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The Recovery-Adaptation Paradox: A Review Comparing Cold Water Immersion and Hot Water Immersion on Immediate Recovery vs. Long-Term Muscular Adaptations

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ABSTRACT

Background. Cold water immersion (CWI) has long been the gold standard for post-exercise recovery, primarily due to its potent analgesic effects and perceived reduction in muscle soreness. However, recent molecular and longitudinal evidence suggests that CWI may fundamentally interfere with the adaptive processes following resistance exercise. In contrast, hot water immersion (HWI) is emerging as an alternative that may support recovery without compromising hypertrophy or explosive power.

Objectives. This review synthesizes current evidence regarding the molecular, vascular, and functional responses to thermal interventions, specifically contrasting the "regeneration paradox" of CWI with the facilitative mechanisms of HWI. The objective is to determine which immersion strategy optimizes the balance between acute recovery, characterized by the alleviation of muscle soreness (DOMS) and restoration of performance, and long-term hypertrophic gains. By synthesizing current evidence, this work intends to provide a clearer framework for athletes and recreational trainees in choosing recovery modalities that do not compromise chronic training outcomes.

Methods. A synthesis of recent meta-analyses, systematic reviews and experimental trials was conducted, focusing on myogenic signaling, ribosome biogenesis, vascular kinetics, and long-term performance outcomes.

Findings. CWI effectively alleviates DOMS via reduced nerve conduction and vasoconstriction. However, this hemodynamic response impairs nutrient delivery and reduces muscle protein synthesis by approximately 20%. Molecularly, CWI suppresses mTORC1 signaling, satellite cell activity, and ribosome biogenesis, leading to attenuated hypertrophy and reduced rate of force development (RFD). Conversely, HWI enhances microvascular perfusion and angiogenesis without suppressing anabolic signaling. By inducing heat shock proteins (HSP70), HWI protects cellular proteostasis and accelerates the recovery of explosive strength, effectively bridging the gap between acute recovery and long-term adaptation where CWI fails.
Conclusion. This review addresses the "Recovery-Adaptation Paradox" by highlighting that the optimal immersion strategy is strictly objective-dependent. While CWI effectively optimizes acute recovery through analgesia and masking fatigue, it may compromise long-term hypertrophic gains and explosive power by disrupting anabolic signaling. In contrast HWI provides a strategic alternative for athletes prioritizing chronic training outcomes, as it facilitates restoration without interfering with the muscle's endogenous adaptive pathways.
Keywords: cold water immersion (CWI), hot water immersion (HWI), hypertrophy, mTORC1, muscle protein synthesis, heat shock proteins, recovery, adaptation, EIMD, DOMS

INTRODUCTION

To achieve superior outcomes in athletic performance, the standards for effective muscle adaptation and regeneration have evolved. For decades, cold water immersion (CWI) has been a widely adopted recovery modality, effectively reducing edema and acute muscle inflammation – and consequently pain, swelling and loss of force production – while attenuating markers of muscle damage, such as creatine kinase, in both elite and recreational settings (Bleakley et al., 2012; Pournot et al., 2011; Wilcock et al., 2006). Immersive cryotherapy has also been praised for its role in mitigating exercise-induced muscle damage (EIMD) and alleviating delayed-onset muscle soreness (DOMS), based on the assumption that an accelerated recovery directly translates to enhanced long-term performance (Bleakley et al., 2012; Machado et al., 2016; Wang et al., 2025). However, research by Broatch et al. (2014)

suggests that these perceived benefits might be partially attributed to a placebo effect, challenging the assumption that cold-induced physiological changes are the sole drivers of improved recovery and subsequent performance.

As highlighted by Halson (2013), efficient regeneration is invaluable for maintaining high training loads while minimizing the risk of overtraining and injury, particularly among elite athletes. While the efficacy of CWI on recovery of exercise performance is well-documented, comprehensive reviews (Versey et al., 2013) emphasize that its superiority over modalities like contrast water therapy (CWT) or passive recovery following high-intensity training is highly dependent on specific protocols to optimize these outcomes. The therapeutic success of these protocols is primarily driven by cold-induced sympathetic activation, which triggers localized vasoconstriction and a subsequent decrease in metabolic activity and decreased cardiovascular strain (Ihsan et al., 2016; Wilcock et al., 2006). By inducing the narrowing of blood vessels, low temperatures limit the infiltration of inflammatory cells into damaged areas, thereby modulating the initial inflammatory cascade and mitigating secondary tissue damage (Ihsan et al., 2016; Pournot et al., 2011; Wang et al., 2025). This process, combined with the analgesic effect of cold on nerve conduction velocity, significantly elevates the pain threshold (Versey et al., 2013). Furthermore, the benefits of water immersion therapy are not confined to thermal effects. The role of hydrostatic pressure – promoting fluid translocation from interstitial spaces into the vascular system – enhances venous return and reduces post-exercise edema (Ihsan et al., 2016; Wilcock et al., 2006).

