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REVIEW ARTICLE

What are the Physiological Mechanisms for Post-Exercise Cold Water Immersion in the Recovery from Prolonged Endurance and Intermittent Exercise?

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Abstract Intense training results in numerous physiological perturbations such as muscle damage, hyperthermia, dehydration and glycogen depletion. Insufficient/untimely restoration of these physiological alterations might result in sub-optimal performance during subsequent training sessions, while chronic imbalance between training stress and recovery might lead to overreaching or overtraining syndrome. The use of post-exercise cold water immersion (CWI) is gaining considerable popularity among athletes to minimize fatigue and accelerate post-exercise recovery. CWI, through its primary ability to decrease tissue temperature and blood flow, is purported to facilitate recovery by ameliorating hyperthermia and subsequent alterations to the central nervous system (CNS), reducing cardiovascular strain, removing accumulated muscle metabolic by-products, attenuating exercise-induced muscle damage (EIMD) and improving autonomic nervous system function. The current review aims to provide a comprehensive and detailed examination of the mechanisms underpinning acute and longer term recovery of exercise performance following post-exercise CWI. Understanding the mechanisms will aid practitioners in the application and optimisation of CWI strategies to suit specific recovery needs and consequently improve athletic performance. Much of the literature indicates that the dominant mechanism by which CWI facilitates short term recovery is via ameliorating hyperthermia and consequently CNS mediated fatigue and by reducing cardiovascular strain. In contrast, there is limited evidence to support that CWI might improve acute recovery by facilitating the removal of muscle metabolites. CWI has been shown to augment parasympathetic reactivation following exercise. While CWI-mediated parasympathetic reactivation seems detrimental to high-intensity exercise performance when performed shortly after, it has been shown to be associated with improved longer term physiological recovery and day to day training performances. The efficacy of CWI for attenuating the secondary effects of EIMD seems dependent on the mode of exercise utilised. For instance, CWI application seems to demonstrate limited recovery benefits when EIMD was induced by single-joint eccentrically biased contractions. In contrast, CWI seems more effective in ameliorating effects of EIMD induced by whole body prolonged endurance/intermittent based exercise modalities.

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Key Points

The current review provides a comprehensive examination of the mechanisms underpinning acute and longer term recovery of exercise performance following post-exercise cold water immersion (CWI).

Acute recovery mechanisms include the amelioration of hyperthermia mediated fatigue, reductions in cardiovascular strain, removal of accumulated muscle metabolic by-products. Longer term mechanisms include improvements in the autonomic nervous system function and decreases in exercise-induced muscle damage.

Understanding the mechanisms will aid practitioners in the application and optimisation of CWI strategies to suit specific recovery needs and consequently improve athletic outcomes.

1 Introduction

Endurance training results in profound cardiovascular and skeletal muscle adaptations that co-ordinately improve fatigue resistance and enhance exercise capacity. Some of the centrally occurring adaptations following endurance training include an increase in stroke volume/cardiac output [1, 2], ventricular hypertrophy [3], enhanced cardiac contractile properties [4], blood volume expansion [5] and haematological changes [6, 7]. In the skeletal muscles, an increase in mitochondrial content [8], metabolic enzymes [9], capillary density [10], transformations from fast to slow fibre-types [11] as well as improved conduit vessel and microvascular function [12, 13] are typically evident following endurance training. Taken together, human tissues demonstrate remarkable ability to alter morphological, metabolic and functional characteristics to improve aerobic function and better accommodate changes imposed by physical activity.

In order to drive such adaptations, progressive and continuous increases in training stimuli are needed, at least until genetically pre-disposed upper limits are reached [14]. This increase in training load and associated physiological stress induced by exercise has been termed progressive overload and has had an important role in the high training loads currently performed by athletes. For instance, elite long distance runners are reported to train 10–16 sessions•week^{-1}, with weekly running mileage amounting to between 150 and 200 km [15, 16]. Numerous physiological perturbations such as muscle damage, hyperthermia, dehydration and glycogen depletion can be expected as a consequence of such immense training stress [17, 18]. Insufficient restoration of these physiological alterations might result in sub-optimal performance in subsequent training sessions, with chronic imbalance between training stress and recovery resulting in overreaching or overtraining syndrome [19].

The use of recovery interventions between training sessions has emerged as a potential mechanism to enhance post-exercise recovery [17, 18]. One such modality is the use of post-exercise cold water immersion (CWI), This recovery strategy is widely utilised among athletes of all levels in both hot and normal environments in an attempt to ameliorate hyperthermia-induced fatigue and reduce exercise-induced muscle damage [20–25]. Indeed, post-exercise CWI has been shown to maintain subsequent exercise performance [20], preserve day-to-day performance [26, 27] and in some [22] but not all cases [28] attenuate the increase in indirect markers of muscle damage. A number of excellent reviews have recently examined the influence of CWI, and other hydrotherapy modalities, on acute exercise performance and recovery [29–33]. While there is strong evidence to indicate that post-exercise CWI may enhance both short and longer term recovery, the precise factors responsible for such improvements are unclear, with numerous mechanisms proposed. These putative mechanisms include the amelioration of hyperthermia and subsequent alterations to the central nervous system (CNS), reductions in cardiovascular strain, removal of accumulated muscle metabolic by-products, improvements in autonomic nervous system function, decreases in exercise-induced muscle damage (EIMD) and delayed onset muscle soreness. To date, a review specifically examining these potential mechanisms is currently lacking. Elucidating the mechanisms by which CWI enhances recovery likely provides practitioners with an evidence-based platform, from which this modality can be utilised to target specific recovery objectives (e.g. ameliorate hyperthermia vs. EIMD). Moreover, clearly defining the recovery mechanisms enables a more guided practice with regards to periodization of recovery alongside longer term goals for training-induced adaptation. This is especially important because adaptations in recovery are complex, with evidence indicating that CWI may enhance muscle oxidative adaptations to endurance training [34–36] while impeding hypertrophic/strength adaptations derived from resistance training [37, 38]. As such, the purpose of this review is to provide a comprehensive and detailed examination of the mechanisms that may be responsible for acute and longer term recovery of exercise performance following post-exercise CWI. Within this review, acute recovery is defined as the post-exercise period of $\leq$60 min, while longer term recovery stipulates a post-exercise time frame between 2 h and 1 week.

2 Acute Recovery Mechanisms Associated with Cold Water Immersion

2.1 Central Nervous System Fatigue

CNS fatigue refers to the decrement in force production due to the reduction in voluntary activation (VA) and neural drive to the muscle [39]. The progressive rise in body temperature and subsequent hyperthermia is strongly implicated in the development of central fatigue during exercise [40–43]. A primary mechanism by which CWI is suggested to enhance performance is by rapidly reducing body temperature, given that the thermal conductivity of water is 25 times greater compared with that of air [44]. This enhances the capacity for heat storage, allowing greater energy expenditure before physiological limitations in core body temperature (>40 °C) are attained (Fig. 1). While a number of studies have demonstrated the effectiveness of CWI in reducing post-exercise body temperature and improving subsequent exercise performance [20, 21, 25, 45, 46], evidence of CNS involvement has only recently been demonstrated [24, 47]. For instance, when compared with a control trial, Pointon et al. [47] observed immediate improvements in maximal voluntary contraction (MVC) force and VA following CWI (2 × 9 min at ~9 °C), which was performed following 60 min of intense intermittent running in the heat. Similarly, Minett et al. [24] reported improved recovery of MVC force and VA at 1 h post-exercise when CWI (20 min at 10 °C) was applied following a 70-min intermittent running protocol performed in the heat. However, it must be noted that contradictory results were evident at 24 h post-exercise with Minett et al. [24] reporting improved and Pointon et al. [47] reporting attenuated MVC force following CWI treatments. These results indicate that while CWI is effective in improving acute recovery via ameliorating hyperthermic-induced CNS fatigue, the efficacy of CWI in aiding longer term recovery likely involves other mechanisms.

