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Using Recovery Modalities between Training Sessions in Elite Athletes
Does it Help?

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Abstract

Achieving an appropriate balance between training and competition stresses and recovery is important in maximising the performance of athletes. A wide range of recovery modalities are now used as integral parts of the training programmes of elite athletes to help attain this balance. This review examined the evidence available as to the efficacy of these recovery modalities in enhancing between-training session recovery in elite athletes. Recovery modalities have largely been investigated with regard to their ability to enhance the rate of blood lactate removal following high-intensity exercise or to reduce the severity and duration of exercise-induced muscle injury and delayed onset muscle soreness (DOMS). Neither of these reflects the circumstances of between-training session

recovery in elite athletes. After high-intensity exercise, rest alone will return blood lactate to baseline levels well within the normal time period between the training sessions of athletes. The majority of studies examining exercise-induced muscle injury and DOMS have used untrained subjects undertaking large amounts of unfamiliar eccentric exercise. This model is unlikely to closely reflect the circumstances of elite athletes. Even without considering the above limitations, there is no substantial scientific evidence to support the use of the recovery modalities reviewed to enhance the between-training session recovery of elite athletes. Modalities reviewed were massage, active recovery, cryotherapy, contrast temperature water immersion therapy, hyperbaric oxygen therapy, nonsteroidal anti-inflammatory drugs, compression garments, stretching, electromyostimulation and combination modalities. Experimental models designed to reflect the circumstances of elite athletes are needed to further investigate the efficacy of various recovery modalities for elite athletes. Other potentially important factors associated with recovery, such as the rate of post-exercise glycogen synthesis and the role of inflammation in the recovery and adaptation process, also need to be considered in this future assessment.

The stressful components of training and competition may temporarily impair an athlete's performance. This impairment may be transitory, lasting minutes or hours after training or competition, or last for a longer period, up to several days. Short-term impairment results from metabolic disturbances following high-intensity exercise.[1] Recovery is also dependent on the restoration of glycogen stores, which usually occurs within 24 hours following exhaustive exercise[2] and rehydration.[3] Longer-lasting impairment may be related to exercise-induced muscle injury and delayed onset muscle soreness (DOMS).[4] Also, although the underlying mechanisms are not well understood, an imbalance between the stress of training and recovery over extended periods seemingly affects performance independently of the above. Such an imbalance can have potentially long-term debilitating effects in the form of overtraining.

Lack of appropriate recovery may result in the athlete being unable to train at the required intensity or complete the required load at the next training session. Higher levels of fatigue may also predispose the athlete to injury. Furthermore, full recovery is necessary for optimal competition performance. To enhance the recovery process, athletes often perform structured recovery sessions, sometimes in purpose-built facilities, as part of their regular training and post-competition regime. These sessions are designed to shift the stress-recovery balance away from the stresses induced by training and towards recovery. The overall advantage of these recovery sessions might be to allow the elite athlete to tolerate higher training loads (intensity, volume or frequency) or to enhance the effect of a given training load.

Only a few studies on recovery modalities have involved elite athletes,[5-12] possibly because the negative effects of training are more easily induced in untrained subjects and because elite athletes are often justifiably reluctant to participate in controlled studies that change their training. A population should have similar characteristics to the study sample for the results to apply to that population with reasonable certainty. Because of their genetic make-up and training history, elite athletes respond differently to training stress, and possibly to the following recovery, than do athletes at a lower level or non-athletes. So, for results to apply convincingly to elite athletes, the subjects should be elite athletes. However, where this information is unavailable, qualified inferences can be made with data from other human subjects and animals.

Various recovery modalities are currently used, and it is important to know the effectiveness of and

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the rationale underlying each modality. This article reviews the literature on the different modalities currently used to aid athlete recovery after exercise training. These are: massage, active recovery, cryotherapy, contrast temperature water immersion, hyperbaric oxygen therapy (HBOT), nonsteroidal anti-inflammatory drugs (NSAIDs), compression garments, stretching, electromyostimulation (EMS) and combination modalities. To relate recovery to the usual type of training by elite athletes, it has been assumed that training sessions are separated by ≥4 hours and that to be considered effective, a modality must be more effective than simple rest.

The main purpose of this article is to examine the evidence of the effectiveness of recovery modalities as part of the between-training recovery of elite athletes. This has been done by discussing relevant aspects of recovery and then the recovery modalities. Conclusions are drawn and recommendations for future research presented.

1. Possible Influences on Post-Training Recovery

1.1 Metabolic Disturbances Resulting from High-Intensity Exercise

Elite athletes typically engage in high-intensity training. Such exercise elicits increases in lactate and hydrogen ion concentrations within the muscle, both of which have been historically associated with muscle fatigue.[13] This association has lead to the rate of removal of lactate, as indicated by decreases in blood lactate concentration, being one of the main criteria used to test recovery quality. However, lactate removal may not be a valid criterion for assessing recovery, especially in relation to between-training recovery in elite athletes.