While regeneration focuses on the restoration of homeostasis, adaptation involves structural and functional remodeling, such as muscle hypertrophy and increased force-generating capacity, triggered by exercise-induced stress (Schoenfeld, 2010). This adaptive process relies on the very physiological disruptions that recovery modalities often aim to neutralize: specifically, mechanical tension and metabolic stress initiate an anabolic cascade, where transient inflammation and reactive oxygen species (ROS) serve as essential molecular triggers for muscle protein synthesis (Peake et al., 2017). Consequently, the systematic suppression of these pro-adaptive stressors creates a divergence between the efficiency of acute recovery and the extent of chronic structural remodeling. Crucially, the acute inflammatory response, which CWI is traditionally intended to suppress, is now recognized as an essential signaling cue for these adaptations. At the molecular level this is evidenced by the attenuated phosphorylation of key signaling proteins within the mTORC1 pathway (Roberts et al., 2015), the suppression of ribosome biogenesis (Figueiredo et al., 2016), and cold-induced vasoconstriction limiting the

delivery and uptake of amino acids into the muscle tissue, further reducing the rates of muscle protein synthesis (Betz et al., 2025; Fuchs, Kouw, et al., 2020).

Considering these limitations, hot water immersion (HWI) has emerged as a promising alternative. Unlike CWI, heat therapy may promote muscle adaptation by enhancing microvascular blood flow – mediated by vasodilation – and stimulating the expression of heat shock proteins (HSPs) (Dablainville et al., 2025; Iguchi et al., 2012; McGorm et al., 2018). Research in human skeletal muscle has demonstrated that whole body or localized heat stress can increase the phosphorylation of key anabolic signaling proteins, such as Akt/mTOR and p70S6K (Ihsan et al., 2020; Kakigi et al., 2011; McGorm et al., 2018), while elevating cyto-protective HSP levels, which protect against proteotoxic stress, facilitate protein folding and prevent the aggregation of damaged ones during post-recovery (Iguchi et al., 2012; Ihsan et al., 2020). Nevertheless, longitudinal studies have noted that heating may not provide additional hypertrophic benefits beyond those of resistance training alone (Stadnyk et al., 2018). However, as suggested by McGorm et al. (2018) in their comprehensive evaluation, heat-based modalities could serve as a viable alternative to cooling, as they do not appear to induce the same suppressive effects on anabolic signaling that authors previously observed with CWI. This is reinforced by Betz et al. (2025), whose vascular data imply that avoiding the cold-induced perfusion deficit is critical for sustaining long-term hypertrophic gains. Furthermore, recent experimental evidence by Benoît et al. (2024) demonstrated that HWI specifically restores explosive capacity following muscle damage, whereas CWI fails to do so. This shift in focus suggests that heat-based modalities might offer a superior balance between recovery and long-term training adaptations.

This study aims to critically evaluate and compare the impact of cold water immersion (CWI) and hot water immersion (HWI) on post-exercise recovery and skeletal muscle adaptation. Specifically, this review analyses molecular and physiological mechanisms, including the mTORC1 pathway, ribosome biogenesis, microvascular perfusion, and heat shock protein expression. The objective is to determine which immersion strategy optimizes the balance between acute recovery, characterized by the alleviation of muscle soreness (DOMS) and restoration of performance, and long-term hypertrophic gains. By synthesizing current evidence, this work intends to provide a clearer framework for athletes and recreational trainees in choosing recovery modalities that do not compromise chronic training outcomes.

METHODS

To ensure a comprehensive analysis for this review, a systematic search of the literature was conducted across major databases, including PubMed, Scopus, Google Scholar, covering the period from 2006 to 2026. While the primary focus was on studies published between 2015 and 2026 to capture the most recent advancements in physiological and molecular responses to post-exercise water immersion, earlier seminal studies (2006-2014) were also included.

