic acid and exercise performance: culprit or friend? *Sports Med.* 2006;36(4):279–291. PubMed ID: 16573355 doi:10.2165/00007256-200636040-00001
16. Pedersen TH, Nielsen OB, Lamb GD, Stephenson DG. Intracellular acidosis enhances the excitability of working muscle. *Science.* 2004;305(5687):1144–1147. PubMed ID: 15326352 doi:10.1126/science.1101141
17. Fitts RH. Cellular mechanisms of muscle fatigue. *Physiol Rev.* 1994;74(1):49–94. PubMed ID: 8295935 doi:10.1152/physrev.1994.74.1.49
18. Ascensão A, Rebelo A, Oliveira E, Marques F, Pereira L, Magalhaes J. Biochemical impact of a soccer match—analysis of oxidative stress and muscle damage markers throughout recovery. *Clin Biochem.* 2008;41(10–11):841–851. PubMed ID: 18457670 doi:10.1016/j.clinbiochem.2008.04.008
19. Close GL, Ashton T, McArdle A, Maclaren DP. The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comp Biochem Physiol A Mol Integr Physiol.* 2005;142(3):257–266. PubMed ID: 16153865 doi:10.1016/j.cbpa.2005.08.005
20. Ross EZ, Goodall S, Stevens A, Harris I. Time course of neuromuscular changes during running in well-trained subjects. *Med Sci Sports Exerc.* 2010;42(6):1184–1190. PubMed ID: 19997016
21. Duffield R, Murphy A, Snape A, Minett GM, Skein M. Post-match changes in neuromuscular function and the relationship to match demands in amateur rugby league matches. *J Sci Med Sport.* 2012;15(3):238–243. PubMed ID: 22137196 doi:10.1016/j.jsams.2011.10.003
22. 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(5):423–431. PubMed ID: 18806550 doi:10.1097/JSM.0b013e3181818e0b
23. Rampinini E, Bosio A, Ferraresi I, Petruolo A, Morelli A, Sassi A. Match-related fatigue in soccer players. *Med Sci Sports Exerc.* 2011;43(11):2161–2170. PubMed ID: 21502891 doi:10.1249/MSS.0b013e31821e9c5c

24. Taylor JL, Amann M, Duchateau J, Meeusen R, Rice CL. Neural contributions to muscle fatigue: from the brain to the muscle and back again. *Med Sci Sports Exerc.* 2016;48(11):2294–2306. PubMed ID: 27003703 doi:10.1249/MSS.0000000000000923
25. Roelands B, Meeusen R. Alterations in central fatigue by pharmacological manipulations of neurotransmitters in normal and high ambient temperature. *Sports Med.* 2010;40(3):229–246. PubMed ID: 20199121 doi:10.2165/11533670-000000000-00000
26. Meeusen R, Watson P, Hasegawa H, Roelands B, Piacentini MF. Central fatigue: the serotonin hypothesis and beyond. *Sports Med.* 2006;36(10):881–909. PubMed ID: 17004850 doi:10.2165/00007256-200636100-00006
27. Meeusen R, Piacentini MF, De Meirleir K. Brain microdialysis in exercise research. *Sports Med.* 2001;31(14):965–983. PubMed ID: 11735681 doi:10.2165/00007256-200131140-00002
28. Klass M, Roelands B, Levenez M, et al. Effects of noradrenaline and dopamine on supraspinal fatigue in well-trained men. *Med Sci Sports Exerc.* 2012;44(12):2299–2308. doi:10.1249/MSS.0b013e318265f356
29. Goodall S, Charlton K, Howatson G, Thomas K. Neuromuscular fatigability during repeated-sprint exercise in male athletes. *Med Sci Sports Exerc.* 2015;47(3):528–536. PubMed ID: 25010404 doi:10.1249/MSS.0000000000000443
30. Thomas K, Dent J, Howatson G, Goodall S. Etiology and recovery of neuromuscular fatigue after simulated soccer match play. *Med Sci Sports Exerc.* 2017;49(5):955–964. PubMed ID: 28060035 doi:10.1249/MSS.0000000000001196
31. Thomas K, Goodall S, Stone M, Howatson G, St Clair Gibson A, Ansley L. Central and peripheral fatigue in male cyclists after 4-, 20-, and 40-km time trials. *Med Sci Sports Exerc.* 2015;47(3):537–546. PubMed ID: 25051388 doi:10.1249/MSS.0000000000000448
32. De Pauw K, Roelands B, Marusic U, Tellez HF, Knaepen K, Meeusen R. Brain mapping after prolonged cycling and during recovery in the heat. *J Appl Physiol.* 2013;115(9):1324–1331. PubMed ID: 23990240 doi:10.1152/japplphysiol.00633.2013
33. Rattray B, Argus C, Martin K, Northey J, Driller M. Is it time to turn our attention toward central mechanisms for post-exertional recovery strategies and performance? *Front Physiol.* 2015;6:79. PubMed ID: 25852568 doi:10.3389/fphys.2015.00079
34. Gonzalez-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol.* 1999;86(3):1032–1039. PubMed ID: 10066720 doi:10.1152/jappl.1999.86.3.1032
35. Brad RA. Exercise and fluid replacement: brought to you by the American College of Sports Medicine www.acsm.org. *ACSM Health Fitness J.* 2013;17(4):4–7.