The historical and somewhat entrenched beliefs on lactate, acidosis and muscle fatigue during high-intensity exercise are not supported by recent research. As an in-depth review of the causes of fatigue during high-intensity exercise is beyond the scope of this article, a brief summary is given and the reader is referred to recent, extensive reviews and discussion elsewhere.[1,13-15] First, there is a strong argument for lactate production not being a cause of metabolic acidosis.[15-17] Second, recent evidence suggests that acidosis has little, if any, negative effect on muscle contraction at physiological temperatures.[1,13] Third, acidosis has even been shown to have protective effects.[13,14] None of the above support the use of rate of post-training lactate removal as an indicator of the quality of a recovery modality. In apparent contrast to the recent findings in muscle, most studies that have induced acidosis in humans have found a negative effect on exercise performance, and it has been hypothesised that this effect may be via the CNS.[13] While these studies may indicate a role of acidosis in fatigue, they do not justify lactate as an indicator of recovery if, as described by Robergs et al.,[15] lactate is not responsible for the acidosis resulting from high-intensity exercise.

Moreover, there has been a lack of a consistent relationship between recovery lactate and subsequent performance in studies comparing active and passive recovery. Several studies found that, despite lower lactate concentrations, performance was not improved following active recovery.[18-21] In other studies, performance was better following active recovery, although there was no significant difference in post-recovery lactate between active and passive recovery modalities.[22-24] Even when better performance has been associated with lower lactate after active recovery, a correlation of only $R = -0.19$ was found between end-recovery blood lactate and post-recovery performance.[25] Similar low correlations have been reported by other investigators.[19,26] Jones et al.[27] found that prior exercise, which caused a significant elevation of blood lactate, resulted in an increased time to exhaustion in subsequent exercise at 100%, 110% and 120% peak oxygen uptake. Also, the half-life of muscle lactate is around 9.5 minutes.[28] Blood lactate has a half-life of around 15 minutes during resting recovery[29] and returns to around resting levels 90 minutes after very high-intensity exercise.[30] This is a shorter timeframe than is typical between the training sessions of elite athletes and any recovery modality that is undertaken on the basis of its effect on blood lactate

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removal seems superfluous. Thus, lactate removal does not appear to be a valid indicator of recovery quality, especially when considering recovery between training sessions in elite athletes.

1.2 Rehydration and Glycogen Resynthesis between Training Sessions

Prior dehydration can be detrimental to performance.[31] So, rehydration following training sessions is an appropriate nutritional strategy for ensuing that work capacity is not diminished at the beginning of the next session. When drinks of an appropriate volume and sodium content are consumed following dehydration by 2% body mass, net fluid balance and plasma volume can be restored within 4 hours.[32] It is unlikely that currently used recovery modalities would compromise this fluid replacement.

In sports where a training session leads to glycogen depletion, the load attainable by the athlete in a subsequent session may be limited by the post-training synthesis that has occurred. Rapid post-training glycogen synthesis is important to recovery between training sessions when these athletes complete more than one session per day.[2] Although muscle glycogen stores can be restored completely within 24 hours with adequate carbohydrate intake, complete resynthesis is unlikely to occur during the recovery between sessions in the same day.[2] However, an appropriate dietary routine can enhance the synthesis that occurs over the short-term. Maximising glycogen synthesis during this shorter recovery period is best achieved by consuming large amounts of carbohydrate (1.2 g/kg/hour) immediately post-exercise and at regular intervals up to 5 hours post-exercise.[2] Muscle glycogen synthesis is most likely limited by a combination of carbohydrate intake, intestinal glucose absorption, glucose delivery via the bloodstream, glucose extraction by other tissues, and the muscle's glucose-transport capacity.[2] Because recovery modalities typically target blood flow and muscle, they may theoretically alter glucose availability to the muscle during recovery and therefore alter the process of glycogen synthesis. Research is required to examine the possible impact of recovery modalities on the rate of post-training glycogen synthesis in athletes following the current dietary recommendations.

1.3 Exercise-Induced Muscle Damage and Delayed Onset Muscle Soreness

Many studies examining the efficacy of recovery modalities have focused on exercise-induced muscle damage, usually associated with DOMS, a sensation of pain or discomfort occurring 1–2 days post-exercise. Although the underlying mechanism is not well understood, full recovery of strength and power after a training session that causes DOMS may take several days.[4] Therefore, its occurrence may be detrimental to an ongoing training programme. Modalities that enhance the rate of recovery from DOMS and exercise-induced muscle damage may enhance the overall training of elite athletes.