Search Strategy

The search was structured around three core domains: Intervention (e.g., CWI, HWI, hydrotherapy), Mechanism (e.g., mTORC1, muscle protein synthesis, heat shock proteins), and Outcome (e.g., hypertrophy, strength recovery, RFD). Keywords within each domain were combined using the OR operator, while domains were interconnected using the AND operator to refine the search:

- Intervention terms: “cold water immersion”, “CWI”, “hot water immersion”, “HWI”, “thermal therapy”, “hydrotherapy”
- Mechanistic terms: “mTORC1 signaling”, “ribosome biogenesis”, “anabolic signaling”, “satellite cells”, “muscle protein synthesis”, “microvascular perfusion”, “inflammatory response”, “heat shock proteins”.
- Outcome terms: “muscle recovery”, “muscle adaptation”, “muscle hypertrophy”, “strength recovery”, “rate of force development”.

Scope of analysis: studies focusing on acute molecular markers (e.g., mTORC1 signaling, heat shock proteins, myogenic regulatory factors, ribosome biogenesis) vascular dynamics (e.g., microvascular blood volume and long-term structural adaptations (hypertrophy, strength) to ensure a comprehensive understanding of the thermal-adaptation interface.

Inclusion and Exclusion Criteria

Studies were selected based on their relevance to the divergence between acute recovery and chronic muscular adaptations following specific modality of water immersion.

Inclusion criteria were:

- Study design and intervention: original, peer-reviewed experimental studies, systematic reviews and meta-analyses evaluating cold water immersion (CWI) or hot water immersion (HWI) as the primary intervention. Studies utilizing other thermal modalities (e.g., microwave diathermy, heating pads) were used only when they provided mechanistic

insights into mechanistic insights into molecular signaling (e.g., HSP expression, mTORC1 activity).

- Participants: studies conducted on healthy participants, ranging from recreationally active to elite athletes. While search parameters were inclusive of all genders, the final selection primarily reflects male cohorts, as they represent the majority of existing mechanistic research regarding post-exercise molecular signaling and thermal interventions.
- Protocols: involving whole-body or partial-body immersion following resistance exercise or isolated thermal exposures, involving protocols where heat or cold stress was applied independently of mechanical loading to elucidate baseline molecular responses, such as angiogenesis, mitochondrial biogenesis, and the induction of heat shock proteins.

Exclusion criteria included:

- Non-thermal focus: studies focused exclusively on non-thermal aspects of immersion (unless discussing hydrostatic pressure as a secondary mechanism), non-immersion cryotherapy or non-thermal therapy.
- Animal models: studies involving animal models excluded, except where human data on specific molecular pathways were critically limited and required for mechanistic theoretical framework.
- Clinical populations: studies involving clinical populations with chronic diseases or acute musculoskeletal injuries.

DISCUSSION

Thermal Regulation of Recovery: From Psychosensory Perceptions to Molecular Signaling

The widespread adoption of cold water immersion is primarily supported by its efficacy in acute recovery, specifically regarding the mitigation of delayed onset muscle soreness (DOMS) (Bleakley et al., 2012; Wang et al., 2025). As demonstrated by Machado et al. (2016) in their extensive meta-analysis, there is a specific “therapeutic window” for achieving the optimal analgesic effect. The authors established that immersion lasting 10-15 minutes in water of temperatures between 10 and 15°C constitutes the most effective protocol for reducing subjective sensation of pain. The efficacy of this protocol is further corroborated by meta-analytical data from Hohenauer et al. (2015), confirming that CWI significantly reduces the subjective perception of muscle pain and fatigue for up to 24 hours post-exercise compared to passive recovery tactics. This analgesic effect is largely attributed to reduced nerve conduction

and localized vasoconstriction limiting edema (Wilcock et al., 2006). Furthermore, studies utilizing systemic markers have reported that CWI led to a protracted immune response. This is characterized by a delayed rise in pro-inflammatory cytokines such as IL-6 and neutrophil counts, alongside a decrease in TNF-alpha (Eimonte et al., 2021), and an increase of anti-inflammatory IL-10 following CWI after intense exercise (Rowsell et al., 2014).

