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Muscle cooling: too much of a good thing?

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It is well documented that strenuous exercise elicits stress on the body, which may lead to physiological impairments and associated reductions in muscle function and fatigue, in the hours and days post-exercise. Whilst fatigue is specific to the mode, intensity and duration of exercise, a failure in any of the related neural, metabolic or mechanical processes may hinder subsequent athletic performance, particularly when insufficient restoration periods are provided. This has led to significant interest in recovery strategies to ameliorate post-exercise fatigue and to optimize an athlete's physical capabilities.

During prolonged high-intensity exercise, the substantial reliance on oxidative glycolytic-phosphorylation may lead to fatigue via depletion of local muscle and liver glycogen stores. The low glycogen levels not only compromise the rate of adenosine triphosphate (ATP) regeneration but may have a glycogen-dependent role in excitation-contraction coupling (E-C coupling) failure; proposed to cause fatigue by impairing Ca^{2+} release from the sarcoplasmic reticulum (Ørtenblad *et al*. 2011). Consequently, it is important that post-exercise glycogen stores are sufficiently replenished before the next bout of exercise to limit the effects of fatigue on muscle function and performance. Passive recovery methods, which do not raise metabolism (i.e., without muscle activation), may therefore be more suitable to maximize the rate of post-exercise glycogen resynthesis when only a short recovery period is permitted. A popular and widely used passive recovery method, cold water immersion (CWI), is thought to expedite recovery via associated reductions in tissue temperature to benefit recovery outcomes. Importantly, local changes in muscle temperature (heating or cooling) may influence enzymatic activity and affect rates of intramuscular glycogen synthesis or breakdown. It has therefore become of interest to relate changes in intramuscular temperature with local muscle glycogen kinetics to quantitatively assess the impact of applied recovery strategies, especially methods that can markedly alter tissue temperature, i.e., CWI.

In a recent study published in the *Journal of Physiology*, Cheng and colleagues (2017) investigated whether the manipulation of intramuscular temperature affected the acute recovery of

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exercise performance following fatigue induced by endurance exercise. In a randomized cross-over designed study, subjects initially performed 3 x 5 min all-out arm exercise at 100 rpm (fatigue test) before completing 4 x 15 min bouts of exhaustive arm cycling exercise at 50% VO_{2peak} (endurance exercise). This was followed by a 2 h recovery period, where each arm was either 1) continuously perfused with ice chilled water using modified arm cuffs (cooling), or 2) maintained at physiological temperature (~33°C) using modified arm cuffs (control), or 3) heated to 5°C above physiological temperature (~38°C) using a temperature controlled bath (heating). During the recovery period, intramuscular triceps brachii temperature (1.5 cm depth) was continuously recorded and subjects were provided with 1.1 $g \cdot kg \cdot h^{-1}$ bodyweight of carbohydrates to maximize glycogen repletion. The fatigue test was then repeated to determine if the recovery intervention had an effect on muscle function. The main findings showed that the maintenance of power output (i.e., endurance) during the arm cycling fatigue test was markedly preserved (increased in final set) when each arm was heated to an intramuscular temperature of ~38°C, compared with cooling the muscle (to as low as 15°C).

In order to identify the cellular mechanisms underlying the temperature dependent recovery of muscle force and $[Ca2^{+}]_i$ after fatigue, the authors carried out a series of isolated whole mouse muscle experiments, obtaining intact single *flexor digitorum brevis* (FDB) muscle fibres from hindlimbs. The fibres were initially stimulated with 350 ms duration tetani once every 1 min at 30, 70 and 120 Hz, with peak force, and spatial and time-averaged $[Ca^{2+}]_i$ (fluorescent indicator indo-1), measured during tetani. To markedly decrease glycogen, fibres were perfused with glucose-free Tyrode before being repeatedly stimulated for ~12 min at 350 ms (70 Hz) tetani at 10 s intervals until force had decreased to 30% of starting values. The fibres were then perfused with glucose-containing Tyrode for up to 2 h at either 16°C (<10°C FDB muscle temperature), 26°C (<5°C FDB muscle temperature), 31°C (~FDB muscle temperature) or 36°C (>5°C FDB muscle temperature) temperatures. Additional isolated fibres were also perfused with glucose-free Tyrode, at the same temperatures, to assess whether force recovery was dependent on restoration of glycogen *per se*. During the recovery period, fibres were stimulated every 30 min by repeating the tetani (350 ms every 1 min) applied at 30, 70 and 120 Hz, to assess recovery of contractile muscle function. Upon completion of the 2 h recovery period, the fibres were again exposed to the glycogen-depleting fatiguing stimulation protocol to elucidate the effects of the different perfusion temperatures on glucose availability and muscle fatigue.

A further experiment determined the effects of temperature on muscle glycogen content using a fluorometric glycogen assay. Whole FDB muscles were fatigued in glucose-free Tyrode with repeated 70 Hz tetani at 31°C using both moderate (700 ms duration contractions every 10 s for 150 tetani) and severe (1 s contractions every 5 s for 300 tetani) glycogen depleting protocols. The fatigued muscles were subsequently assessed for the extent of glycogen depletion with additional isolated FDB muscles, which were not prior stimulated, used to represent glycogen content before fatigue. The stimulated muscles were then bathed in 26°C or 36°C Tyrode containing glucose for a duration of 30 min to assess glycogen resynthesis.