Although DOMS can be induced in both trained[5,33] and untrained individuals[34-36] it is typically associated with unaccustomed high-intensity physical activity, usually with a large eccentric component, rather than with regular training. Prior training attenuates DOMS and changes in performance, serum creatine kinase concentration and muscle morphology occurring after an acute bout of exercise.[37] This training effect has been shown for endurance running, cycle ergometry, weightlifting, isometric exercise, downhill running and eccentric exercise.[37] Even a single bout of eccentric exercise has a protective effect against exercise-induced muscle injury, DOMS, and loss of strength from exercise undertaken up to 6 months later; this phenomenon is called the 'repeated-bout effect'.[38]

The repeated-bout effect was first noted for serum creatine kinase concentration, an indicator of exercise-induced muscle damage, after repeated sessions of isometric forearm flexion separated by 1 week,[39] and for muscle soreness and serum creatine kinase and myoglobin concentrations after bouts of downhill running separated by 6, but not 9, weeks.[40] Clarkson and Tremblay[41] found that the changes in isometric strength, muscle soreness and pain, and serum creatine kinase concentration after 70 maximal, eccentric forearm flexions were significantly smaller when the exercise bout was preceded

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2 weeks earlier by 24 maximal eccentric forearm flexions. Isometric strength was maintained near the baseline value 1 day after the bout with the preceding exercise, whereas, without the preceding exercise, isometric strength was impaired and did not return to the initial level after 5 days.[41] Similarly, Jones et al.[42] reported reduced pain and discomfort, muscle stiffness and plasma creatine kinase responses when eccentric forearm flexion was repeated 2 weeks after the initial bout.

Two more recent studies[38,43] have found that the repeated-bout effect lasts several months for some variables. Nosaka et al.[43] examined the repeated-bout effect on the post-exercise response to eccentric elbow flexion. The effects on recovery of muscle strength, shortening ability and soreness were apparent when bouts were separated by 6, but not by 10, weeks. In contrast, plasma creatine kinase activity after the second bout was still significantly lower when the bouts were separated by 6 months. This long-lasting effect has been confirmed in a more recent investigation showing a repeated-bout effect of up to 9 months for maximal isometric force and flexed-elbow joint angle.[38] Muscle soreness, T2 relaxation times (an indicator of muscle damage using magnetic resonance imaging [MRI]), and increases in upper arm circumference and plasma creatine kinase activity were lower in the second bout when the bouts were separated by up to 6 months. No repeated-bout effect was seen at 12 months.

Although the duration of the protective effect differs between studies, it seems to be greatest within 2 weeks of the preceding bout and diminishes with time.[38] In contrast, Pierrynowski et al.[44] found that, although 4-day prior downhill running reduced the sensation of DOMS after downhill running, it did not prevent the loss of muscular strength. Similarly, another study involving pre-training using either downhill or uphill running for 1 or 2 weeks before a 45-minute downhill run found that the DOMS sensation decreased, but serum creatine kinase activity was unchanged, after downhill running.[45] The lack of any difference in serum creatine kinase activity may be explained by the small number of subjects in some training groups and the large inter-individual variation in the serum creatine kinase response, which has also been reported by other investigators.[38,42,43]

An athlete may experience DOMS at the beginning of a training season or during the transition to high-intensity training, although DOMS can be minimised with a well designed, progressive programme. Prior concentric-only training may predispose the muscle to dysfunction and injury after intense eccentric exercise,[46] which may have implications for programme design in sports using periods of concentric-only training. High-intensity training induces muscle soreness in elite junior track and field athletes, but much less than that in untrained subjects.[5] Much of the current information on the efficacy of recovery modalities is from research on their effect on muscle injury and DOMS in untrained subjects. For example, in a recent review, none of the nine cited studies of the effects of massage on muscle soreness used trained subjects.[47] In summary, prior training reduces the negative effects of an exercise bout, so elite athletes may respond to recovery modalities differently than do sub-elite or untrained individuals. The relevance to elite athletes of current exercise-induced muscle damage and DOMS-based recovery research is unclear. Research using models more applicable to elite athletes is needed.

1.4 The Role of Inflammation in Muscle Repair and Adaptation During Recovery

It has generally been considered that restricting inflammation following muscle use or injury and enhancing its rate of removal have a positive effect on muscle repair and adaptation. While the exact mechanisms are still to be elucidated, post-exercise inflammatory processes appear to be involved in both damage and repair after modified muscle use or injury.[48] Neutrophils and macrophages are the major contributors to the inflammatory response. While neutrophils have been linked to the promotion of muscle damage, their role in processing and removal of damaged tissue may be important in muscle regeneration. Similarly, macrophages can injure muscle cells, but there is increasing evidence supporting

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the existence of macrophage-derived factors that influence muscle growth and regeneration.[48] Also, muscle cells can modulate the influence of inflammatory cells in ways that inhibit their muscle damaging effects. Inflammation is important in the repeated-bout effect adaptive response to exercise-induced muscle damage, probably via the strengthening of muscle structural elements.[49] So, repression of the acute inflammatory process seems inappropriate, as it appears to have an integral role in adaptation and repair. Application of a recovery modality designed to reduce inflammation may not be in the best interests of the athlete.