However, Peake et al. (2017) demonstrated that while CWI is effective for alleviating perceived muscle soreness, its impact on objective markers of muscle inflammation (e.g., neutrophil and macrophage infiltration) and cellular stress is not superior to active recovery. This evidence suggests that the primary benefit of immediate recovery may be sensory and psychological rather than structural or physiological. This distinction is critical as it unveils the paradox: the very protocol identified by Machado et al. (2016) as most effective for sensory relief and perceived readiness is nearly identical to the conditions found to most severely hinder anabolic signaling and ribosome biogenesis (Figueiredo et al., 2016; Roberts et al., 2015). Furthermore, research into the psychological components of recovery (Broatch et al., 2014) suggests that a substantial portion of acute performance restoration may be driven by a robust placebo effect, which enhances the athlete's perceived readiness to return to competition, despite the underlying suppression of myogenic processes (Fuchs, Kouw, et al., 2020; Roberts et al., 2015). Therefore, cold water immersion seems to have far more profound impact on muscle metabolism than the temporary relief provided by its analgesic effects (Peake et al., 2017; Roberts et al., 2015). While both elite and recreational athletes frequently utilize CWI for its potent analgesic effects to reduce perceived muscle soreness (DOMS), it has been well documented that CWI directly interferes with anabolic signaling pathways following resistance training (Fyfe et al., 2019; Roberts et al., 2015). In their study, Roberts et al. (2015) utilized a two-phase approach: an acute phase (n=9 males) comparing CWI (10 minutes in ~10°C) following resistance training with active recovery (10 minutes on stationary bike) using a within-subject design, and a long-term phase (n=21) involving 12 weeks of strength training (twice per week). Muscle biopsies taken during the acute phase, before and after each training session, revealed several key regulatory disruptions. Most notably, the phosphorylation of p70S6K (primary indicator of mTORC1 activity and a crucial regulator of protein synthesis) was significantly suppressed in the leg subjected to cold water immersion compared to the leg treated with active recovery. Furthermore, downstream elements of the pathway, such as rps6, have also been shown to be inhibited by post-exercise water cooling (Fyfe et al., 2019). Additionally, CWI was found to attenuate the normal increase in satellite cells (CD56+)

proliferation for up to 2 days post-exercise (Roberts et al., 2015). This suppression is particularly detrimental as these cells are essential for long-term muscle repair and growth.
The findings from the long-term phase corroborated these acute molecular disruptions. After 12 weeks of strength training, the CWI group exhibited significantly smaller gains in muscle mass – specifically in muscle fiber cross-sectional area – and lower improvements in maximum strength (1RM) compared to the active recovery group. Collectively, these results demonstrate that by blunting the acute anabolic signaling response and inhibiting satellite cell activity, regular use of CWI following resistance exercise effectively attenuates chronic muscle hypertrophy and strength adaptations (Peake et al., 2020; Roberts et al., 2015).

It has been speculated that this molecular interference extends to the regulation of myogenesis. Research by Peake et al. (2020) yielded more complex results: CWI did not significantly alter the mRNA expression of specific regulatory factors, regulating muscle growth and remodeling, specifically myogenin, compared to active recovery. However, they concluded that regular CWI still attenuates adaptations (by blunting some hypertrophy pathways independently of these specific remodeling factors) suppressing a broader range of genes and proteins involved in protein translation and ribosomal biogenesis. A crucial extension of this evidence is provided by Figueiredo et al. (2016), who shifted the focus from basic protein signaling (mTORC1) to the genetic drivers of muscle hypertrophy, specifically ribosome biogenesis. In their study, nine recreationally active men performed lower-body resistance exercise followed by either 10 minutes of CWI (10°C) or active recovery, after which muscle biopsies were taken at 2, 24 and 48 hours post-exercise. Figueiredo demonstrated that CWI drastically impairs the muscle's translational capacity by suppressing 45S pre-rRNA, a precursor for new ribosomes. Notably, this effect was linked to the inhibition of upstream binding factor (UBF), a key regulator of rDNA transcription, with the suppression lasting for up to 48 hours post-exercise. When combined with the findings of Roberts et al. (2015) – who, using the same experimental model, reported a significant reduction in satellite cell activity – it becomes evident that cold exposure does not merely slow down protein synthesis, but effectively reduces the "cellular machinery" required for long-term muscle growth.