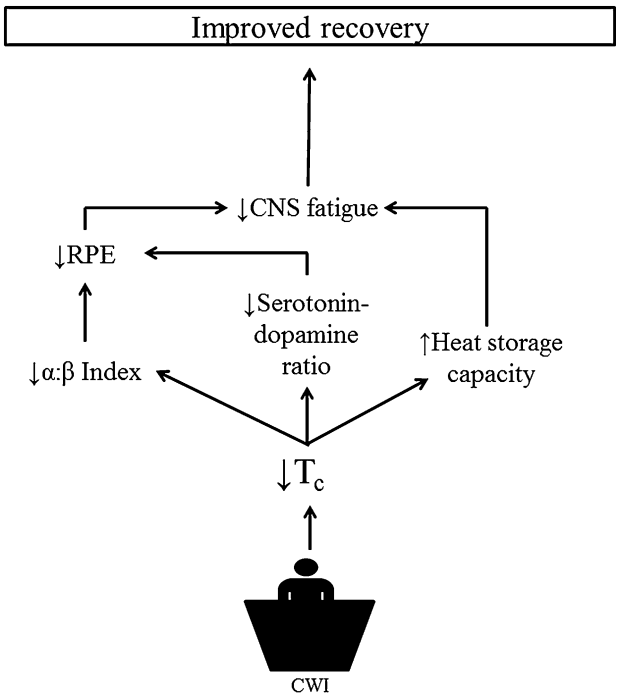

Fig. 1 Suggested mechanisms by which CWI enhances recovery from CNS fatigue. The decrease in core body temperature (T_c) following CWI results in a reduced α:β index and serotonin-dopamine ratio. This is reflected in a reduced sense of perceived exertion (RPE) which closely mirrors the extent of central fatigue during exercise. The decrease in T_c also increases the heat storage capacity, allowing higher energy expenditure before the physiological T_c (>40 °C) associated with voluntary exhaustion is reached. *CWI* cold water immersion, *CNS* central nervous system, ↑ increase, ↓ decrease

The effects of hyperthermia on brain function are highly complex and involve changes in electroencephalographic activity [48, 49], cerebral neurotransmitters [50, 51], cerebral blood flow/oxygenation [52] and cerebral metabolism [53]. A detailed commentary on the aetiology of these factors is beyond the scope of this review. Nevertheless, there is evidence to suggest that CWI may alleviate some of these exercise-induced cerebral perturbations either directly or via its effect on core temperature (T_c) (Fig. 1). For instance, CWI performed in between successive bouts of exercise might ameliorate CNS fatigue by modifying the α:β index; an electroencephalographic parameter that progressively increases during exercise-induced hyperthermia and is suggested to reflect a decreased state of arousal and alertness [42, 48]. The basis for this speculation is gathered from studies that have demonstrated reduced ratings of perceived exertion (RPE) during exercise following CWI [54], which has been shown to be well correlated with changes in α:β ratio during hyperthermic exercise [49]. Further evidence is gathered from a recent study by De Pauw et al. [55], where post-exercise CWI has been shown to increase global electroencephalographic β activity (and presumably overall α:β ratio), which was otherwise depressed following prolonged cycling in the heat. Although performances during a subsequent 12-min simulated time trial were similar between CWI and control conditions, CWI resulted in more even pacing strategy, such that higher power outputs were better maintained at the onset of exercise [55]. As such, it seems that CWI is able to alter the α:β index and the resulting functional outcome is potentially a change in overall pacing profile. Further studies are clearly warranted to better understand the mechanisms underpinning CWI, electroencephalographic activity and pacing.

It is also suggested that CWI might ameliorate CNS fatigue by enhancing cerebral perfusion and oxygenation [24], which has been shown to be depressed during

exercise-induced hyperthermia and implicated in the development in CNS fatigue [52, 53]. The restoration of cerebral perfusion and presumably oxygenation is purportedly achieved through increases in mean arterial pressure and cardiac output, as a consequence of increased central blood volume following CWI [24, 56] (see Sect. 2.3). However, contrary to this hypothesis, Minett et al. [24] showed that post-exercise CWI further exacerbated the exercise-induced reductions in prefrontal cortex blood perfusion and oxygenation, despite an enhanced recovery in quadriceps MVC force and VA. These findings therefore indicate that the mechanisms by which CWI might ameliorate central fatigue are dissociated from changes in cerebral perfusion/oxygenation.

The alteration of cerebral neurotransmitters, namely the dopaminergic and serotonergic systems is an alternate mechanism by which cold exposure may attenuate the development of CNS fatigue (Fig. 1). These systems influence mood state, sleep, emotion, motivation, attention, reward and thus have been implicated in the development of CNS fatigue [50, 51, 57]. For instance, treatment with a dopamine re-uptake inhibitor or serotonin antagonists has been shown to improve endurance performance in humans and rodents, respectively [58, 59]. However, the role of serotonin in central fatigue mechanisms is less clear in humans, as serotonin re-uptake inhibition has been shown to have no influence on endurance performance [60]. Nevertheless, Mundel et al. [61, 62] found that facial cooling significantly reduced blood prolactin concentration, which is stimulated by serotonin and inhibited by dopamine. In this regard, it is highly plausible that CWI might facilitate acute recovery via a similar mechanism. However, studies specifically investigating effect of post-exercise CWI treatment on the activity of these neurotransmitters are currently lacking and this warrants further investigation.

2.2 Cardiovascular Strain

CWI application may facilitate short-term recovery from exercise through alleviating cardiovascular strain. Indeed, cardiovascular strain is elevated during exercise in the heat as blood flow is redirected from the active musculature to the cutaneous circulation for heat dissipation and temperature regulation [63]. The redirection of blood to the peripheries results in reduced central blood volume, causing a decline in muscle blood flow and, as a consequence, may impair oxygen (O_2) delivery and performance [64, 65]. CWI results in rapid cutaneous vasoconstriction, redirecting blood back into the central circulation. Moreover, the decrease in T_c resulting from CWI reduces the thermoregulatory demand for heat dissipation and therefore limits the need to redirect blood to the skin (Fig. 2).

The results of early studies demonstrating reduced cardiovascular strain and circulatory conflict following CWI were largely inferred from changes in heart rate responses. For instance, Hayashi et al. [66] first reported a reduction in heart rate during submaximal exercise in the heat as a result of 5 min of CWI, which was performed after an initial 40 min cycling bout. Utilising similar experimental designs (i.e. CWI in between two exercise bouts), numerous studies have since demonstrated decreased heart rate during rest [24, 47, 54] or during subsequent exercise bouts undertaken in both neutral [25] and hot ambient conditions [46, 67]. Further support for CWI in ameliorating cardiovascular strain is gathered from recent studies directly investigating haemodynamic changes resulting from post-exercise CWI [67–69]. These studies collectively demonstrate reduced limb blood flow to, or reduced blood volume across the exercised muscle following CWI, and hence support the notion that CWI might ameliorate cardiovascular strain by redistributing blood flow from the periphery to the core [67–69]. However, it must be mentioned that limitations in the techniques utilised within these studies preclude definitive evidence of reduced muscle perfusion per se following post-exercise CWI [67–69]. Interestingly,

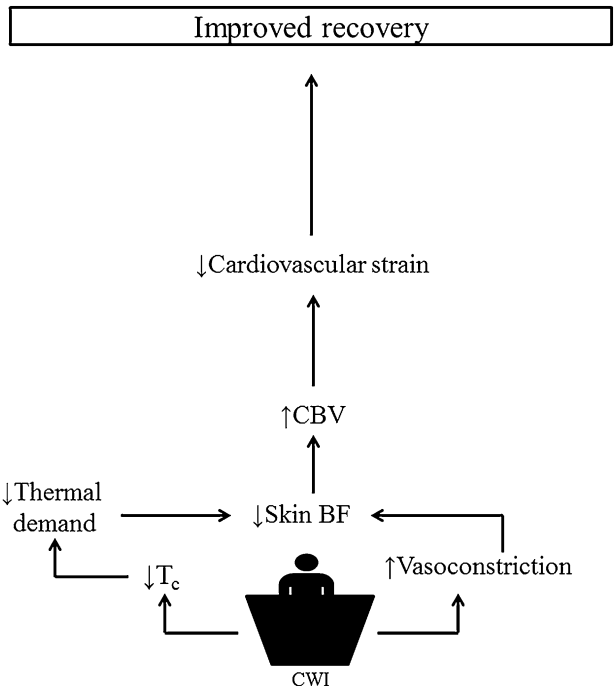

Fig. 2 Suggested mechanisms by which CWI attenuates cardiovascular strain and improves recovery. CWI reduces blood flow to the skin (skin BF) through cutaneous vasoconstriction and reduced thermoregulatory demand to dissipate heat. The reduction in skin BF results in an increased central blood volume (CBV), leading to the improved availability of oxygen and substrate for the exercising muscle. *CWI* cold water immersion, T_c core temperature, ↑ increase, ↓ decrease

in terms of increasing central circulation, Mawhinney et al. [68] recently demonstrated that lower limb immersion at 22 °C decreased femoral artery blood flow and conductance as well as thigh and calf vascular conductance to a similar extent compared to immersion at 8 °C, despite skin and muscle temperatures being lower following the 8 °C immersion. This indicates that additional treatment benefits in ameliorating other aspects of recovery [i.e. CNS fatigue (Sect. 2.1), exercise-induced muscle damage (Sect. 3.2)] are likely to be mediated through the effects of reduced tissue temperature rather than further reductions in muscle blood flow.