The magnitude of the inflammatory response, the history of muscle use, and possibly injury-specific interactions between the muscle and inflammatory cells influence whether the inflammatory process will have an overall detrimental or beneficial effect on muscle adaptation and repair.[48] The muscles of elite athletes are subjected to different types and magnitudes of stress than untrained or moderately trained individuals, and their history of muscle use differs greatly. Therefore, the inflammatory process may also differ. It follows that investigations are needed that examine the effect of recovery interventions in elite athletes on the inflammatory process and the consequences for recovery from and adaptation to muscle use and damage.

1.5 Decrements in Performance Resulting from an Imbalance between Stress and Recovery Over Time

Biochemical, physiological or immunological markers that consistently detect an imbalance between training and recovery resulting in future performance decrements, along with the mechanisms that underlie this stress-recovery imbalance, have yet to be identified.[50] Petibois et al.[51] monitored the metabolic response to exercise in elite rowers over a 37-week training period using Fourier-transform infrared spectroscopy. The metabolic response to a standard exercise load was determined from the difference in pre- and post-exercise spectra. They observed a sequence of changes in carbohydrate, lipid and protein metabolism, which seemed to indicate a progression towards overtraining. Further studies need to be undertaken with other groups of athletes to confirm the observations made. If the early-stage changes in carbohydrate metabolism are shown to reflect an imbalance of stress and recovery, this method could be used to assess the efficacy of recovery modalities.

There is some indication that certain scales of self-report questionnaires may provide an indication of the stress-recovery balance. The Somatic Complaints scale of the Recovery-Stress Questionnaire for Athletes and the Stress scale of the Daily Analysis of Life Demands for Athletes self-report appear to reflect the stress-recovery balance.[52-55] The vigour and fatigue scales of the Profile of Mood States questionnaire also appear to reflect the interaction between training load and performance.[54,56] While the underlying mechanism has still to be determined, the effect of recovery modalities on these markers during periods of training is a potential area for future research.

2. Recovery Modalities

2.1 Massage

Massage is used extensively in the training of elite athletes and is commonly thought to decrease oedema and pain, enhance blood lactate removal, enhance healing and alleviate DOMS[47] largely by increasing muscle blood flow. However, the most recent studies using Doppler ultrasound to measure blood flow and vessel diameter found no increase in muscle blood flow during massage with[57,58] or without[59] preceding exercise. Hinds et al.[58] examined the effect of massage following isokinetic quadriceps exercise on leg and skin blood flow. No difference in leg blood flow was observed between the massage and control (rest) trials. However, skin blood flow was higher in the massage trial. These findings suggest that massage may divert blood flow from the muscle. However, based on no observed differences in muscle temperature and blood lactate between trials, the authors surmise that any diversion is probably minimal. Earlier studies of the effect of massage on blood flow had design limita-

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tions[47] and used either venous occlusion pethysmography or a ^{133}Xenon wash-out technique, both of which have methodological weaknesses.[57]

None of the methods are able to measure the intramuscular distribution of blood flow[59] and therefore possible changes in the microcirculation.[47] However, an investigation of skeletal muscle capillaries in rats found no alterations that could impair blood flow or capillary exchange up to 24 hours after running.[60]

Increased post-exercise muscle blood flow may help increase the rate of blood lactate removal. However, massage does not increase the rate of blood lactate removal after exercise,[47] which is consistent with the observation that massage has no effect on post-exercise muscle blood flow. Mild activity increases muscle blood flow. If increased muscle blood flow has any post-exercise benefit, mild activity would be superior to massage[57,59] and would be more cost effective.

Several studies have examined the effect of massage on post-exercise loss of muscle strength and the rate of strength recovery. Tiidus and Shoemaker[57] found no difference in post-exercise strength recovery between a massaged and control leg for 96 hours after intense eccentric exercise. This finding is supported by other studies that found no benefit of massage in preventing post-exercise loss of strength or rate of strength recovery.[36,61-63] However, these studies did find that massage reduces the intensity of soreness[36,62,63] and tenderness,[62] but not the unpleasantness of soreness.[36] Tiidus and Shoemaker[57] also found significantly lower DOMS sensation with massage treatment than without treatment 48 hours after exercise, but not at 24, 72, or 96 hours post-exercise. Boxers undertaking simulated boxing bouts separated by 1 hour showed higher perception of recovery, but no physiological or performance benefits, from a 20-minute massage treatment.[64] In contrast, other studies have found no beneficial effect of massage on DOMS sensation.[58,61,65,66]

If massage reduces perceived soreness without accompanying physiological and performance recovery, the athlete may attempt training loads beyond current capacity. This may lead to an inappropriate level of stress, undesirable levels of fatigue and potentially greater risk of injury. The sensation of muscle soreness after eccentric exercise is not necessarily synchronised with the degree of strength loss and indicators of muscle damage, even without the application of any recovery modality.[44,67]

It is possible that post-training massage could cause further trauma where training has caused tissue damage. In elite junior athletes given warm water-jet massage following high-intensity exercise, serum creatine kinase and myoglobin concentrations were significantly higher in athletes given massage than in those given no special recovery treatment.[5] However, the post-training decrease in power and increase in ground contact time during continuous jumping were less in athletes treated with massage than those given no special recovery, although massage had no effect on drop jump or successive rebound jump heights.[5]

In summary, most evidence does not support massage as a modality that improves recovery or that benefits performance.