This genetic suppression is further compounded by the findings of Fuchs, Kouw, et al. (2020), who utilized stable isotope tracers to demonstrate that CWI-induced vasoconstriction significantly reduces the incorporation of dietary protein-derived amino acids into the myofibrillar protein pool. Recent work by Betz et al. (2025) has elucidated the precise

mechanism driving this deficit. Using contrast-enhanced ultrasound (CEUS), they confirmed that CWI leads to a drastic reduction in microvascular blood volume (~68% immediately post-immersion), which correlates directly with the blunted amino acid uptake. By limiting nutrient delivery during the critical post-exercise window, CWI creates a dual barrier to hypertrophy: a reduction in translational capacity and a simultaneous shortage of the necessary substrates for protein synthesis (Betz et al., 2025; Fuchs, Kouw, et al., 2020).

While the molecular evidence against CWI is compelling, particularly regarding the suppression of ribosome biogenesis, heat-based interventions offer a physiological counterpoint. In contrast research by Ihsan et al. (2020) and Kakigi et al. (2011) demonstrates that whole-body heat stress (microwave diathermy and water-perfused suit accordingly) can independently stimulate the mTOR signaling pathway, specifically increasing the phosphorylation of Akt, p70S6K and rpS6, which is critical for mRNA translation. This thermal activation of the protein synthesis is further supported by the induction of mRNA levels of heat shock proteins (HSPs), notably HSP70, HSP72 and HSP90 (Ihsan et al., 2020; McGorm et al., 2018). As highlighted by (Dablainville et al., 2025; McGorm et al., 2018), these molecular chaperones play a pivotal role in maintaining cellular proteostasis, protecting proteins and consequently cells from degradation, and potentially enhancing the hypertrophic response to mechanical loading.

Furthermore, Hafen et al. (2019) provided compelling evidence, that using pulsed heat stress may actually enhance the cellular environment for adaptation. Their research on human skeletal muscle revealed that local heat application significantly improves HSP – associated with increase of mitochondrial adaptation and stimulation of mitochondrial biogenesis – processes that are essential for meeting the metabolic demands of muscle repair and growth. Although Hafen's protocol utilized localized heating rather than whole-body immersion, the underlying physiological response – driven by the activation of heat-sensitive pathways – suggests that HWI may similarly promote muscle refueling and glycogen resynthesis (Dablainville et al., 2025; Kim et al., 2020). This contrast is vital: while cold exposure induces metabolic dormancy and vascular restriction (Betz et al., 2025), heat-based strategies facilitate the energetic and vascular capacity required for protein synthesis and long-term adaptation (Hyldahl & Peake, 2020; Kim et al., 2020).

Recent evidence from Dablainville et al. (2025) further strengthens the case for heat-based recovery. Specifically, the study demonstrated that HWI possesses the potential to effectively alleviate muscle soreness and reduce circulating markers of muscle damage, such as creatine

kinase (CK) and myoglobin. Their findings suggest that HWI facilitates a superior regenerative environment by suppressing the pro-inflammatory p-NF-κB expression (likely via HSP70 upregulation) while concurrently upregulating the anti-inflammatory cytokine IL-10. By modulating these key immunological and intracellular signaling pathways, HWI appears to mitigate the secondary damage cascade without inducing the metabolic arrest characteristic of cold-based modalities.

Beyond Protein Synthesis: Vascular Kinetics

Beyond molecular signaling, the physical impact of water immersion and temperature on vascular transport plays a critical role in muscle adaptation and immediate recovery. As highlighted by Wilcock et al. (2006) the hydrostatic pressure exerted during immersion facilitates the displacement of fluids from the periphery to the central circulation, which can assist in the reduction of exercise-induced edema. This mechanism effectively mitigates swelling and perceived fatigue, however the addition of cold stress significantly alters local perfusion dynamics.

While Fuchs, Kouw, et al. (2020) focused on the metabolic outcome – muscle protein synthesis, (Betz et al., 2025; Mawhinney et al., 2017) provided the plausible mechanical explanation. The former was a highly controlled study involving 12 young male participants. A defining feature of this methodology was the ingestion of 20g of intrinsically [1-13C]-phenylalanine-labeled milk protein following resistance exercise, combined with CWI (8°C for 20 min) on one leg and thermoneutral water (30°C) on contralateral leg as a control over a 2-week period. By analyzing muscle biopsies collected at two and five hours post training, vital window for immediate recovery, the study revealed that CWI significantly reduced the delivery of dietary amino acids, thereby lowering incorporation of dietary-derived amino-acids into postprandial myofibrillar protein synthesis. This post-exercise cold-induced vasoconstriction resulted in a 20% lower rate of myofibrillar protein synthesis (MPS) during the acute phase. Furthermore, repetitive application of cooling was shown to lower the daily MPS rate by approximately 12% over a two-week period.