2.3 Muscle Metabolite Removal

High-intensity exercise elicits the formation and accumulation of metabolites that are implicated in the development of muscle fatigue [70, 71]. Post-exercise CWI is suggested to accelerate the removal of these muscle metabolites, consequently improving metabolic recovery from intense exercise bouts [47, 72, 73]. The transportation of metabolites from the muscle into the central circulation is facilitated by the combined effects of hydrostatic pressure, as well as limb arterial and cutaneous vasoconstriction. This in turn facilitates haemodilution and blood displacement from the peripheral regions (Fig. 3) [56, 74, 75]. Haemodilution refers to fluid shifts from the interstitial to the intravascular spaces. Fluids leaving the interstitial space are then rapidly replaced by intracellular fluid, resulting in a higher extracellular (intravascular) to intracellular fluid content [56]. This consequently results in an intracellular-intravascular osmotic gradient, facilitating the efflux of intracellular constituents and metabolic by-products from the intracellular and interstitial space into the peripheral circulation. This osmotic gradient is further accentuated by cold exposure, possibly due to increased pressure gradient as a result of cutaneous vasoconstriction [56, 76]. Blood displacement through hydrostatic pressures, as well as limb arterial and cutaneous vasoconstriction further facilitates the removal of metabolites from the peripheries into the central (i.e. intrathoracic) circulation [75, 77]. For instance increased hydrostatic pressure has been shown to displace blood from the splanchnic, abdominal regions and to a lesser extent the leg regions by increasing central venous pressures [75, 77]. Moreover, central circulation may be further augmented by decreases in arterial limb and cutaneous blood flow due to vasoconstriction in the limb artery and subcutaneous network [68]. However, it should also be noted that while acutely facilitating blood flow from the periphery to central circulation, CWI induced peripheral vasoconstriction may also reduce muscle blood flow. Such a reduction in blood flow may compromise oxygen and nutrient delivery, enhance reliance on anaerobic metabolism and be detrimental rather than beneficial to recovery.

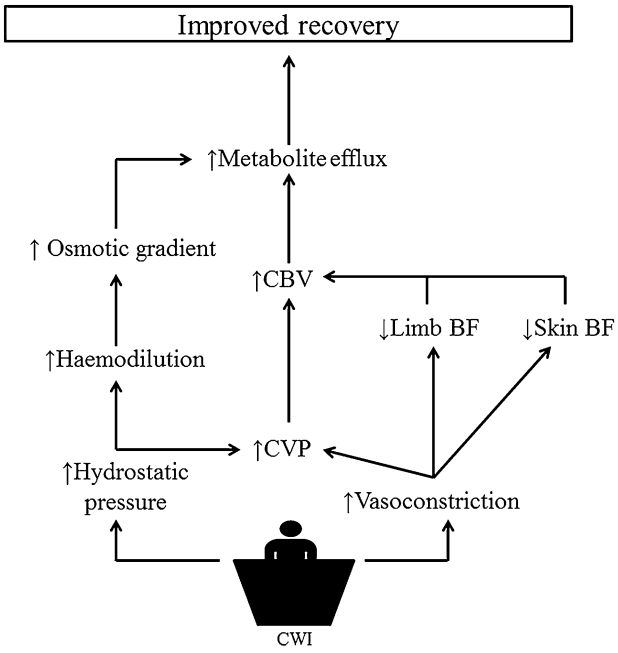

Fig. 3 Mechanisms by which CWI is suggested to improve clearance of post-exercise muscle metabolites and improve recovery. The increase in osmotic gradient resulting from haemodilution drives the efflux of intracellular constituents and metabolic by-products from the extravascular space into the peripheral circulation. These metabolites are subsequently displaced from the peripheries into the central circulation through the effects of vasoconstriction and hydrostatic pressures. *CWI* cold water immersion, *CVP* central venous pressure, *CBV* central blood volume, *BF* blood flow, ↑ increase, ↓ decrease

This conflicting response to CWI may be responsible for the limited evidence demonstrating enhanced metabolite removal following CWI. Indeed, a plethora of studies have observed no change in blood pH [47, 72, 73] or on the clearance of metabolites such as potassium, choride [72] or blood lactate [46, 47, 66, 72, 73, 78] following CWI. In actual fact, some studies have reported a tendency for attenuated clearance of blood lactate when compared with passive resting [25] or light active recovery at 30 to 40 % of peak cycling power output [54, 67]. Yet, the attenuation of lactate clearance in the aforementioned studies was not found to impair performance during subsequent exercise [25, 54, 67]. Instead, CWI resulted in improved performance in all of these studies, indicating that the benefits of CWI were associated with mechanisms other than alterations in local metabolic products, such as altering thermal and cardiovascular strain [25, 54, 67].

While CWI does not seem to enhance post-exercise blood lactate clearance, evidence suggests it may alter

blood lactate kinetics during subsequent high-intensity (30–60 s) exercise bouts. For instance, while Parouty et al. [78] found no effect of CWI (5 min at 14–15 °C) on blood lactate clearance following 100 m (~60 s) swimming performance, they found increased blood lactate accumulation during a subsequent 100 m swimming bout. In contrast, Crowe et al. [79] found reduced blood lactate accumulation during the second bout of 30-s cycle sprint following CWI (15 min at 13–14 °C) treatment. While the disparity in lactate kinetics data between the two studies may be due to differences in immersion duration, it must be noted that exercise performance was impaired following CWI treatment in both studies [78, 79]. Indeed, it is generally accepted that CWI is detrimental to short duration (>30 s) high-intensity sprint performance, possibly due to lowered muscle temperature and subsequent impairments in muscle contractile function [80].

2.4 Autonomic Nervous System Function

Cardiac autonomic nervous system function is considered an important global marker of athlete recovery status and ability to train/perform [81, 82]. Specifically, indices of parasympathetic activity have been shown to be significantly correlated with numerous exercise-induced physiological perturbations during the recovery period, including changes in plasma epinephrine levels [83], blood lactate [84], blood pH [85] and arterial oxygenation [86]. Accordingly, monitoring the time course in the restoration of cardiac parasympathetic activity seems a logical indicator of global body recovery and may be a useful tool to easily and non-invasively assess the recovery status of athletes or the effectiveness of recovery interventions. CWI is an ideal method to accelerate parasympathetic reactivation (Fig. 4) due to its ability to increase central blood volume (see Sects. 2.3 and 3.3) [75–77, 87], which consequently results in increased stroke volume and cardiac output [76, 77]. These changes consequently activate the arterial and cardiopulmonary baroreflexes [88], inhibiting sympathetic activity and augmenting parasympathetic activation, leading to bradycardia [77, 88].

Given the sound rationale indicating that CWI could enhance overall physiological recovery through augmenting parasympathetic activation, it is somewhat surprising that only a few studies have investigated the effects of post-exercise CWI on indices of parasympathetic reactivation [27, 78, 89–92]. In these studies, it has generally been shown that while post-exercise CWI enhanced parasympathetic activation and improved sense of perceptual recovery, subsequent exercise performance was either not enhanced [89, 90] or impaired [78]. This might be due to

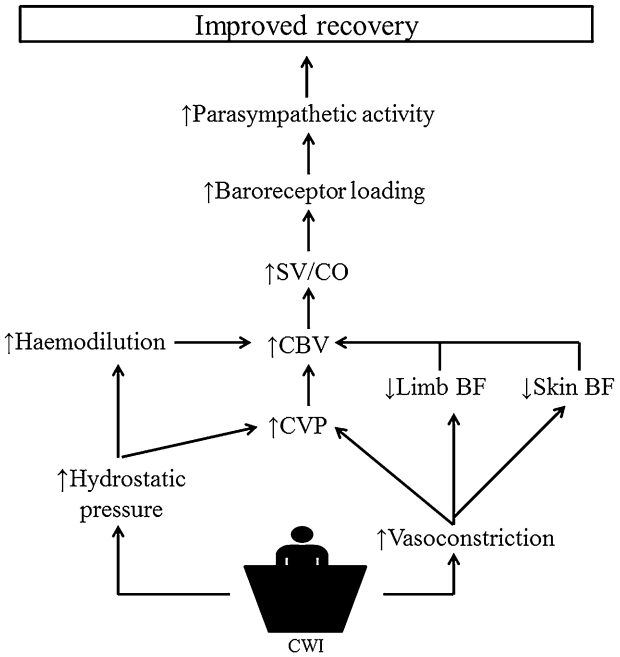

Fig. 4 Mechanisms of parasympathetic reactivation following CWI. The increase in hydrostatic pressures and vasoconstriction following CWI increases central blood volume (CBV) and consequently stroke volume (SV) and cardiac output (CO). Higher SV and CO activate the arterial and cardiopulmonary baroreflexes, inhibiting sympathetic activity and augmenting parasympathetic activation. *CWI* cold water immersion, *BF* blood flow, *CVP* central venous pressure, ↑ increase, ↓ decrease

the fact that sympathetic activation increases skeletal muscle O_2 consumption and glucose metabolism, and has a positive inotropic effect on contracting skeletal muscles [93]. In this regard, parasympathetic reactivation via CWI between high-intensity bouts might counteract subsequent performance. Alternatively, the lack of performance gain observed in these studies may be due to the cooling duration employed (i.e. 5 min at 14 °C) and the performance task involved. Specifically, the CWI protocol (5 min at 14 °C) utilised by Stanley et al. [90] would have minimal influence on post-exercise T_c. This, coupled with the prolonged recovery (160 min) separating exercise bouts, is likely to be responsible for the limited effect of CWI, compared with control. Conversely, the performance protocols utilised by Buchheit et al. [89] and Parouty et al. [78] were sprint based lasting ~60 to 80 s (i.e. 1-km cycle and 100-m swim, respectively), and thus not likely to have resulted in significant thermoregulatory strain necessary for CWI to be effective in the short term. Moreover as mentioned (see Sect. 2.3), decrements in muscle temperature following CWI are detrimental to sprint performance [79, 80] although it is contentious if the CWI protocol utilised (5 min at 14–15 °C) in these studies [78, 89] could have considerably reduced muscle temperatures.