Supporting the results in the human experiment, it was observed that measurements of submaximal force (30 Hz and 70 Hz), tetanic myoplasmic free Ca^{2+} (30 Hz, 70 Hz and 120 Hz) and fatigue resistance (number of contractions performed) were all reduced when glucose-containing Tyrode was perfused in to the fibres at temperatures between 16-26°C (i.e., cooling below FDB physiological temperature), but better maintained after the perfusion temperature was raised to 36°C (i.e., heating above physiological FDB temperature). This confirmed the author's earlier findings that contractile force was dependent on muscle fibre temperature by influencing the rate of glycogen

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resynthesis, which in turn modified the release of tetanic $[Ca^{2+}]_i$. Indeed, in the absence of perfused glucose, tetanic myoplasmic free Ca^{2+} and force were considerably impaired. In addition, after the severe glycogen depletion protocol, muscle glycogen content was greater at the end of the 30 min recovery period after perfusion with glucose at a 36°C compared with 26°C, indicating a greater rate of glycogen resynthesis at the higher temperature. In combination, the authors therefore concluded that the lower contractile force recorded at the lower frequencies (30 Hz and 70 Hz) demonstrated that the fatigue protocol was both glucose and temperature dependent.

Cheng and colleagues observations that greater reductions in intramuscular temperature decrease both the rate of glycogen resynthesis and contractile force challenges the use of CWI as a recovery strategy, since its application is partly based on expediting these aspects of recovery in the acute post-exercise period. However, it must be stated that the design of Cheng and colleagues utilised a 2 h recovery period (cooling or heating), whereas most CWI protocols are typically applied over much shorter durations (i.e., 10-15 min) that do not permit intramuscular temperatures to reach such low temperatures (i.e., ~15°C). Previous work in our laboratory (Gregson et al, 2013) has shown 10 min of 8°C CWI does not negatively impact on the rate of glycogen resynthesis compared with seated rest (control) after initially depleting muscle glycogen content with a cycling exercise protocol. The disparity in our findings is likely related to the shorter duration of cooling influencing muscle temperature to a lesser degree, with deep intramuscular temperature only reduced to ~30°C in our study. Thus negative implications within contraction kinetics and muscle glycogen resynthesis highlighted by Cheng et al. may not be apparent in recovery protocols regularly used in research and practice because of shorter cooling durations (10-15 min) and smaller decreases in intramuscular temperature (~30°C). This perhaps highlights the need to identify the extent of intramuscular cooling required to impair post-exercise recovery of skeletal muscle contractile function and metabolism, as opposed to improve recovery. Moreover it emphasises the need for context to be applied to mechanistic results before they can be applied appropriately in practice.

In Cheng et al's study there was no reported reduction in core temperature or increase in oxygen uptake during the 2 h arm-cooling period. It would be expected that with a greater skin exposure to CWI (i.e., lower body or sternum CWI) that core body temperature would decrease over a similar cooling duration. In an attempt to maintain core body temperature, shivering thermogenesis requires contribution from muscle glycogen stores as a fuel source (Haman *et al*. 2005). Consequently, a prolonged duration of post-exercise CWI cooling, which leads to a drop in core body temperature and onset of shivering, may potentially decrease the rate of glycogen resynthesis. Moreover, Cheng and colleagues utilised upper limbs only, constituting a small area of muscle mass. It should be noted that the potential for heat loss is greater when a larger muscle mass is cooled (i.e. lower limbs) and the understanding of the implications this may have upon shivering thermogenesis and muscle glycogen utilisation becomes essential for those utilising cooling as a method of athletic recovery.

Interestingly, it has also been reported that both 10°C and 20°C CWI applied over 30 min exacerbates blood pro-inflammatory markers (Interleukin-8, myeloperoxidase) 2 h post-exercise compared with the same temperatures applied over a 10 min duration (White *et al*. 2014). This suggests that longer CWI protocols may increase the inflammatory response and associated oedema and swelling, possibly leading to secondary damage in muscle fibres, a reduction in muscle force generating capacity and an increased sensation of muscle pain (i.e., delayed onset muscle soreness). Importantly, in athletic settings, an excessive duration of cooling, like the 2 h utilised by Cheng and colleagues, loses its practicality as a post-exercise recovery strategy due to the time investment of the

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athlete, impaired contraction kinetics and metabolic recovery and the potential for further negative implications via exacerbated humoral inflammatory markers.

The author's findings of an increased rate of glycogen reysnthesis and improved muscle function with higher intramuscular temperatures partly justifies the recent anecdotal interest surrounding hot baths and saunas to aid post-exercise recovery. Moreover, data from Cheng and colleagues provide interesting and commendable detail to the already established notion of improved muscle force generation and function at higher intramuscular temperatures. The direct application of the results to post-exercise recovery in athletes needs careful attention. In the future, it is expected that the selection of hot and cold therapies will be dependent on the athlete's individual needs and based on the main goal of the recovery session, i.e., reduce inflammation, enhance fuel repletion, and/or promote cellular adaptations.

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Additional Information

Competing Interests

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