These findings are further supported by Mawhinney et al. (2017) who utilized laser Doppler flowmetry and contrast-enhanced ultrasonography to demonstrate a greater reduction in femoral artery conductance and cutaneous vasoconstriction following CWI compared to cryotherapy. According to Betz et al. (2025), the correlation between vascular limitations

induced by CWI and the impaired delivery of amino-acids – therefore anabolic efficiency – is significant. Consequently, while athletes often use CWI for its rapid analgesic effects and edema reduction via hydrostatic pressure (Wilcock et al., 2006), the resulting impairment of muscular perfusion appears to fundamentally obstruct the early substrate-dependent stages of muscle repair and remodeling.

In contrast to the vascular impairment caused by CWI, heat-based strategies such as hot water immersion (HWI) appear not to impair the physiological processes of immediate recovery. Unlike the vasoconstrictive limitations observed in CWI, heat-induced vasodilation (Akerman et al., 2016; Roxburgh et al., 2026) improves microvascular perfusion, and cardiac output (Akerman et al., 2016; Wilcock et al., 2006). Crucially, Kim et al. (2020) demonstrated that repeated heat exposure can promote angiogenesis and increase microvascular density, thereby expanding the capillary network available for nutrient exchange. By facilitating this hyperemic response, HWI ensures a more efficient elimination of metabolic by-products, such as creatine kinase and myoglobin (Dablainville et al., 2025) and delivery of energetic substrates and amino acids to the recovering musculature.

While Fuchs, Smeets, et al. (2020) demonstrated that HWI does not further augment postprandial myofibrillar protein synthesis rates or amino acid incorporation beyond levels observed in thermoneutral conditions, it crucially avoids the ~20% reduction in MPS associated with cooling. By maintaining vascular patency and avoiding the cooling-induced restriction of nutrient delivery, HWI ensures that the post-exercise anabolic environment remains undisturbed. Consequently, HWI serves as a superior alternative for immediate recovery when the primary objective is to maximize the hypertrophic stimulus and maintain the integrity of muscle protein synthesis (Dablainville et al., 2025; Kim et al., 2020).

The Impact of Water Immersion on Chronic Training Adaptations: Muscle Hypertrophy, Maximum Strength, and Rate of Force Development

Shifting focus from the biochemical and vascular impairments to practical performance outcomes, it is crucial to analyze the studies of Fyfe et al. (2019). Their protocol involved a seven-week progressive resistance training program performed by sixteen men who had been sedentary regarding strength training for at least six months prior to the study. To ensure baseline equivalence, a pair-matching strategy was employed based on initial leg press one-repetition maximum (1RM) strength before subjects were assigned to either a post-exercise CWI protocol (10-15°C for 10-15 min) or a thermoneutral control condition. It demonstrated

that chronic use of CWI significantly attenuates muscle hypertrophy – specifically type II muscle fibber cross-sectional area and whole-body lean mass measured via DXA and immunohistochemy (muscle biopsies) and does not necessarily impair maximal strength (1RM). Crucially, Fyfe highlights a potential discordance between these outcomes, suggesting that the CWI-mediated blunting of anabolic responses and increased markers of protein degradation (e.g., FOXO1) may specifically target muscle growth without a proportional deficit in maximal strength. This suggests that training-induced changes in muscle mass and strength can diverge, potentially due to preserved neural adaptations that compensate for the attenuated fiber hypertrophy. Nevertheless, the findings highlight the ability of CWI to blunt muscle growth and suggest the avoidance of post-exercise CWI when hypertrophy is the primary desired outcome.

Building upon these findings, Benoît et al. (2024) examined the effects of different water temperatures on recovery following exercise-induced muscle damage (EIMD) protocol in thirty physically active males. Participants were assigned to either cold water immersion (11°C), hot water immersion (41°C) or thermoneutral water control (36°C) 30 minutes post exercise. The study assessed both maximal isokinetic strength (peak torque) and explosive strength, specifically the late-phase rate of force development (RFD). The data suggest that the selection between extreme water temperatures dictates which specific aspect of muscle function is preserved following EIMD. While CWI appears to offer a protective effect limited to maximal isokinetic strength, it fails to prevent the decline in explosive force production. In contrast, the application of HWI provides superior physiological stimulus for rapid restoration of the late-phase rate of force development. This implies CWI may be counterproductive in athletic context where power and explosiveness are invaluable, however it still may hold value in disciplines, where absolute force production and pain management are prioritized (e.g., multi-day strength competitions, powerlifting).