3 Longer Term Recovery Mechanisms Associated with Cold Water Immersion

3.1 Autonomic Nervous System Function

While the effects of parasympathetic re-activation on subsequent performance seem counteractive, regular use of this recovery modality (5 min at 10–14 °C) seems beneficial with regards to longer term vagal modulation and training performance [27, 91]. For instance, regular CWI following swim training sessions increased vagal related heart rate variability indices at rest and during a 1-week training period, indicating improved overall physiological recovery [91]. Stanley et al. [27] indicated that despite no differences in vagal related heart rate variability indices, regular post-exercise CWI recovery during an intensified training block (3 days intense, 2 days recovery) resulted in greater self-selected power outputs. Given that the indices investigated by Stanley et al. [27] were inversely related to training intensity, the data indicate that the superior training performances by the athletes were due to an enhanced CWI mediated recovery. Taken together, these studies indicate that parasympathetic reactivation via post-exercise CWI may be detrimental to subsequent high-intensity performance. However, the limited data currently available also show that regular CWI application improves day to day training performance and physiological status, as assessed by indices of heart rate variability.

3.2 Glycogen Re-synthesis

Fatigue during endurance exercise appears to coincide with significant reductions in muscle glycogen availability [71, 94]. Indeed, it is well established that pre-exercise muscle glycogen content is well correlated with performance [95], with carbohydrate loading often resulting in significant improvements in endurance performance [96]. The restoration of muscle glycogen is therefore considered to be one of most crucial physiological components of recovery from prolonged moderate-intensity or intermittent high-intensity exercise [18, 97]. Glycogen replenishment is especially important for athletes training or competing multiple times per day or on successive days.

To date, research examining the influence of CWI and body cooling on post-exercise muscle glycogen synthesis is equivocal, with studies reporting either no effect [98–100], or attenuated glycogen synthesis [101] following cooling interventions. Specifically, Gregson et al. [98] and Slivka et al. [99, 100] demonstrated no differences in post-exercise glycogen synthesis following CWI (10 min at 8 °C) or cold air exposure (3–4 h at 7 °C) when compared with a control condition. In contrast, Tucker et al. [101] reported attenuated muscle glycogen repletion when localised quadriceps cooling via ice pack application was undertaken at 30 min intervals throughout a 4-h recovery period. Differences in cooling modality and duration which would alter muscle temperature, shivering thermogenesis and blood flow responses could account for the disparity in findings between these studies. For instance, Gregson et al. [98] reported intramuscular temperatures of 30–35 °C at 1- to 3-cm depth immediately post-CWI, while Tucker et al. [101] attained muscle temperatures of ~25 °C at a 4.3-cm depth, which was maintained for 4-h following cooling. This indicates that cooling resulting in prolonged decrements in muscle temperature could potentially attenuate post-exercise glycogen synthesis. However, recent studies have also shown no effect in muscle glycogen re-synthesis despite utilising an aggressive post-exercise cooling strategy (3–4 h at 7 °C air) [99, 100]. One possibility is that muscle contractions during shivering thermogenesis evident in these studies might have attenuated cold-induced decrements in muscle temperature and blood flow. Alternatively, shivering could have resulted in contraction dependent glucose transporter 4 (GLUT4) translocation to the cell membrane, facilitating glycogen repletion [102]. Unfortunately, neither muscle temperature nor GLUT4 trafficking were reported in these studies [99, 100]. Irrespectively, it is plausible that the presence of shivering thermogenesis during whole body cooling may counteract the negative effects on muscle glycogen synthesis.

3.3 Exercise-Induced Muscle Damage

Cryotherapy is a well-recognised treatment modality for acute traumatic injuries [103]. As such, it is somewhat befitting that CWI is often used as a recovery strategy to treat EIMD following training sessions. CWI is suggested to ameliorate EIMD via several mechanisms associated with localised cooling, hydrostatic pressures and redistribution of blood flow (Fig. 5) [31, 103, 104]. For instance, CWI is suggested to promote recovery by reducing muscle oedema [104, 105]. Presence of oedema impedes O_2 delivery to the muscles, as mechanical compression of the local capillaries is increased [105], resulting in an increased transit distance between capillaries and muscle fibres for O_2 exchange [106]. CWI reduces oedema by decreasing incoming blood flow and facilitating the clearance of peripheral fluid. These effects are collectively mediated through cold-induced vasoconstriction [68, 69, 107] and hydrostatic pressures [56, 75–77], leading to an increase in central blood volume. As detailed in Sect. 2.3, vasoconstriction and hydrostatic effects increase central blood volume by increasing the central venous pressure and facilitating the movement of fluids from the

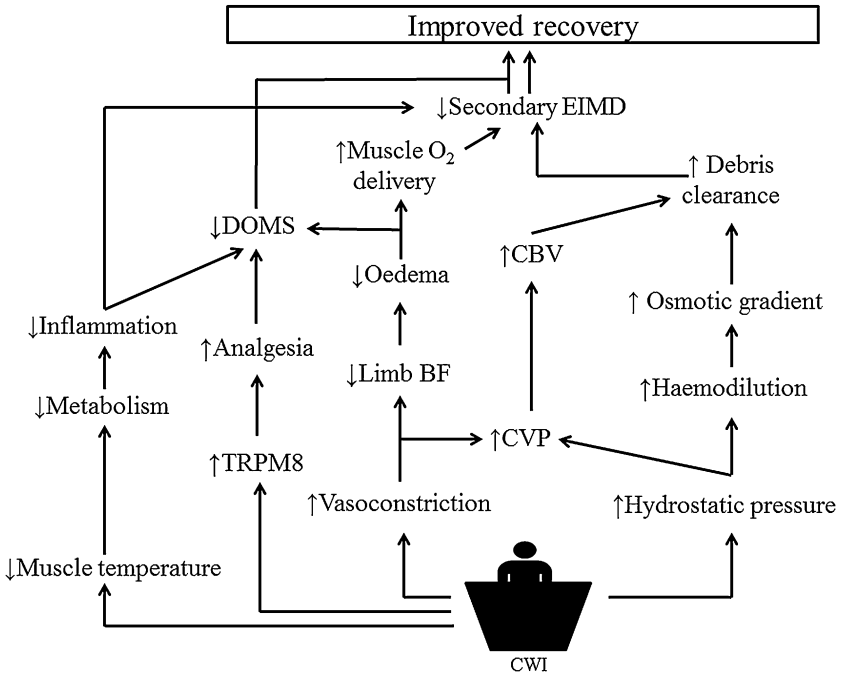

Fig. 5 Suggested mechanisms by which CWI improves recovery from EIMD. The increase in osmotic gradient resulting from haemodilution drives the efflux of debris from the extravascular space into the peripheral circulation, whence it is subsequently facilitated into the central circulation through the effects of vasoconstriction and hydrostatic pressures. Vasoconstriction also reduces muscle blood flow (muscle BF), leading to athe decrease in oedema and a resultant improvement in muscle O_2 delivery. The decrease in intramuscular metabolism following CWI reduces inflammatory events. Collectively, the enhanced debris clearance, improved muscle O_2 delivery and reduced inflammation reduce secondary EIMD, thus improving recovery. The decrease in inflammation and oedema may aid perceived perceptual recovery by through alleviating DOMS. Moreover, the analgesic effects of CWI may directly reduce the sensation of DOMS through TRPM8-mediated mechanisms. *EIMD* exercise-induced muscle damage, *CWI* cold water immersion, *CVP* central venous pressure, *CBV* central blood volume, *DOMS* delayed onset muscle soreness, *TRPM8* transient receptor potential cation channel M8, ↑ increase, ↓ decrease

intracellular and interstitial (extravascular) spaces to the intravascular compartments, respectively [56, 74–77]. Extravascular to intravascular fluid movements are also suggested to promote recovery from EIMD by facilitating the clearance of dead tissue cells and debris [104]. Indeed, movement of fluids from the extravascular to the intravascular compartments [74] results in an intracellular-extracellular osmotic gradient, hence encouraging the translocation of cellular debris and necrotic tissue from the local muscle into the central circulation [104]. Finally, cold-induced decrements in muscle temperature [35] further reduce intramuscular metabolism [69], which may minimise extraneous damage due to hypoxic cell death and inflammatory events [108, 109].

While the decrease in inflammation and oedema will likely reduce delayed onset muscle soreness (DOMS) [110], it is noteworthy that CWI through its analgesic effects may directly modulate the sensation of DOMS, consequently improving perceptual recovery (Fig. 5). The sensation of DOMS is likely due to the activation of group III and group IV muscle nociceptive afferent neurons [111]. Cold exposure in turn, has been shown to activate the transient receptor potential cation channel M8 (TRPM8) receptors located in the Aδ and C fibers; the cutaneous equivalents of muscle group III and group IV afferents, respectively [112, 113]. Once activated, TRPM8 mediates analgesia through inhibitory inputs either through spinal inhibitory interneurons or directly to nociceptors [113]. Improved perception of DOMS is indeed critical for the recovery of exercise performance, as MVC force has been shown to be impaired in the presence of muscle pain (independent of EIMD), induced by infusion of hypertonic saline [114]. Such reasoning is in line with the growing body of evidence implicating CNS-mediated mechanisms in facilitating longer term athletic recovery [115].

