Cold water immersion (CWI) and other forms of cryotherapy have existed for centuries but have recently become increasingly popular as recovery modalities for athletes. CWI is commonly used in athletic populations for speeding recovery following high intensity exercise including endurance events, highly eccentric exercise, high impact exercise, and other mechanically/metabolically ‘stressful’ exercise types. However, the research regarding the efficacy of CWI and the mechanism through which it promotes recovery and facilitates performance is equivocal, and the methodology, heterogeneous. Despite this heterogeneity, the putative mechanism through which CWI is thought to be effective is through anti-inflammatory effects.

High-intensity exercise is known to cause inflammation (Pedersen, 2000), which can lead to secondary tissue damage leading to soreness and performance decrements associated with stressful exercise. While inflammation is necessary for repair and adaptation of muscles (Tidball, 2004; Ten Broek et al., 2010), excessive or unabated inflammation is thought to contribute to further damage to stressed tissues due to the non-specific phagocytic function of inflammatory cells like neutrophils. CWI and other cryotherapeutic modalities are thought to be effective recovery strategies by reducing the inflammatory process following exercise, thereby mitigating any additional damage caused by the inflammatory response and not the exercise per se. The mechanisms by which CWI is supposed to blunt inflammation are twofold. Firstly, it attenuates metabolic processes in stressed tissues, thereby slowing the up-regulation of cytokines and myokines which mediate inflammation. Secondly, CWI causes vasoconstriction of the microvasculature perfusing stressed tissue beds, thereby reducing the circulatory exposure of the tissue to inflammatory cells (White & Wells, 2013).

While some convincing evidence of the anti-inflammatory efficacy of cryotherapies exists in animal models (Takagi et al., 2011; Vieira Ramo et al., 2016), the majority of human studies focus on performance outcomes, typically muscle function and/or soreness outcomes of CWI and a large degree of variability exists. In this issue of The Journal of Physiology, Peake et al. (2017) attempt to validate the anti-inflammatory effects of CWI as a post-exercise muscle recovery mechanism by comparing these effects post exercise with active recovery, which was not expected to affect the inflammatory response. They used unilateral resistance training exercise in trained males to elicit an inflammatory response and then treated them with either active recovery (self-selected cycling for 10 min) or CWI (10°C water immersion to the waist for 10 min). Inflammatory processes were measured from change from baseline of cytokine/chemokine and cell signalling gene regulation and inflammatory cell infiltration (neutrophil and macrophage counts) from vastus lateralis biopsies and blood samples at 2 h, 24 h, and 48 h post exercise. Overall, Peake et al. found that exercise induced a robust inflammatory response in both trials and no difference in inflammatory processes were observed between trials.

This is an important research question in this area of sports science and has yet to be thoroughly vetted, undermining the existing research reports in this area which cite anti-inflammatory effects of CWI without substantiation. The authors should be commended on their attempt to elucidate the mechanisms of this common recovery modality and comprehensive investigation of post-exercise inflammatory processes. Their previous work in this area indicates that when CWI is used chronically, blunted adaptive processes post exercise occur (Roberts et al., 2015). This observation leads naturally into this study’s research question, strengthening the theoretical purpose of the study. The use of biopsies and blood samples to measure inflammatory signalling at the gene and circulatory level and subsequent infiltration of inflammatory cells, in addition to damage markers, allows for relevant elements of the post-exercise inflammatory process that may be influenced by CWI and that may affect soreness and recovery processes to be evaluated simultaneously. Overall, the study has many strengths and many of the sampling techniques used should be employed by other studies in this field.

We would like to address some points which detract from the ultimate potential of the findings by Peake et al. (2017). The major criticisms we have of this study are the lack of a control condition and functional measures and oversimplification of their findings, possibly related to statistical limitations. While many of these issues are acknowledged by the authors, and in some cases justified, the implications are worth elaborating upon.

The use of active recovery as the comparison condition to validate CWI as an anti-inflammatory treatment is a major concern of this paper. The fundamental intention of the research question is to understand if CWI reduces the inflammatory response to exercise and to answer this question accurately a comparison to no recovery method must be made. While the authors acknowledge this and rationalize it by noting that athletes using CWI are not likely to employ a sedentary post-exercise strategy, it undermines their ability to accurately address the research question. A non-intervention control group is required to elucidate the inflammatory effects of one protocol when comparing two interventions with known haemodynamic and metabolic effects, of which both processes are thought to be mechanistic in inflammatory regulation post exercise. While the rationalization that the beneficiaries of CWI research are athletes who would be unlikely to use a sedentary recovery strategy holds merit, it ignores a potentially larger population of recreationally active individuals who may engage in resistance training without
the use of any intentional recovery modality.
Although the comparison of CWI to active recovery has some practical justification, the lack of functional measurements, such as muscle soreness and/or performance, makes it difficult to infer applicable conclusions regarding the efficacy of the respective recovery types. We acknowledge that a very comprehensive panel was used to evaluate the inflammatory processes post exercise; however, this would have been complemented by some indicators of performance, function, or feeling. These could have been added very simply without much additional strain on resources and could have provided insights into the efficacy of the treatments as it relates to the purported mechanism, inflammation.

While the authors concluded that there was no difference between active recovery, a supposedly inflammation-neutral modality, and CWI, a purported anti-inflammatory treatment, there are many subtleties in their results that indicate a possible oversimplification of their findings. These null findings may be a function of the small sample size (n = 9) and high degrees of freedom for omnibus statistics, but the lack of discussion regarding the variables in which CWI did not appear to have an anti-inflammatory effect over active recovery is surprising. For example, a number of variables were observed to have a significant increase over baseline in the active recovery condition that were trends or non-significant increases in the CWI condition. Specifically, non-significant changes from baseline were observed in CD66b+ cells at 2 h, MAC1, TNFα, CCL4, IL-8 at 24 h, and CD68+ cells at 48 h.

While there were some time points in which the opposite occurs, responses at the majority of time points appear to be either similar between conditions or to suggest attenuated inflammation in the CWI condition, especially at the later time points. This is important given that the fundamental purpose of using these types of recovery following intense exercise is to facilitate recovery and improve function in the days following (i.e. 24 h and 48 h later). Although it is likely that these discrepancies reflect the low statistical power of the study, it seems worth discussing this in the paper.

Overall, Peake et al. (2017) ask a very crucial question in sports science research and one that is extremely overdue given the litany of studies investigating the efficacy of CWI in various exercise scenarios. While the methods used for the investigation of the inflammatory processes from cellular to systemic level provide a detailed look at post-exercise processes, the lack of a control condition and functional measures leave some aspects of this research question unanswered. A closer inspection of their findings lends some support to the anti-inflammatory effects of CWI but the extent of these changes remain elusive.

References


Additional information
Competing interests
The authors declare no competing interests.