However, reliance on CWI for strength preservation warrants caution, particularly for trained athletes. While Fyfe observed preserved 1RM strength in novice lifters, Roberts et al. (2015) presented a more concerning scenario for resistance-trained individuals. In their comprehensive 12-week study, regular CWI (10 min at 10°C) significantly attenuated not only muscle mass accrual but also long-term gains in absolute strength (1RM leg press) compared to active recovery. The authors identified a distinct suppression of the acute anabolic signaling pathway – specifically p70S6K – and, crucially, a blunting of satellite cell activity (NCAM+ and Pax7+ cells) within 48 hours post-exercise. This indicates that CWI acts as a “cellular brake”,

disrupting the proliferation of satellite cells necessary for repairing and reinforcing muscle fibers.

This observation challenges the “potential discordance” hypothesis proposed by Fyfe et al. (2019), who suggested that while CWI blunts hypertrophy without impairing maximal strength, attributing this to a preservation of neural function, the underlying mechanistic data suggest a more specific failure. Specifically, CWI appears to selectively impair the structural cellular machinery required for fiber adaptation while leaving certain neural or regulatory signals untouched. Consequently, while 1RM strength may be preserved in the short term, the accumulated evidence suggests that CWI compromises long-term gains in muscle mass and explosive capacity and ultimately limits the muscle's genetic potential for long-term hypertrophy.

Furthermore, reviews by McGorm et al. (2018) and experimental trials by Stadnyk et al. (2018) suggest that regular heat exposure does not hinder maximal strength or hypertrophy. Instead, heat therapy may support skeletal muscle mass preservation and regeneration through the increased basal expression of heat shock proteins (specifically HSP70) and enhanced muscle perfusion (Dablainville et al., 2025; Hafen et al., 2019). By facilitating rather than obstructing the immediate recovery process, HWI allows athletes to benefit from the full hypertrophic stimulus of each training session, avoiding the suppression of regenerative pathways associated with cooling., Crucially, Benoît et al. (2024) demonstrated that HWI actively restores the late-phase rate of force development (RFD), whereas CWI fails to prevent its decline. This stands in contrast to the interference of CWI, where the blunting of anabolic signaling and satellite cell activity leads to diminished hypertrophy and compromised explosive power, as identified by Benoît et al. (2024) and Fyfe et al. (2019).

Practical Applications

To facilitate evidence-based decision-making for practitioners, the strategic application of thermal modalities based on specific physiological objectives is summarized in Table 1.

For athletes, whose primary objective is the accrual of muscle mass or maximal strength (e.g., bodybuilders, powerlifters), the routine use of CWI during the immediate recovery phase is counterproductive.

- Recommendation: These individuals should prioritize active recovery or HWI. Utilizing HWI maintains the muscle perfusion and amino acid delivery (Fuchs, Kouw, et al., 2020;

Kim et al., 2020) while supporting mitochondrial health through heat-induced signaling (Hafen et al., 2019).

- The Verdict: If hypertrophy is the goal, CWI is the enemy of adaptation (Fuchs, Kouw, et al., 2020; Fyfe et al., 2019).

In scenarios where athletes must perform multiple times within a 24-48-hour window (e.g., tournament formats, martial arts, or multi-stage cycling), the priority shifts from long-term adaptation to acute performance maintenance.

- Recommendation: In this context, the analgesic and edema-reducing of CWI are invaluable. By reducing perceived soreness (DOMS) and limb swelling via hydrostatic pressure and vasoconstriction, CWI allows the athlete to "mask" fatigue and return to competition with higher psychological and perceived readiness (Hohenauer et al., 2015; Machado et al., 2016; Wilcock et al., 2006). Crucially, while CWI may blunt growth, it can preserve maximal isokinetic strength in the short term better than passive recovery, although it is less effective than heat for recovering explosive rate of force development (Benoît et al., 2024). Note that CWI does not necessarily reduce histological inflammation compared to active recovery but rather alters the perception of pain (Dablainville et al., 2025; Peake et al., 2017).
- The verdict: During competition, CWI is a tool for survival and maintenance, during off-season training, it should be avoided.