Despite the sound mechanistic evidence, applied research on the efficacy of CWI in facilitating the recovery of EIMD has been shown to be rather controversial, with studies showing improved [22, 116], unchanged [117, 118] or impaired [45, 47] recovery of muscle function and/or indirect muscle damage markers (i.e. DOMS and creatine kinase) following CWI. Accordingly, the exercise modality used to induce EIMD seems to influence effectiveness of CWI in facilitating recovery. Specifically, the effects of

Table 1 Indirect markers of muscle damage following single-joint eccentrically biased exercise

Variables measured	Variables response[a]			Cooling protocol	Measurement time points
	Increase	Decrease	No change		
CK/Mb		Eston and Peters [122] (48–72 h)	Goodall and Howatson [117]	Eston and Peters [122]—15 min @15 °C arm cooling (repeated 12 hourly for 7 times)	Eston and Peters [122]—0 to 72 h @ 24 h intervals
		Vaile et al. [22] (24 and 72 h)	Jakeman et al. [118]	Goodall and Howatson [117]—(12 min @ 15 °C (repeated 24 hourly for 3 days)	Goodall and Howatson [117]—0 to 96 h @ 24 h intervals
			Pointon et al. [28]	Jakeman et al. [118]—10 min @ 10 °C	Jakeman et al. [118]—0 h, 1 h, 24 to 96 h @ 24 h intervals
			Sellwood et al. [119]	Kuligowski et al. [121]—24 min @ 12.8 °C arm cooling (repeated 24 hourly for 3 days)	Kuligowski et al. [121]—0 to 96 h @ 24 h intervals
MVC	Vaile et al. [22] (48–72 h)		Eston and Peters [122]	Paddon-Jones et al. [120]—5 × 20 min @ 5 °C arm cooling @ 60 min intervals	Paddon-Jones et al. [120]—0 to 144 h @ 24 h intervals
			Goodall and Howatson [117]	Pointon et al. [28]—20 min leg cooling by cooling sleeve	Pointon et al. [28]—0 h, 2 h, 24 to 48 h @ 24 h intervals
			Jakeman et al. [118]	Sellwood et al. [119]—3 × 1 min @ 5 °C	Sellwood et al. [119]—0 to 72 h @ 24 h intervals
			Kuligowski et al. [121]	Vaile et al. [22]—14 min @ 15 °C	Vaile et al. [22]—0 to 72 h @ 24 h intervals
			Paddon-Jones et al. [120]		
			Pointon et al. [28]		
			Sellwood et al. [119]		
DOMS		Kuligowski et al. [121] (NS)	Eston and Peters [122]		
			Goodall and Howatson [117]		
			Jakeman et al. [118]		
			Paddon-Jones et al. [120]		
			Pointon et al. [28]		
			Vaile et al. [22]		
Pain	Sellwood et al. [119] (24–48 h)	Pointon et al. [28] (48 h)			
Girth/volume		Vaile et al. [22] (24–72 h)	Eston and Peters [122]		
			Goodall and Howatson [117]		
			Paddon-Jones et al. [120]		
			Sellwood et al. [119]		
ROM	Eston and Peters [122] (48–72 h)		Goodall and Howatson [117]		
	Kuligowski et al. [121] (NS)				
Power	Vaile et al. [22] (48–72 h)		Sellwood et al. [119]		

CK creatine kinase, *Mb* myoglobin, *MVC* maximal voluntary contraction, *DOMS* delayed onset muscle soreness, *ROM* range of motion, *NS* timing not specified

[a] Time points in parentheses indicate periods where significant changes (i.e. increase/decrease) were observed

Table 2 Indirect markers of muscle damage following whole body endurance based intermittent/continuous exercise

Variables measured	Variables response[a]			Cooling protocol	Measurement time points
	Increase	Decrease	No change		
CK/Mb		Ascensao et al. [116] (30 min, 24-48 h) Bailey et al. [125] (1 h) Minett et al. [24] (24 h) Pournot et al. [126] (24 h)	Corbett et al. [128] Ingram et al. [23] Pointon et al. [47]	Ascensao et al. [116]—10 min @10 °C Brophy-Williams et al. [124]—15 min @15 °C Bailey et al. [125]—10 min @ 10 °C Corbett et al. [128]—12 min @ 12 °C	Ascensao et al. [116]—0 h, 30 min, 24 to 48 h @ 24 h intervals Brophy-Williams et al. [124]—0 h, 24 h Bailey et al. [125]—0 h, 1 h, 24 to 48 h @ 24 h intervals, 168 h Corbett et al. [128]—0 h, 1 h, 24 to 48 h @ 24 h intervals
Inflammation (CRP)		Ascensao et al. [116] (30 min–24 h) Brophy-Williams et al. [124] (24 h)	Ingram et al. [23] Minett et al. [24]	Ingram et al. [23]—2 × 5 min @ 10 °C King et al. [127]—2 × 5 min @ 10 °C	Ingram et al. [23]—0 to 48 h @ 24 h intervals King et al. [127]—0 h, 24 h
Immune cells		Pournot et al. [126] (1 h)		Minett et al. [24]—20 min@10 °C	Minett et al. [24]—0 h, 1 h, 24 h Pointon et al. [47]—0 h, 2 h, 24 h
MVC	Ascensao et al. [116] (24 h) Bailey et al. [125] (24–48 h) Minett et al. [24] (0–24 h) Pointon et al. [47] (0 and 24 h) Pournot et al. [126] (1–24 h)		Corbett et al. [128] Ingram et al. [23]	Pointon et al. [47]—18 min @ 9 °C Pournot et al. [126]—15 min @ 10 °C Rowsell et al. [123]—5 × 1 min @ 10 °C	Pournot et al. [126]—0 h, 1 h, 24 h Rowsell et al. [123]—0 to 72 h @ 24 h intervals
DOMS		Ascensao et al. [116] (24 h) Bailey et al. [125] (1–48 h) Ingram et al. [23] (24–48 h) King et al. [127] (24 h) Minett et al. [24] (0–24 h) Pointon et al. [47] (0 h) Rowsell et al. [123] (24 h)	Brophy-Williams et al. [124] Corbett et al. [128] Pournot et al. [126]		
Power	King et al. [127] (24 h) Pournot et al. [126] (1–24 h)		Ascensao et al. [116] Bailey et al. [125]		
Performance	Brophy-Williams et al. [124] (24 h) Ingram et al. [23] (48 h) Rowsell et al. [123] (24 h)		King et al. [127] Pointon et al. [47]		

CK creatine kinase, *Mb* myoglobin, *CRP* C-reactive protein, *MVC* maximal voluntary contraction, *DOMS* delayed onset muscle soreness

[a] Time points in parentheses indicate periods where significant changes (i.e. increase/decrease) were observed

CWI on EIMD have been mainly investigated using: (1) single joint eccentrically biased exercises (Table 1), or (2) whole body exercise modalities that include intermittent (typifying team sport movement patterns) or continuous exercises (representing cyclic endurance sports) (Table 2). As presented in Table 1, CWI does not generally ameliorate the key indices of EIMD such as the leakage of muscle proteins in the bloodstream [28, 117–119], restorations in MVC force [28, 117–122] or decrements in DOMS [22, 28, 117, 118, 120] and swelling [117, 119, 120, 122]. These data indicate that CWI is ineffective in reducing post-exercise muscle damage, at least when EIMD is sustained via single-joint eccentrically biased exercise modalities. On the other hand, CWI seems more effective in treating EIMD incurred with intermittent, endurance based team sport exercises (Table 2). Indeed, the majority of studies to date have shown CWI to be effective in enhancing the recovery of exercise performance [23, 123, 124], MVC force [24, 47, 116, 125, 126], clearance of blood myoproteins such as creatine kinase or myoglobin [24, 116, 124–126], attenuating inflammation [116, 124, 126] or reducing the sensation of DOMS [24, 47, 116, 123, 125, 127]. The only study not to report a benefit of CWI treatment in any of the markers of EIMD including blood creatine kinase content, MVC and DOMS was that of Corbett et al. [128]. Nevertheless, it is important to note that MVC, which closely reflects the extent of myofibre damage and is regarded the gold standard in the assessment of EIMD [129], was improved in the majority of the studies following CWI (Table 2). However, it is also worthwhile to note that although MVC may be the gold standard in assessing myofibre damage and recovery from EIMD, it may not be reflective of power production and athletic performance. Indeed, Ingram et al. [23] showed an improvement in repeated sprint performance following CWI treatment, despite no recovery in MVC.

