If your goal is to facilitate muscle recovery, think twice before you take an anti-inflammatory drug or strap on a freezing cold bag of ice. A recent study published in the Federation of American Societies for Experimental Biology journal suggests that muscle inflammation is essential for repair. The facts are basic and easy to understand: “drugs” and “ice” slow the recovery process at best, and at worst, prevent it!

The research noted above adds to a growing list of highly-credible peer-reviewed reports that have led many professional and world-class athletes to reject the “quick fix” allure promoted by drug “dealers” and ice “gurus” (both over-the-counter and prescription) and embrace the reality that inflammation is not only desirable, it is essential for optimal recovery!

Further, informed athletes are clearly aware that “inflammation” is not why their muscles feel “tired” and/or “sore,” and more importantly, that the best way to assuage those feelings is a muscle activation technique known as “active recovery,” not by mindlessly popping pills or literally chillin’ out on the couch.

What’s the best muscle activation technique? Theoretically, do whatever you did to get tried and/or sore, but with less intensity. If you played a tennis match and your right shoulder and forearm are tired and/or sore, volley with a friend for 30 minutes or so using only your right hand; if you played a round of golf and your lower back is tired and/or sore, go to the driving range and soft-hit a couple buckets of balls; if you ran a marathon and your thighs are tired and/or sore, jog a mile or two; if you completed a 50-mile bike race through the mountains and your glutes are tired and/or sore, go for a shorter, easier bike ride; if you “over-did-it” in the weight room and your biceps are tired and/or sore, go back to the gym and “under-do-it;” if you worked in the garden all morning pulling big weeds and your hands are tired and/or sore, try pulling some small weeds, etcetera! The point is that it does not matter what caused your muscles to get tired and/or sore. The theoretical best way to facilitate recovery is to activate the involved muscles by “lightly” doing what initially caused you to get tired and/or sore without causing any additional muscle fatigue or soreness.

However, that “theoretical” stuff rarely aligns with reality. Besides, who wants to volley with a friend, hit a couple buckets of balls, jog, ride a bike, go back to the gym, or pull more weeds when their brain says “you’re finished, now rest!”

So what’s truly the best, simplest, most practical, and least stressful way to facilitate muscle recovery? That’s easy. Activate your tired and/or sore muscles using a scientifically validated, FDA cleared, non-fatiguing, rhythmic, slow-pulsed muscle-recovery device.

For related research, see: http://faculty.css.edu/tboone2/asep/JEPonlineOctober2011Blum.pdf.
For various reasons, almost everyone believes that they need to prevent or, at least, limit inflammation related to tissue damage. It doesn’t seem to matter to those with such beliefs that inflammation is phase one of a three-phase, life-saving response to injury or that without it, normal healing is impossible.

But what do the experts say? Is it okay to significantly modify or skip the inflammatory phase of the healing process? What are the facts related to this topic?

Although there are many viable answers to these questions from a wide variety of sources, here are two concise, well-referenced explanations that are consistent with related clinical textbooks and other published evidence-based material:

“A major rationale for using NSAIDs in the treatment of musculoskeletal injuries has been their anti-inflammatory quality. The prevailing argument is that healthy tissue is not inflamed; therefore, if we stop the inflammation in an injured tissue, the tissue will be healthy. The problem with this viewpoint is that, in addition to being a sign of injury, inflammation is a necessary component of the healing process. As noted by Leadbetter ‘inflammation can occur without healing, but healing cannot occur without inflammation.’

Whether the injured tissue is a ligament, tendon, or muscle, the body responds to injury with a sequence of events that begins with an influx of inflammatory cells and blood. The inflammatory cells remove debris and recruit cytokines and other growth factors toward the injury site. This inflammatory phase is partly mediated by the same prostaglandins that are blocked by NSAIDs. In a healthy healing process, a proliferative phase consisting of a mixture of inflammatory cells and fibroblasts naturally follows the inflammatory phase. The fibroblasts build a new extracellular matrix and persist into the final phase of repair, the maturation phase, where, if all goes well, functional tissue is laid down. The key point is that each phase of repair is necessary for the subsequent phase.”

[The Physician and Sportsmedicine: Volume 31: No.1 January 16, 2003 NSAIDs and Musculoskeletal Treatment What Is The Clinical Evidence? Steven D. Stovitz, MD; Robert J. Johnson, MD]

“NSAID use probably should be avoided by patients during fracture healing.”


Retrieved from the Arthritis Foundation

Overwhelmingly, this review demonstrates that NSAIDs inhibit or delay fracture healing.”

[Pharmaceuticals, Vol 3, No 5, 2010: Effect of Non-Steroidal Anti-Inflammatory Drugs on Bone Healing, by Jessica Cottrell, and J. Patrick O’Connor]

What about Swelling?