2.2 Active Recovery

The investigation of the efficacy of active recovery has mainly been based on its effect on the rate of post-exercise lactate removal. While this effect is well established,[6,68-75] lactate removal, as discussed in section 1.2, does not appear to be a valid indicator of recovery quality. A recent study on rugby players examined post-match recovery using creatine kinase in forearm transdermal exudate samples to indicate muscle damage.[12] The reported rates of recovery were very similar for active recovery, contrast temperature water immersion therapy and wearing lower-body compression garments. All were significantly faster than for passive recovery. This method does not appear to have been specifically validated as an indicator of plasma or interstitial creatine kinase concentrations and others have found no effect of active recovery on post-rugby match recovery.[11] Also, as there is a high level of direct impact in elite rugby matches, the findings cannot be generalised to training and competitive sports perform-

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ances that would not be expected to induce similar levels of muscle damage.

Only one study has examined the effect of active recovery on post-recovery performance following a period representative of the duration of between-training session recovery.[76] Well trained men performed exercise to exhaustion at both 120% and 90% of peak running speed, pre- and post-recovery. They were administered 15 minutes of one of three recovery modalities, active, passive or contrast temperature immersion, after the pre-runs. No significant differences in performance between modalities were found after 4 hours of recovery. Based on current evidence, the common practice of post-training warm-down does not seem to offer much benefit to athletes.

Another consideration is whether active recovery affects the rate of glycogen restoration between training sessions. Studies of the effect of active versus passive recovery on post-exercise muscle glycogen resynthesis have found either a higher rate of resynthesis during passive recovery[77-79] or no difference between active and passive recovery.[80-82] However, two studies reporting no difference found no significant glycogen resynthesis after either passive or active recovery, possibly because the duration of recovery was only 10[82] or 15 minutes,[80] which may be insufficient for significant synthesis to occur. One study[78] that found a higher rate of resynthesis during passive compared with active recovery involved consumption of carbohydrate 1.5 g/kg of bodyweight 10–12 and 130–132 minutes post-exercise during a 4-hour recovery. Although the amount of carbohydrate was less than that recommended for rapid restoration of muscle glycogen (1.2 g/kg immediately after exercise and then 1.2 g/kg/hour for the following 4–6 hours),[2] this study suggests that active recovery may limit glycogen synthesis when athletes attempt to maximise synthesis by consuming carbohydrate post-exercise. No carbohydrate appears to have been consumed during recovery in any of the other studies. Further research is needed to investigate the effects of active and passive recovery on the rate of post-exercise glycogen resynthesis when athletes consume an optimal amount of carbohydrate after exercise. It is also possible that undertaking active recovery immediately post-training may affect the athlete's consumption of carbohydrate during that period.

In summary, evidence that active recovery enhances recovery between training sessions is currently lacking. Active recovery may be detrimental to rapid glycogen resynthesis.

2.3 Cryotherapy

Cryotherapy is used widely to treat acute traumatic injury and may be appropriate as a recovery modality after training and competition that cause some level of traumatic injury, such as in team and contact sports and martial arts. However, evidence about its effectiveness and appropriate treatment guidelines are limited.[83] In a recent review of treatment strategies for DOMS, Cheung et al.[4] concluded that research does not support the efficacy of cryotherapy, apart from an analgesic effect.

Perhaps more important to elite athletes, the rate of strength recovery in resistance-trained males after high-intensity eccentric exercise was not enhanced by post-exercise cryotherapy.[33,84] Similar results have been found in untrained men[85] and women.[86] However, an investigation of recovery from baseball pitching in a simulated game suggests that a combination of ice treatment and light recovery exercise may enhance 24-hour shoulder strength recovery.[87] Cold-water immersion, massage, or active recovery treatments, but not rest, maintained total work output when a high-intensity exercise session was repeated after 24 hours.[88]

The effect of post-exercise cryotherapy on adaptation to training has recently been investigated.[89] In a series of experiments, untrained men undertook leg endurance training or forearm flexor resistance training over 4–6 weeks. Training protocols were designed not to induce DOMS. The cryotherapy comprised of two or one 20-minute cold water immersions for the endurance training experiments and one 20-minute immersion for the resistance training experiments after each training session. The results of these experiments indicate that post-exercise

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cooling lessens the effects of training in untrained men by retarding post-training adaptive processes associated with improvement in performance.[89]

In summary, there is some indication of cryotherapy effectiveness over recovery periods relevant to between training session intervals following exercise that may be more representative of training than that used to induce DOMS.[87,88] However, overall, the evidence supporting cryotherapy as a recovery modality is weak and recent research indicates that it may actually have negative effects on adaptation to training.[89] Further research using training more specific to that used by elite athletes is needed.