Recently, it has been suggested that CWI mediated MVC recovery following whole body muscle damaging exercise may not exclusively reflect recovery from EIMD, but might also include recovery from central fatigue [24, 47]. Indeed, Pointon et al. [47] and Minett et al. [24] demonstrated improved MVC recovery from exhaustive intermittent running in the heat when CWI was administered. However the improvements in MVC were concomitant with the amelioration of indices relating to both CNS fatigue (i.e. reduced T_c and improved VA) and EIMD (i.e. reduced DOMS sensation and enhanced clearance of circulating myoproteins) [24, 47]. While these data highlight the difficulties in isolating the mechanisms by which CWI facilitates recovery, it is evident that multiple mechanisms are involved in CWI mediated recovery from whole body exhaustive exercise.

4 Conclusions

In summary, a number of mechanisms have been suggested to be responsible for the enhanced acute and longer term recovery associated with post-exercise CWI (Fig. 6). Under heat stress, CWI facilitates short term recovery by

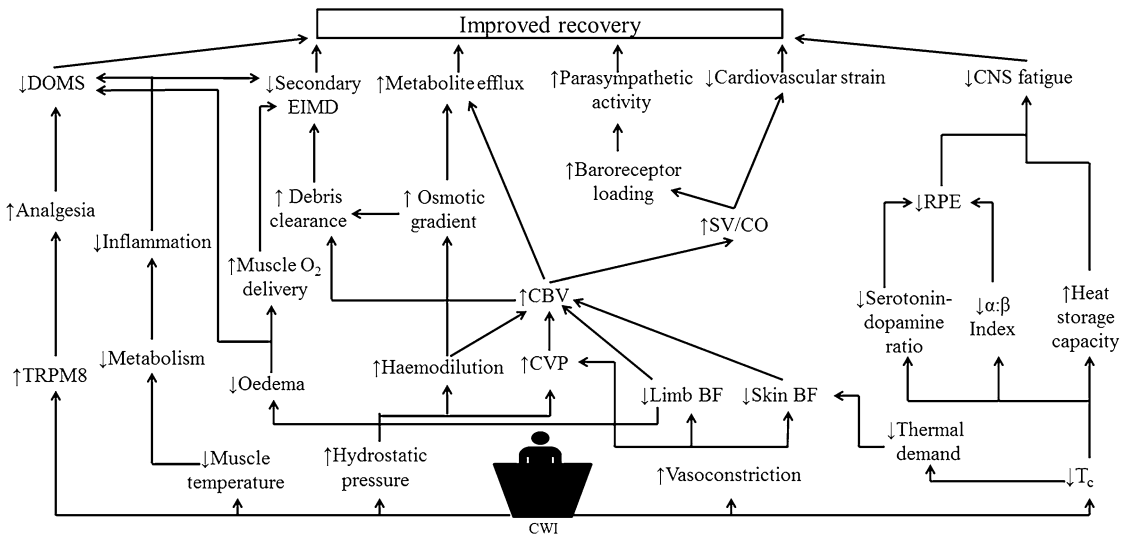

Fig. 6 Integrated mechanisms by which CWI enhances recovery. CWI enhances recovery and improves athlete preparedness through multiple mechanisms namely; decreasing secondary EIMD, decreasing the sensation of DOMS, improving the clearance of muscle metabolites, increasing post-exercise parasympathetic activity, decreasing cardiovascular strain and decreasing CNS fatigue. *EIMD* exercise-induced muscle damage, *DOMS* delayed onset muscle soreness, *TRPM8* transient receptor potential cation channel M8, *CWI* cold water immersion, T_c core temperature, BF blood flow, CVP central venous pressure, CBV central blood volume, *SV/CO* stroke volume/cardiac output, *CNS* central nervous system, *RPE* ratings of perceived exertion, ↑ increase, ↓ decrease

rapidly reducing body temperatures, consequently ameliorating CNS mediated fatigue, and by reducing cardiovascular strain. To date, there is only marginal evidence supporting the notion that CWI might improve acute recovery by facilitating the removal of muscle metabolites. Moreover, parasympathetic reactivation following CWI seems detrimental to high-intensity performances performed shortly after, but seems beneficial with regards to longer term physiological recovery and day to day training performances. The efficacy of CWI for attenuating the secondary effects of EIMD seems dependent on the mode of exercise utilised with CWI having limited influence on EIMD induced by single joint eccentrically biased contractions. In contrast, CWI seems more effective in ameliorating effects of EIMD induced by whole body prolonged endurance/intermittent based exercise modalities. Understanding these mechanisms will aid practitioners in the application and optimisation of CWI strategies to suit specific recovery needs, improve athletic performance and enhance adaptations to exercise.

Compliance with Ethical Standards

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References

1. Saltin B, Blomqvist G, Mitchell JH, et al. Response to exercise after bed rest and after training. Circulation. 1968;38(5 Suppl):VII1–78.
2. Saltin B, Rowell LB. Functional adaptations to physical activity and inactivity. Fed Proc. 1980;39(5):1506–13.
3. Ehsani AA, Hagberg JM, Hickson RC. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. Am J Cardiol. 1978;42(1):52–6.
4. Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training. Annu Rev Physiol. 1983;45:169–89.
5. Convertino VA. Blood volume: its adaptation to endurance training. Med Sci Sports Exerc. 1991;23(12):1338–48.
6. Weight LM, Alexander D, Elliot T, et al. Erythropoietic adaptations to endurance training. Eur J Appl Physiol Occup Physiol. 1992;64(5):444–8.
7. Mier CM, Turner MJ, Ehsani AA, et al. Cardiovascular adaptations to 10 days of cycle exercise. J Appl Physiol (1985). 1997;83(6):1900–6.
8. Hoppeler H, Luthi P, Claassen H, et al. The ultrastructure of the normal human skeletal muscle. A morphometric analysis on untrained men, women and well-trained orienteers. Pflugers Arch. 1973;344(3):217–32.
9. Gollnick PD, Saltin B. Significance of skeletal muscle oxidative enzyme enhancement with endurance training. Clin Physiol. 1982;2(1):1–12.
10. Daussin FN, Zoll J, Dufour SP, et al. Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary subjects. Am J Physiol Regul Integr Comp Physiol. 2008;295(1):R264–72.
11. Inbar O, Kaiser P, Tesch P. Relationships between leg muscle fiber type distribution and leg exercise performance. Int J Sports Med. 1981;2(3):154–9.
12. Lash JM, Bohlen HG. Functional adaptations of rat skeletal muscle arterioles to aerobic exercise training. J Appl Physiol (1985). 1992;72(6):2052–62.
13. Rakobowchuk M, Tanguay S, Burgomaster KA, et al. Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. Am J Physiol Regul Integr Comp Physiol. 2008;295(1):R236–42.
14. Hickson RC, Hagberg JM, Ehsani AA, et al. Time course of the adaptive responses of aerobic power and heart rate to training. Med Sci Sports Exerc. 1981;13(1):17–20.
15. Billat V, Lepretre PM, Heugas AM, et al. Training and bioenergetic characteristics in elite male and female Kenyan runners. Med Sci Sports Exerc. 2003;35(2):297–304.
16. Billat VL, Demarle A, Slawinski J, et al. Physical and training characteristics of top-class marathon runners. Med Sci Sports Exerc. 2001;33(12):2089–97.
17. Reilly T, Ekblom B. The use of recovery methods post-exercise. J Sports Sci. 2005;23(6):619–27.
18. Barnett A. Using recovery modalities between training sessions in elite athletes: does it help? Sports Med. 2006;36(9):781–96.
19. Budgett R. Fatigue and underperformance in athletes: the overtraining syndrome. Br J Sports Med. 1998;32(2):107–10.
20. Peiffer JJ, Abbiss CR, Watson G, et al. Effect of a 5-min cold-water immersion recovery on exercise performance in the heat. Br J Sports Med. 2010;44(6):461–5.
21. Peiffer JJ, Abbiss CR, Watson G, et al. Effect of cold-water immersion duration on body temperature and muscle function. J Sports Sci. 2009;27(10):987–93.
22. Vaile J, Halson S, Gill N, et al. Effect of hydrotherapy on the signs and symptoms of delayed onset muscle soreness. Eur J Appl Physiol. 2008;102(4):447–55.
23. Ingram J, Dawson B, Goodman C, et al. Effect of water immersion methods on post-exercise recovery from simulated team sport exercise. J Sci Med Sport. 2009;12(3):417–21.
24. Minett GM, Duffield R, Billaut F, et al. Cold-water immersion decreases cerebral oxygenation but improves recovery after intermittent-sprint exercise in the heat. Scand J Med Sci Sports. 2014;24(4):656–66.
25. Dunne A, Crampton D, Egana M. Effect of post-exercise hydrotherapy water temperature on subsequent exhaustive running performance in normothermic conditions. J Sci Med Sport. 2013;16(5):466–71.
26. Vaile J, Halson S, Gill N, et al. Effect of hydrotherapy on recovery from fatigue. Int J Sports Med. 2008;29(7):539–44.
27. Stanley J, Peake JM, Buchheit M. Consecutive days of cold water immersion: effects on cycling performance and heart rate variability. Eur J Appl Physiol. 2013;113(2):371–84.
28. Pointon M, Duffield R, Cannon J, et al. Cold application for neuromuscular recovery following intense lower-body exercise. Eur J Appl Physiol. 2011;111(12):2977–86.
29. Poppendieck W, Faude O, Wegmann M, et al. Cooling and performance recovery of trained athletes: a meta-analytical review. Int J Sports Physiol Perform. 2013;8(3):227–42.
30. Versey NG, Halson SL, Dawson BT. Water immersion recovery for athletes: effect on exercise performance and practical recommendations. Sports Med. 2013;43(11):1101–30.

