Post-exercise recovery regimes; blowing hot and cold

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Athletes aiming to improve upon past performances often look to train harder and longer than ever before, but training hard in a single session is of little use if ensuing muscle damage and soreness conspire with other factors to limit performance over the following days. The promise that post-exercise habits may be modified to boost physiological adaptation or recovery is therefore very appealing. The utilisation of post-exercise recovery techniques, via active recovery, cold-water immersion (CWI) or other therapies is a component of the training regimes of many elite athletes. Clearly, the use of a regime post-exercise to aid in recovery is demonstrably better than adopting a sedentary approach. However, the choice of using CWI, or potential benefits of using one recovery strategy over the other needs to be justified, preferably using evidence of efficacious effects, rather than being based solely on personal preferences. In the current issue of *The Journal of Physiology*, Peake et al. attempt to address this question by comparing the impact of CWI with active recovery on a range of parameters relating to skeletal muscle adaptation, inflammation and repair post-exercise. It is reasoned that reducing the temperature of the muscle and thus, blood flow using CWI may dampen the local inflammatory response, helping to reduce the extent of cell and tissue damage, and thereby boost post-exercise recovery. What is striking in the blanket use of CWI as a post-exercise recovery technique is the assumption that inflammation needs to be dampened down or suppressed. The inflammation, which occurs post-exercise, is a crucial component of the adaptive response of skeletal muscle to training. A significant proportion of the scientific evidence justifying the use of CWI as a means of aiding post-exercise recovery (in an anti-inflammatory context) stems from a range of studies conducted in rodents, many using models of gross muscle injury, where the application to human physiology is not clear.

In the present study, Peake et al. examined inflammatory cell invasion into muscle, alongside a range of cytokines, heat shock proteins (HSPs) and neurotrophins in muscle, at 2, 24 and 48 hours post-heavy resistance exercise, with or without a period of CWI. The authors observed expected increases in inflammatory and cellular stress components in muscle after the exercise. However, there was no anti-inflammatory effect observed in any of the parameters, upon intervention with CWI. These

findings are striking and may well pave the way for an alternative approach to post-exercise recovery; however, a closer examination of the impact of CWI is needed before we say anything that may discredit the vocal advocates. The mechanisms, which regulate inflammation in muscle post-exercise, are complex, encompassing a range of cytokines, chemokines and adhesion molecules. Peake and colleagues examined a discrete range of components involved in these processes, for reasons that were well justified, but the interrogation of a wider range of inflammatory markers may yield a more robust assessment of the impact of CWI. Examination of receptors complimentary to the cytokines/chemokines (e.g. CCR2/4 are receptors for CCL2) described in this study, alongside expression of the cyclo-oxygenases (COX) and nitric oxide synthases (NOS) may provide additional mechanistic insight into the adaptive responses. The measurements taken by Peake et al. are to a degree temporal, and the examination of a range of parameters between 2 and 24 hours may again provide greater insight into the complex mechanisms of inflammation post-exercise.

The study by Peake et al. focuses heavily on the inflammatory aspect of exercise; however, there are numerous adaptive processes, which are occurring in muscle post-exercise. CWI has been demonstrated to benefit some contexts and less so in others. Thus, it is important to consider that the differential effect of post-exercise CWI are likely dependent upon the nature of exercise undertaken, as well as the duration and regimen of CWI. For instance, a recent study examined the impact of post-exercise CWI, following intermittent sprint training, on expression of Peroxisome proliferator activated-receptor γ co-activator-1 α (PGC-1α) a regulator of mitochondrial biogenesis, and Vascular endothelial growth factor (VEGF) (Joo *et al.*, 2016). In this study, participants carried out an acute bout of exercise followed by CWI (10mins at 8^{o}C) and it was reported that CWI augmented the exercised-induced gene expression of PGC-1α, and that CWI alone could activate VEGF and PGC1-α. In a similar study, CWI (15mins 10^{o}C) was also shown to enhance PGC1-α gene expression in muscle after 30mins continuous running (70% VO_2max), which was followed by intermittent running to exhaustion (100%VO_2max). In contrast, there was no significant effect of post-exercise CWI on VEGF or NOS gene expression (Ihsan *et al.*, 2014). Given the well-established role of PGC-1α and VEGF in the adaptive

response of muscle to exercise, these studies indicate that CWI may play an important role in mediating these changes.

In the context of resistance training, regular use of post-exercise CWI has been reported to attenuate the long-term adaptations (increased strength and muscle mass) to a 3 month period of training (Roberts *et al.*, 2015). In contrast, regular post-exercise CWI throughout a 4 week period of endurance training enhanced p38 mitogen activated protein kinases (p38 MAPK), adenosine monophosphate-activated protein kinase (AMPK) and PGC-1α (Ihsan *et al.*, 2015). Collectively, these studies highlight the need to understand the impact of CWI in both the short- and long-term, taking into account the mode of exercise (Ihsan *et al.*, 2016). Thus, an integrated approach, encompassing molecular and physiological analyses, is needed to comprehensively address the impact of post-exercise CWI.

Muscle pain and soreness are a typical feature of the adaptive processes occurring after exercise (Leeder *et al.*, 2012). There is clear evidence to demonstrate the efficacy of CWI in improving the perception of pain and fatigue post-exercise; however, Peake et al suggest this may not be derived from changes at the muscle level. Thus, there may be a psychological element or centrally-mediated effects to the (possibly perceived) benefits of CWI post-exercise (Broatch *et al.*, 2014) – further investigation into this aspect is needed, as psychological-based approaches are already commonplace in the training of elite athletes.

Overall, the study by Peake et al challenges the dogma that CWI is a crucial anti-inflammatory component of post-exercise recovery. However, further work is needed to stratify the impact of CWI on a wide range of adaptive markers in muscle, and in relation to exercise type, duration and intensity.

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