2.4 Contrast Temperature Water Immersion

Contrast temperature water immersion entails alternating immersion in warm-to-hot and cold water. As this modality does not appear to induce fluctuations in muscle tissue temperature,[90] it is difficult to attribute a mechanism by which it would enhance recovery. However, contrast water immersion therapy has recently been shown to enhance post-match creatine kinase clearance (estimated from transdermal exudate samples) in rugby players compared with passive recovery.[12] Despite its popularity as a recovery modality, only one study has investigated the effect of this modality on post-recovery performance. Coffey et al.[76] found no difference in performance after a 4-hour recovery between active, passive and contrast temperature water immersion therapies administered for 15 minutes after the initial exercise bout. The subjects performed treadmill runs to exhaustion at both 120% and 90% peak running speed pre- and post-recovery. Given the frequent use of this modality, more research on its effectiveness is warranted.

2.5 Hyperbaric Oxygen Therapy

HBOT involves exposure to whole-body pressure >1 atmosphere while breathing 100% oxygen.[91] The proposed mechanisms by which HBOT may increase the rate of recovery from soft tissue injury include: reduction of local hypoxia and inflammation; promotion of vasoconstriction; reduction of neutrophil adhesion; free radical quenching; control of oedema; enhancement of leukocyte killing; and promotion of collagen synthesis and vessel growth processes.[92] The underlying assumption for its use as a recovery modality is that training sessions cause some degree of trauma and that post-training HBOT enhances recovery by speeding the repair of such trauma.

No studies have examined the effects of HBOT on recovery from training in elite athletes. Studies have used eccentric exercise to induce DOMS-type muscle injury in untrained subjects.[34,91,93,94] Staples et al.[91] undertook a study comprising two experiments where subjects performed 30 sets of 10-repetition maximum (RM), eccentric quadriceps contractions. The first compared HBOT (treatment 0, 24, 48 hours post-exercise), delayed HBOT (treatment 48, 72, 96 hours post-exercise), sham and a control condition (no treatment). The second compared sham, 3 and 5 days of HBOT. No differences in pain scores were found. Contradictory results were reported for mean torque: significant differences between the sham treatment and 5-day HBOT treatment groups in the second, but not first experiment of the study. No other significant differences in mean torque between groups were found. However, recovery of eccentric torque from immediate post-exercise to 96 hours post-exercise was significantly greater in the HBOT group compared with the other groups and over 5 days of HBOT treatment compared with 3 days.

Another study found no differences in the rate of strength recovery, perceived soreness, or arm circumference between HBOT- and placebo-treatments after high-force eccentric elbow flexor exercise.[34] Harrison et al.[93] compared the effect of immediate HBOT, delayed HBOT (treatment begun at 24 hours) and control treatments on recovery after eccentric exercise-induced muscle damage. The groups did not differ in isometric strength, serum creatine kinase concentration, rating of perceived soreness, or MRI-assessed indicators of oedema (cross-sectional area and T_2 relaxation time) during recovery. In another study, HBOT or sham treatment was given 3–4 hours, 24 and 48 hours after five sets of 80% of 1RM calf raises to failure.[94] The

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groups did not differ significantly in peak torque, muscular endurance, muscle cross-sectional area, inorganic phosphate levels, or T_2 relaxation time during recovery. The sham group displayed significantly lower peak isometric torque for the first 2 days post-exercise, but not on days 3 and 5, and significantly higher perceived pain sensation and unpleasantness on day 5. A recent meta-analysis examining the effect of HBOT on DOMS induced in untrained subjects found no evidence of improved speed of recovery and indication of increased interim pain during recovery.[95]

In summary, the published research does not support the effectiveness of HBOT as a recovery modality in the training programmes of elite athletes. The cost of treatment, both of equipment and appropriately qualified personnel, possible risk of oxygen toxicity and the risk of explosion are additional barriers to the use of HBOT.[93,96]

2.6 Nonsteroidal Anti-Inflammatory Drugs

Millions of people throughout the world use NSAIDs because of their pain relief and anti-inflammatory properties.[97,98] These properties also make them an attractive modality to treat athletes and possibly enhance recovery between training sessions. NSAIDs have an anti-inflammatory effect by inhibiting cyclo-oxygenase (COX), an enzyme involved in the synthesis of prostaglandins, potent modulators of inflammation.[99] The efficacy of NSAIDs in attenuating exercise-induced muscle injury has been reviewed elsewhere.[97] The COX-2 inhibitor class of NSAIDs have also recently been reviewed with regard to their use by athletes with acute musculoskeletal injuries.[100] More recent research further heightens the concerns regarding their use by athletes.

There are three known isoforms of COX, of which COX-1 has homeostatic and inflammatory roles and COX-2 is pro-inflammatory as well as having a role in inflammation resolution. NSAIDs differ according to their relative inhibition of COX-1 and COX-2.[101] Traditional NSAIDs are associated with increased risk of serious gastrointestinal and renal problems related to COX-1 inhibition; selective COX-2 inhibitors were developed to treat inflammation while reducing the risk of gastrointestinal toxicity.