For recreationally active individuals (the demographic primarily studied by Fyfe and Benoit), the choice depends on the motive for exercise.

- Recommendation: If the goal is general health and metabolic fitness, HWI may offer superior benefits by enhancing mitochondrial biogenesis (Hafen et al., 2019), promoting angiogenesis and microvascular density (Ihsan et al., 2020; Kim et al., 2020), and improving cardiovascular health by reducing blood pressure (Roxburgh et al., 2026). As a result, this enhanced perfusion ensures better nutrition and hydration of the tissues while facilitating a favorable regenerative environment (Dablainville et al., 2025).
- The Verdict: For longevity and metabolic health, heat-based recovery offers a more synergistic profile with the body's natural adaptive processes.

Table 1. Strategic Application of Water Immersion Modalities: A Guideline Based on Physiological Objectives and Training Context.

(Benoît et al., 2024; Betz et al., 2025; Bleakley et al., 2012; Dablainville et al., 2025; Figueiredo et al., 2016; Fyfe et al., 2019; Iguchi et al., 2012; Ihsan et al., 2016; Machado et al., 2016; Mawhinney et al., 2017; McGorm et al., 2018; Roberts et al., 2015; Versey et al., 2013; Wang et al., 2025; Wilcock et al., 2006).

Scenario	Recommended Modality	Rationale
Hypertrophy/Strength Training	HWI or Neutral/Active	Preserves mTORC1 signaling and ribosome biogenesis, improves microvascularization; preserves RFD.
High-frequency Competition	CWI (10-15°C, 10-15 min)	Prioritizes pain management, perceived readiness, and CNS recovery; preserves maximal isokinetic strength.
Metabolic health/longevity	HWI/Heat Therapy	Stimulates mitochondrial biogenesis and HSP expression.
Acute Injury/Inflammation	CWI	Targeted application to limit secondary tissue damage via localized vasoconstriction and reduced metabolic demand.

CONCLUSIONS

This review confirms the existence of a profound “Recovery-Adaptation Paradox” inherent in thermal water immersion. The objective was to determine which strategy optimizes the balance between immediate relief and chronic gains. the evidence suggests that the two goals are often physiologically antagonistic.

Cold water immersion (CWI) achieves the objective of acute recovery through potent analgesia and edema reduction, but it simultaneously disrupts the essential molecular triggers – such as mTORC1 signaling and ribosome biogenesis – required for structural remodeling. This results

in the observed potential discordance, where subjective readiness is restored at the expense of long-term hypertrophy and explosive force production (RFD). In contrast, hot water immersion (HWI) emerges as a facilitative modality that aligns with the body's endogenous adaptive pathways. By promoting vasodilation, enhancing nutrient delivery, and inducing cytoprotective heat shock proteins, HWI supports the restoration of muscle function without compromising the anabolic infrastructure of the muscle characteristic of CWI.

In conclusion, to optimize the balance between recovery and adaptation, the routine use of CWI should be replaced by an objective-dependent framework proposed in this study. Athletes prioritizing structural growth and explosive power should avoid routine CWI in favor of HWI or active recovery. CWI should be reserved exclusively for scenarios where immediate performance maintenance and "fatigue masking" are prioritized over long-term muscular development. This shift in perspective ensures that the chosen recovery modality does not inadvertently compromise the adaptive response.

DISCLOSURE

Author's contribution

Conceptualization: K. Brankowska; methodology: Z. Wiater, A. Włodarczyk; software: K. Swoboda; check: K. Brankowska, A. Dziegciarczyk; formal analysis: M. Olejnik, P. Górka, Sz. Domagała; investigation: W. Kądziołka, M. Jakubowska, M. Olejnik; resources: K. Brankowska, M. Jakubowska, A. Włodarczyk; data curation: K. Brankowska, A. Dziegciarczyk; writing-rough preparation: K. Brankowska, K. Swoboda, Z. Wiater; writing-review and editing: K. Brankowska, Sz. Domagała, P. Górka; visualization: W. Kądziołka, M. Olejnik, A. Włodarczyk; supervision: M. Jakubowska, Z. Wiater, K. Swoboda; project administration: K. Brankowska.

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The authors deny any conflict of interest.

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