31. Leeder J, Gissane C, van Someren K, et al. Cold water immersion and recovery from strenuous exercise: a meta-analysis. Br J Sports Med. 2011;46(4):233–40.
32. Halson SL. Does the time frame between exercise influence the effectiveness of hydrotherapy for recovery? Int J Sports Physiol Perform. 2011;6(2):147–59.
33. Bleakley C, McDonough S, Gardner E, et al. Cold-water immersion for preventing and treating muscle soreness after exercise. Cochrane Database Syst Rev. 2012;2:CD008262.
34. Ihsan M, Markworth JF, Watson G, et al. Regular post-exercise cooling enhances mitochondrial biogenesis through AMPK and p38 MAPK in human skeletal muscle. Am J Physiol Regul Integr Comp Physiol. 2015;309(3):R286–94.
35. Ihsan M, Watson G, Choo HC, et al. Postexercise muscle cooling enhances gene expression of PGC-1alpha. Med Sci Sports Exerc. 2014;46(10):1900–7.
36. Ihsan M, Watson G, Abbiss C. PGC-1α mediated muscle aerobic adaptations to exercise, heat and cold exposure. Cell Mol Exerc Physiol. 2014;3(1):e7.
37. Frohlich M, Faude O, Klein M, et al. Strength training adaptations after cold water immersion. J Strength Cond Res. 2014;28(9):2628–33.
38. 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):4285–301.
39. Taylor JL, Todd G, Gandevia SC. Evidence for a supraspinal contribution to human muscle fatigue. Clin Exp Pharmacol Physiol. 2006;33(4):400–5.
40. Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. J Appl Physiol (1985). 2001;91(3):1055–60.
41. Morrison S, Sleivert G, Cheung S. Passive hyperthermia reduces voluntary activation and isometric force production. Eur J Appl Physiol. 2004;91(5):729–36.
42. Cheung SS, Sleivert GG. Multiple triggers for hyperthermic fatigue and exhaustion. Exerc Sport Sci Rev. 2004;32(3):100–6.
43. Nybo L. Brain temperature and exercise performance. Exp Physiol. 2012;97(3):333–9.
44. Wakabayashi H, Kaneda K, Sato D, et al. Effect of non-uniform skin temperature on thermoregulatory response during water immersion. Eur J Appl Physiol. 2008;104(2):175–81.
45. Peiffer JJ, Abbiss CR, Nosaka K, et al. Effect of cold water immersion after exercise in the heat on muscle function, body temperatures, and vessel diameter. J Sci Med Sport. 2009;12(1):91–6.
46. Yeargin SW, Casa DJ, McClung JM, et al. Body cooling between two bouts of exercise in the heat enhances subsequent performance. J Strength Cond Res. 2006;20(2):383–9.
47. Pointon M, Duffield R, Cannon J, et al. Cold water immersion recovery following intermittent-sprint exercise in the heat. Eur J Appl Physiol. 2012;112(7):2483–94.
48. Nielsen B, Hyldig T, Bidstrup F, et al. Brain activity and fatigue during prolonged exercise in the heat. Pflugers Arch. 2001;442(1):41–8.
49. Nybo L, Nielsen B. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. J Appl Physiol (1985). 2001;91(5):2017–23.
50. Meeusen R, Watson P, Hasegawa H, et al. Central fatigue: the serotonin hypothesis and beyond. Sports Med. 2006;36(10):881–909.
51. Davis JM, Bailey SP. Possible mechanisms of central nervous system fatigue during exercise. Med Sci Sports Exerc. 1997;29(1):45–57.
52. Nybo L, Rasmussen P. Inadequate cerebral oxygen delivery and central fatigue during strenuous exercise. Exerc Sport Sci Rev. 2007;35(3):110–8.
53. Nybo L, Moller K, Pedersen BK, et al. Association between fatigue and failure to preserve cerebral energy turnover during prolonged exercise. Acta Physiol Scand. 2003;179(1):67–74.
54. Vaile J, Halson S, Gill N, et al. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. J Sports Sci. 2008;26(5):431–40.
55. De Pauw K, Roelands B, Marušič U, et al. Brain mapping after prolonged cycling and during recovery in the heat. J Appl Physiol (1985). 2013;115(9):1324–31.
56. Stocks JM, Patterson MJ, Hyde DE, et al. Effects of immersion water temperature on whole-body fluid distribution in humans. Acta Physiol Scand. 2004;182(1):3–10.
57. Newsholme E, Acworth I, Blomstrand E. Amino acids, brain neurotransmitters and a functional link between muscle and brain that is important in sustained exercise. Adv Myochem. 1987;1987:127–38.
58. Roelands B, Hasegawa H, Watson P, et al. The effects of acute dopamine reuptake inhibition on performance. Med Sci Sports Exerc. 2008;40(5):879–85.
59. Bailey SP, Davis JM, Ahlborn EN. Serotonergic agonists and antagonists affect endurance performance in the rat. Int J Sports Med. 1993;14(6):330–3.
60. Roelands B, Goekint M, Buyse L, et al. Time trial performance in normal and high ambient temperature: is there a role for 5-HT? Eur J Appl Physiol. 2009;107(1):119–26.
61. Mundel T, Bunn SJ, Hooper PL, et al. The effects of face cooling during hyperthermic exercise in man: evidence for an integrated thermal, neuroendocrine and behavioural response. Exp Physiol. 2007;92(1):187–95.
62. Mundel T, Hooper PL, Bunn SJ, et al. The effects of face cooling on the prolactin response and subjective comfort during moderate passive heating in humans. Exp Physiol. 2006;91(6):1007–14.
63. Rowell LB. Human cardiovascular adjustments to exercise and thermal stress. Physiol Rev. 1974;54(1):75–159.
64. González-Alonso J, Calbet JAL. Reductions in systemic and skeletal muscle blood flow and oxygen delivery limit maximal aerobic capacity in humans. Circulation. 2003;107(6):824–30.
65. Périard JD, Cramer MN, Chapman PG, et al. Cardiovascular strain impairs prolonged self-paced exercise in the heat. Exp Physiol. 2011;96(2):134–44.
66. Hayashi K, Honda Y, Ogawa T, et al. Effects of brief leg cooling after moderate exercise on cardiorespiratory responses to subsequent exercise in the heat. Eur J Appl Physiol. 2004;92(4–5):414–20.
67. Vaile J, O'Hagan C, Stefanovic B, et al. Effect of cold water immersion on repeated cycling performance and limb blood flow. Br J Sports Med. 2010;45(10):825–9.
68. Mawhinney C, Jones H, Joo CH, et al. Influence of cold-water immersion on limb and cutaneous blood flow after exercise. Med Sci Sports Exerc. 2013;45(12):2277–85.
69. Ihsan M, Watson G, Lipski M, et al. Influence of postexercise cooling on muscle oxygenation and blood volume changes. Med Sci Sports Exerc. 2013;45(5):876–82.
70. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. Physiol Rev. 2008;88(1):287–332.
71. Abbiss CR, Laursen PB. Models to explain fatigue during prolonged endurance cycling. Sports Med. 2005;35(10):865–98.
72. Halson SL, Quod MJ, Martin DT, et al. Physiological responses to cold water immersion following cycling in the heat. Int J Sports Physiol Perform. 2008;3(3):331–46.
73. Hausswirth C, Duffield R, Pournot H, et al. Postexercise cooling interventions and the effects on exercise-induced heat stress in a temperate environment. Appl Physiol Nutr Metab. 2012;37(5):965–75.