36. Furlan R, Piazza S, Orto SD, et al. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovasc Res.* 1993;27(3):482–488. PubMed ID: 8490949 doi:10.1093/cvr/27.3.482
37. Hautala A, Tulppo MP, Makikallio TH, Laukkanen R, Nissila S, Huikuri HV. Changes in cardiac autonomic regulation after prolonged maximal exercise. *Clin Physiol.* 2001;21(2):238–245. PubMed ID: 11318832 doi:10.1046/j.1365-2281.2001.00309.x
38. Al Haddad H, Laursen PB, Ahmaidi S, Buchheit M. Nocturnal heart rate variability following supramaximal intermittent exercise. *Int J Sports Physiol Perform.* 2009;4(4):435–447. PubMed ID: 20029095 doi:10.1123/ijspp.4.4.435
39. Skorski S, Hammes D, Schwindling S, et al. Effects of training-induced fatigue on pacing patterns in 40-km cycling time trials. *Med Sci Sports Exerc.* 2015;47(3):593–600. doi:10.1249/MSS.0000000000000439
40. Bessa AL, Oliveira VN, Agostini GG, et al. Exercise intensity and recovery: biomarkers of injury, inflammation, and oxidative stress. *J Strength Cond Res.* 2016;30(2):311–319. PubMed ID: 23604000 doi:10.1519/JSC.0b013e31828f1ee9
41. Peake JM, Neubauer O, Della Gatta PA, Nosaka K. Muscle damage and inflammation during recovery from exercise. *J Appl Physiol.* 2017;122(3):559–570. PubMed ID: 28035017 doi:10.1152/japplphysiol.00971.2016
42. Mujika I, Halson S, Burke LM, Balague G, Farrow D. An integrated, multifactorial approach to periodization for optimal performance in individual and team sports. *Int J Sports Physiol Perform.* 2018;13(5):538–561. PubMed ID: 29848161 doi:10.1123/ijspp.2018-0093
43. Roberts LA, Raastad T, Markworth JF, et al. Post-exercise cold water immersion attenuates acute anabolic signalling and long-term adaptations in muscle to strength training. *J Physiol.* 2015;593(18):4285–4301. PubMed ID: 26174323 doi:10.1113/JP270570
44. Halson SL, Bartram J, West N, et al. Does hydrotherapy help or hinder adaptation to training in competitive cyclists? *Med Sci Sports Exerc.* 2014;46(8):1631–1639. doi:10.1249/MSS.0000000000000268
45. Ihsan M, Markworth JF, Watson G, et al. Regular postexercise cooling enhances mitochondrial biogenesis through AMPK and p38 MAPK in human skeletal muscle. *Am J Physiol Regul Integr Comp Physiol.* 2015;309(3):R286–R294. PubMed ID: 26041108 doi:10.1152/ajpregu.00031.2015
46. Broatch JR, Petersen A, Bishop DJ. Cold-water immersion following sprint interval training does not alter endurance signaling pathways or training adaptations in human skeletal muscle. *Am J Physiol Regul Integr Comp Physiol.* 2017;313(4):R372–R384. PubMed ID: 28679683 doi:10.1152/ajpregu.00434.2016
47. Tavares F, Walker O, Healey P, Smith TB, Driller M. Practical applications of water immersion recovery modalities for team sports. *Strength Cond J.* 2018;40(4):48–60.
48. Fullagar HH, Skorski S, Duffield R, Hammes D, Coutts AJ, Meyer T. Sleep and athletic performance: the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise. *Sports Med.* 2015;45(2):161–186. doi:10.1007/s40279-014-0260-0
49. Nedelec M, Halson S, Abaidia AE, Ahmaidi S, Dupont G. Stress, sleep and recovery in elite soccer: a critical review of the literature. *Sports Med.* 2015;45(10):1387–1400. PubMed ID: 26206724 doi:10.1007/s40279-015-0358-z
50. McCartney D, Desbrow B, Irwin C. Post-exercise ingestion of carbohydrate, protein and water: a systematic review and meta-analysis for effects on subsequent athletic performance. *Sports Med.* 2018;48(2):379–408. doi:10.1007/s40279-017-0800-5
51. Heaton LE, Davis JK, Rawson ES, et al. Selected in-season nutritional strategies to enhance recovery for team sport athletes: a practical overview. *Sports Med.* 2017;47(11):2201–2218. PubMed ID: 28702900 doi:10.1007/s40279-017-0759-2
52. Poppendieck W, Faude O, Wegmann M, Meyer T. Cooling and performance recovery of trained athletes: a meta-analytical review. *Int J Sports Physiol Perform.* 2013;8(3):227–242. PubMed ID: 23434565 doi:10.1123/ijspp.8.3.227
53. Leeder J, Gissane C, van Someren K, Gregson W, Howatson G. Cold water immersion and recovery from strenuous exercise: a meta-analysis. *Br J Sports Med.* 2012;46(4):233–240. PubMed ID: 21947816 doi:10.1136/bjsports-2011-090061