Although COX-2 inhibitors give similar pain relief and have greater anti-inflammatory effects, they have recently been linked to increased risk of serious cardiovascular complications.[102-109] These findings have led to the voluntary withdrawal of two COX-2 inhibitor-specific NSAIDs from the market and the issuing of new guidelines by the US FDA on the use of others. It also appears that this increased risk extends to semi-selective and conventional non-aspirin (non-acetylsalicylic acid) NSAIDs[108-110] and it has been suggested that the widespread use of NSAIDs for non-inflammatory pain be reconsidered.[111] Although elite athletes differ in many ways from the populations on which these studies are based and may be at lower risk of cardiovascular disease, the recent findings raise ethical issues about the use of NSAIDS, especially COX-2-specific inhibitors, as an ongoing prophylactic or therapeutic recovery modality.

Although concluding brief use of NSAIDs is beneficial for short-term recovery of muscle function, Lanier[97] noted that the research is contradictory on the effects of NSAIDs on muscle strength and the rate of recovery of muscle function after exercise. The analgesic effect of NSAIDs on the DOMS sensation appears to be related to the degree of soreness.[97] The severity of soreness is typically linked to unaccustomed exercise, and the more unaccustomed the individual is to the intensity and eccentric component of the exercise, the greater the soreness. This suggests that the analgesic benefits of NSAIDs for an elite athlete may be minimal during normal training.

Accumulating evidence from animal studies indicates COX plays an important role in recovery from and adaptation to training.[49,99,112] NSAIDs adversely affect the adaptive response to eccentric exercise in rats and reduce the repeated-bout effect,[49] ibuprofen has recently been shown to inhibit skeletal muscle adaptation to overload training in rats[112] and COX-2-dependent prostaglandin synthesis is required during the early stages of post-trauma mus-

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cle regeneration in mice.[99] Over-the-counter doses of ibuprofen have also been shown to blunt the protein synthesis response to eccentric resistance exercise in untrained men.[113] These studies imply that repeated use of NSAIDs over extended periods might have a detrimental effect on muscle repair and adaptation to training. In summary, the potential for adverse health outcomes and the possibility of a negative effect on recovery and adaptation preclude the use of NSAIDs as a recovery modality.

2.7 Compression Garments

There are three varieties of compression garments: (i) graduated compression stockings worn for the prevention and treatment of deep vein thrombosis; (ii) compression sleeves worn over limbs and joints to provide support or reduce swelling; and (iii) elastic tights and tops worn as exercise clothing. Along with commercial promotion, there appears to have been a lay acceptance that compression garments aid in post-exercise recovery. To date, only a small number of studies have investigated this assumption.

Highly fit male college students who wore graduated compression stockings during both exercise (3 minutes cycling at 100% maximal oxygen uptake) and recovery had lower recovery blood lactate concentrations than when wearing the stockings only during exercise or not at all.[114] As no plasma volume shifts were observed, the authors suggest that the lower values were due to lactate being retained in the muscular bed, rather than greater lactate removal. Chatard et al.[115] have shown that wearing graduated compression stockings during an 80-minute recovery with the legs elevated decreased blood lactate concentrations in elderly trained cyclists and led to a significantly better post-recovery performance than a control trial. Pre- and post-recovery exercise consisted of maximal 5-minute bouts on a cycle ergometer set to a constant braking force. Post-exercise blood lactate concentrations will return to resting levels in time periods much shorter than that typical between training sessions with passive recovery alone and sitting with the legs elevated for a long time after exercise, as used in this study, would seem impractical for young elite athletes. Therefore, these studies do not provide any evidence applicable to recovery between training sessions in this population.

The magnitude of the increase in plasma creatine kinase concentration was less when untrained subjects wore compression sleeves for 5 days after they performed eccentric exercise of the elbow flexors designed to induce muscle damage.[35] The compression sleeves also prevented the loss of elbow range of motion, decreased perceived soreness, reduced swelling and promoted the recovery of force production. Further investigations of the use of compression sleeves as a recovery modality are warranted.

Berry et al.[116] investigated the effect of wearing elastic tights on post-exercise blood lactate concentrations, oxygen consumption and heart rate. No difference was found between wearing the tights during exercise and recovery, during exercise alone or not at all for any variable measured. Recently, wearing a lower body compression garment for 12 hours post-game was found to enhance recovery from muscle damage in rugby players compared with passive recovery, but not compared with active recovery or contrast water therapy.[12] As noted in section 2.2, the trauma associated with rugby games is likely to be much greater than that associated with training. Therefore, the implications of the findings of this study for post-training recovery are unclear. No studies appear to have investigated wearing elastic tights during post-training recovery alone.

2.8 Stretching

Stretching has long been a commonly used modality pre- and post-training, at various other times during recovery periods and within training sessions. Its primary perceived function has been to increase range of motion about joints, and research has shown that this is achieved by various modes of stretching.[117] The efficacy of stretching as an aid to performance is less apparent. There do not appear to be any studies that have investigated the effect of stretching between exercise sessions on performance during post-recovery exercise.