74. Johansen LB, Bie P, Warberg J, et al. Role of hemodilution on renal responses to water immersion in humans. Am J Physiol. 1995;269(5 Pt 2):R1068–76.
75. Johansen LB, Jensen TUS, Pump B, et al. Contribution of abdomen and legs to central blood volume expansion in humans during immersion. J Appl Physiol (1985). 1997;83(3):695–9.
76. Park KS, Choi JK, Park YS. Cardiovascular regulation during water immersion. Appl Hum Sci. 1999;18(6):233–41.
77. Gabrielsen A, Johansen LB, Norsk P. Central cardiovascular pressures during graded water immersion in humans. J Appl Physiol (1985). 1993;75(2):581–5.
78. Parouty J, Al Haddad H, Quod M, et al. Effect of cold water immersion on 100-m sprint performance in well-trained swimmers. Eur J Appl Physiol. 2010;109(3):483–90.
79. Crowe MJ, O'Connor D, Rudd D. Cold water recovery reduces anaerobic performance. Int J Sports Med. 2007;28(12):994–8.
80. Bergh U, Ekblom B. Influence of muscle temperature on maximal muscle strength and power output in human skeletal muscles. Acta Physiol Scand. 1979;107(1):33–7.
81. Stanley J, Peake JM, Buchheit M. Cardiac parasympathetic reactivation following exercise: implications for training prescription. Sports Med. 2013;43(12):1259–77.
82. Hautala AJ, Kiviniemi AM, Tulppo MP. Individual responses to aerobic exercise: the role of the autonomic nervous system. Neurosci Biobehav Rev. 2009;33(2):107–15.
83. Perini R, Orizio C, Comande A, et al. Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. Eur J Appl Physiol Occup Physiol. 1989;58(8):879–83.
84. Buchheit M, Al Haddad H, Mendez-Villanueva A, et al. Effect of maturation on hemodynamic and autonomic control recovery following maximal running exercise in highly trained young soccer players. Front Physiol. 2011;2:69.
85. Buchheit M, Duche P, Laursen PB, et al. Postexercise heart rate recovery in children: relationship with power output, blood pH, and lactate. Appl Physiol Nutr Metab. 2010;35(2):142–50.
86. Ba A, Delliaux S, Bregeon F, et al. Post-exercise heart rate recovery in healthy, obeses, and COPD subjects: relationships with blood lactic acid and PaO2 levels. Clin Res Cardiol. 2009;98(1):52–8.
87. Mourot L, Bouhaddi M, Gandelin E, et al. Cardiovascular autonomic control during short-term thermoneutral and cool head-out immersion. Aviat Space Environ Med. 2008;79(1):14–20.
88. Pump B, Shiraishi M, Gabrielsen A, et al. Cardiovascular effects of static carotid baroreceptor stimulation during water immersion in humans. Am J Physiol Heart Circ Physiol. 2001;280(6):H2607–15.
89. Buchheit M, Peiffer JJ, Abbiss CR, et al. Effect of cold water immersion on postexercise parasympathetic reactivation. Am J Physiol Heart Circ Physiol. 2009;296(2):H421–7.
90. Stanley J, Buchheit M, Peake JM. The effect of post-exercise hydrotherapy on subsequent exercise performance and heart rate variability. Eur J Appl Physiol. 2012;112(3):951–61.
91. Al Haddad H, Parouty J, Buchheit M. Effect of daily cold water immersion on heart rate variability and subjective ratings of well-being in highly trained swimmers. Int J Sports Physiol Perform. 2012;7(1):33–8.
92. Bastos FN, Vanderlei LC, Nakamura FY, et al. Effects of cold water immersion and active recovery on post-exercise heart rate variability. Int J Sports Med. 2012;33(11):873–9.
93. Richter EA, Ruderman NB, Galbo H. Alpha and beta adrenergic effects on metabolism in contracting, perfused muscle. Acta Physiol Scand. 1982;116(3):215–22.
94. Costill DL, Hargreaves M. Carbohydrate nutrition and fatigue. Sports Med. 1992;13(2):86–92.
95. Rauch HG, St Clair Gibson A, Lambert EV, et al. A signalling role for muscle glycogen in the regulation of pace during prolonged exercise. Br J Sports Med. 2005;39(1):34–8.
96. Rauch LH, Rodger I, Wilson GR, et al. The effects of carbohydrate loading on muscle glycogen content and cycling performance. Int J Sport Nutr. 1995;5(1):25–36.
97. Beelen M, Burke LM, Gibala MJ, et al. Nutritional strategies to promote postexercise recovery. Int J Sport Nutr Exerc Metab. 2010;20(6):515–32.
98. Gregson W, Allan R, Holden S, et al. Postexercise cold-water immersion does not attenuate muscle glycogen resynthesis. Med Sci Sports Exerc. 2013;45(6):1174–81.
99. Slivka D, Heesch M, Dumke C, et al. Effects of post-exercise recovery in a cold environment on muscle glycogen, PGC-1alpha, and downstream transcription factors. Cryobiology. 2013;66(3):250–5.
100. Slivka DR, Dumke CL, Tucker TJ, et al. Human mRNA response to exercise and temperature. Int J Sports Med. 2012;33(2):94–100.
101. Tucker TJ, Slivka DR, Cuddy JS, et al. Effect of local cold application on glycogen recovery. J Sports Med Phys Fitness. 2012;52(2):158–64.
102. Kennedy JW, Hirshman MF, Gervino EV, et al. Acute exercise induces GLUT4 translocation in skeletal muscle of normal human subjects and subjects with type 2 diabetes. Diabetes. 1999;48(5):1192–7.
103. Swenson C, Sward L, Karlsson J. Cryotherapy in sports medicine. Scand J Med Sci Sports. 1996;6(4):193–200.
104. Wilcock IM, Cronin JB, Hing WA. Physiological response to water immersion: a method for sport recovery? Sports Med. 2006;36(9):747–65.
105. Yanagisawa O, Kudo H, Takahashi N, et al. Magnetic resonance imaging evaluation of cooling on blood flow and oedema in skeletal muscles after exercise. Eur J Appl Physiol. 2004;91(5):737–40.
106. Crenshaw AG, Karlsson S, Gerdle B, et al. Differential responses in intramuscular pressure and EMG fatigue indicators during low- vs. high-level isometric contractions to fatigue. Acta Physiol Scand. 1997;160(4):353–61.
107. Gregson W, Black MA, Jones H, et al. Influence of cold water immersion on limb and cutaneous blood flow at rest. Am J Sports Med. 2011;39(6):1316–23.
108. Merrick MA, Rankin JM, Andres FA, et al. A preliminary examination of cryotherapy and secondary injury in skeletal muscle. Med Sci Sports Exerc. 1999;31(11):1516.
109. Carvalho N, Puntel G, Correa P, et al. Protective effects of therapeutic cold and heat against the oxidative damage induced by a muscle strain injury in rats. J Sports Sci. 2010;28(9):923–35.
110. Cheung K, Hume PA, Maxwell L. Delayed onset muscle soreness. Sports Med. 2003;33(2):145–64.
111. Ebbeling CB, Clarkson PM. Exercise-induced muscle damage and adaptation. Sports Med. 1989;7(4):207–34.
112. Proudfoot CJ, Garry EM, Cottrell DF, et al. Analgesia mediated by the TRPM8 cold receptor in chronic neuropathic pain. Curr Biol. 2006;16(16):1591–605.
113. Knowlton WM, Palkar R, Lippoldt EK, et al. A sensory-labeled line for cold: TRPM8-expressing sensory neurons define the cellular basis for cold, cold pain, and cooling-mediated analgesia. J Neurosci. 2013;33(7):2837–48.
114. Graven-Nielsen T, Lund H, Arendt-Nielsen L, et al. Inhibition of maximal voluntary contraction force by experimental muscle pain: a centrally mediated mechanism. Muscle Nerve. 2002;26(5):708–12.
115. 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.

116. Ascensao A, Leite M, Rebelo AN, et al. Effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. J Sports Sci. 2011;29(3):217–25.
117. Goodall S, Howatson G. The effects of multiple cold water immersions on indices of muscle damage. J Sports Sci Med. 2008;7(2):235–41.
118. Jakeman JR, Macrae R, Eston R. A single 10-min bout of cold-water immersion therapy after strenuous plyometric exercise has no beneficial effect on recovery from the symptoms of exercise-induced muscle damage. Ergonomics. 2009;52(4):456–60.
119. Sellwood KL, Brukner P, Williams D, et al. Ice-water immersion and delayed-onset muscle soreness: a randomised controlled trial. Br J Sports Med. 2007;41(6):392–7.
120. Paddon-Jones DJ, Quigley BM. Effect of cryotherapy on muscle soreness and strength following eccentric exercise. Int J Sports Med. 1997;18(8):588–93.
121. Kuligowski LA, Lephart SM, Giannantonio FP, et al. Effect of whirlpool therapy on the signs and symptoms of delayed-onset muscle soreness. J Athl Train. 1998;33(3):222–8.
122. Eston R, Peters D. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. J Sports Sci. 1999;17(3):231–8.
123. Rowsell GJ, Coutts AJ, Reaburn P, et al. Effect of post-match cold-water immersion on subsequent match running performance in junior soccer players during tournament play. J Sports Sci. 2011;29(1):1–6.
124. Brophy-Williams N, Landers G, Wallman K. Effect of immediate and delayed cold water immersion after a high intensity exercise session on subsequent run performance. J Sports Sci Med. 2011;10:665–70.
125. Bailey DM, Erith SJ, Griffin PJ, et al. Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. J Sports Sci. 2007;25(11):1163–70.
126. Pournot H, Bieuzen F, Duffield R, et al. Short term effects of various water immersions on recovery from exhaustive intermittent exercise. Eur J Appl Physiol. 2011;111(7):1287–95.
127. King M, Duffield R. The effects of recovery interventions on consecutive days of intermittent sprint exercise. J Strength Cond Res. 2009;23(6):1795–802.
128. Corbett J, Barwood MJ, Lunt HC, et al. Water immersion as a recovery aid from intermittent shuttle running exercise. Eur J Sport Sci. 2012;12(6):509–14.
129. Warren GL, Lowe DA, Armstrong RB. Measurement tools used in the study of eccentric contraction-induced injury. Sports Med. 1999;27(1):43–59.