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The majority of research on pre-exercise stretching up to 60 minutes prior to performance has reported a negative effect on explosive power.[118] Pre-exercise stretching has also been shown to decrease 20m sprint time in track and field athletes competing in power events.[119] If the reduction in explosive power reduces the benefit attained from training, stretching in the last 60 minutes of a recovery period before an explosive training session may be inappropriate.

An inverse relationship between flexibility and running economy has been found in international and well trained sub-elite male distance runners,[8,120] whereas no relationship between these variables was found in female college track athletes.[10] In combination, these studies suggest no benefit of increased flexibility on distance running performance. This is supported by data showing no significant effect of a 10-week stretching programme on sub-maximal running economy.[121] Therefore, in sports where running is a major component, stretching during recovery periods appears to have no long-term benefit.

While a mechanism by which stretching may enhance the recovery process has yet to be identified, it has been suggested that stretching may disperse oedema accumulated during tissue damage.[122] As discussed earlier in section 1.4, the inflammation process, a component of which is oedema, may be important in recovery and adaptation. Therefore, dispersion of oedema as a general principle may not be an appropriate goal during recovery. DOMS involves an acute inflammatory response with oedema formation, and stretching following eccentric exercise appears to have no preventative effect on DOMS.[4] This could possibly be due to either stretching having no effect on oedema or any oedema removal due to stretching having no beneficial effect on DOMS.

Stretching is commonly seen as a technique for reducing the possibility of injury. Research indicates that stretching does not lead to injury reduction[117] or reduced risk.[123] In summary, there is no apparent short- or long-term benefit from stretching as a recovery modality.

2.9 Electromyostimulation

EMS involves the transmission of electrical impulses via surface electrodes to peripherally stimulate motor neurons eliciting muscular contractions. It has been suggested that these contractions may be advantageous to recovery due to increased blood flow via the 'muscle pump effect', which may enhance tissue repair.[124] Martin et al.[124] examined recovery of voluntary and electrically invoked torque up to 96 hours following one-legged downhill running. They found no difference between EMS, passive and active recovery interventions. Similarly, Lattier et al.[125] found low-frequency EMS did not enhance the recovery of the voluntary force-generating capacity of the knee extensors when compared with passive or active recovery. Transcutaneous electrical nerve stimulation has also been found to have no hypoalgesic effect on pain associated with DOMS up to 72 hours following eccentric exercise to exhaustion.[126] In summary, in the few studies conducted so far, EMS did not enhance the recovery process.

2.10 Combination Modalities

The potential benefits of combined recovery modalities have not been investigated thoroughly. A combination of active recovery and massage, during a 20-minute recovery period in cyclists, was better at maintaining maximal 5km time-trial performance than active recovery, massage, or passive recovery alone when trials were separated by 2 hours.[75] The possible mechanisms underlying these findings are unclear and they cannot be extrapolated to the between-training session recovery periods of athletes. There are many potential combinations of recovery modalities. Future research in this area should be undertaken based on an hypothesised underlying mechanism.

3. Conclusions

Most studies examining the efficacy of recovery modalities have focused on post-exercise lactate removal or recovery of untrained individuals from exercise-induced muscle injury and the symptoms

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of DOMS. The relevance of these to recovery between training sessions for elite athletes has yet to be shown. Blood lactate levels return to baseline with rest alone in a time-frame shorter than is common between training sessions. The applicability of exercise-induced muscle injury and DOMS studies using untrained individuals to training in elite athletes is questionable. With regard to recovery between stressful training sessions, evidence as to any positive effective of current recovery modalities is lacking. Massage, active recovery, contrast temperature water immersion, HBOT, stretching and EMS do not appear to be advantageous. NSAIDs have potential negative health outcomes and may negatively affect muscle repair and adaptation to training. The possible efficacy of both cryotherapy and compression garments needs more investigation for clarification. Because recovery modalities are gaining wide acceptance among elite athletes and sports are investing time and money in providing these modalities, further research and better consideration of the evidence of their effectiveness appear warranted.

4. Recommendations for Future Research

To better understand the effect of modality on athlete recovery between training sessions, future research might consider:

- Developing experimental models to examine the value of recovery modalities as integral components of training specific to the needs of elite athletes. Consideration should be given to temporality, mode and load (volume and intensity) of training.
- The use of mathematical modelling of elite training, recovery modalities and performance over the training season to examine the potential benefits of recovery modalities on performance outcomes.
- Whether recovery modalities can enhance performance by enabling elite athletes to tolerate greater training loads or by augmenting the performance-enhancing effect of training at a given load.
- The possible effect of recovery modality on the rate of post-exercise glycogen synthesis when the athlete follows current dietary guidelines to enhance glycogen synthesis.
- Any influence of recovery modalities on markers of overtraining.
- The possible interaction between recovery modality and inflammation and its effect on recovery from and adaptation to training.

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