Swelling is good thing … not a bad thing. It is a necessary and fundamental component of phase one of the healing process. Additionally, and, contrary to popular belief, the amount of fluid sent to the damaged area is not an arbitrary or chaotic event. Instead, it is a vigilantly regulated process designed to help the body regain a homeostatic state; a process that ultimately depends on the lymphatic system to move the fluid and other waste away from the damaged area and back into general circulation (see next page for details).

Granted, sometimes trapped “waste” triggers the inflammatory response (which sends more fluid to the damaged area). But, generally, it’s not because there is too much swelling … it’s because there is too little lymphatic drainage. This issue is best settled by evacuating the waste, not stifling the healing process.
Is Ice (cryotherapy) a Good Idea?

Yes, if the short-term goal is pain control and the prevention of the body’s normal cellular and vascular response to injury.

However, if the goal is to help the lymphatic system evacuate trapped waste from the damaged area, the answer is … no.

Why? Because ice slows everything down. It abates, or worse, shuts off the signals between the nerves and the muscles … which basically stops lymphatic drainage (the lymphatic system works when the surrounding muscles contract and relax: no muscle action, no drainage). In fact, if ice is used beyond ten minutes or so, it actually increases waste in the involved area by causing the lymphatic vessels to backflow.

Here’s what happens: “When ice is applied to a body part for a prolonged period, nearby lymphatic vessels begin to dramatically increase their permeability (lymphatic vessels are ‘dead-end’ tubes which ordinarily help carry excess tissue fluids back into the cardiovascular system). As lymphatic permeability is enhanced, large amounts of fluid begin to pour from the ‘lymphatics ‘in the wrong direction’ (into the injured area), increasing the amount of local swelling and pressure and potentially contributing to greater pain.”


And, if the goal is to improve outcome … the following abstract from the Journal of Emergency Medicine throws a significant amount of cold water on that idea.

“The lymphatic system is a ‘scavenger’ system that removes excess fluid, protein molecules, debris, and other matter from the tissue spaces. When fluid enters the terminal lymphatic capillaries, any motion in the tissues that intermittently compresses the lymphatic capillaries propels the lymph forward through the lymphatic system, eventually emptying the lymph back into the circulation.”


Is Ice Right? Does Cryotherapy Improve Outcome for Acute Soft Tissue Injury?”

Abstract:

“Aims: The use of ice or cryotherapy in the management of acute soft tissue injuries is widely accepted and widely practiced. This review was conducted to examine the medical literature to investigate if there is evidence to support an improvement in clinical outcome following the use of ice or cryotherapy.

Methods: A comprehensive literature search was performed and all human and animal trials or systematic reviews pertaining to soft tissue trauma, ice or cryotherapy were assessed. The clinically relevant outcome measures were:

1. a reduction in pain;
2. a reduction in swelling or edema;
3. improved function; or
4. return to participation in normal activity.

Results: Six relevant trials in humans were identified, four of which lacked randomization and blinding. There were two well-conducted randomized controlled trials, one showing supportive evidence for the use of a cooling gel and the other not reaching statistical significance. Four animal studies showed that modest cooling reduced edema but excessive or prolonged cooling is damaging. There were two systematic reviews, one of which was inconclusive and the other suggested that ice may hasten return to participation.”

Conclusion: There is insufficient evidence to suggest that cryotherapy improves clinical outcome in the management of soft tissue injuries.
Macrophages recruited via CCR2 produce insulin-like growth factor-1 to repair acute skeletal muscle injury

Haiyan Lu, Danping Huang, Noah Saederup, Israel F. Charo, Richard M. Ransohoff and Lan Zhou [January 2011 The FASEB Journal vol. 25 no. 1 358-369]

CC chemokine receptor 2 (CCR2) is essential to acute skeletal muscle injury repair. We studied the subpopulation of inflammatory cells recruited via CCR2 signaling and their cellular functions with respect to muscle regeneration. Mobilization of monocytes/macrophages (MOs/MPs), but not lymphocytes or neutrophils, was impaired from bone marrow to blood and from blood to injured muscle in Ccr2<−/− mice. While the Ly-6C+ but not the Ly-6C− subset of MOs/MPs was significantly reduced in blood, both subsets were drastically reduced in injured muscle of Ccr2<−/− mice. Expression of insulin-like growth factor-1 (IGF-1) was markedly up-regulated in injured muscle of wild-type but not Ccr2<−/− mice. IGF-1 was strongly expressed by macrophages within injured muscle, more prominently by the Ly-6C− subset. A single injection of IGF-1, but not PBS, into injured muscle to replace IGF-1 remarkably improved muscle regeneration in Ccr2<−/− mice. CCR2 was not detected in myogenic cells or capillary endothelial cells in injured muscle to suggest its direct involvement in muscle regeneration or angiogenesis. We conclude that CCR2 is essential to acute skeletal muscle injury repair primarily by recruiting Ly-6C− MOs/MPs. Within injured muscle, these cells conduct phagocytosis, contribute to accumulation of intramuscular Ly-6C− macrophages, and produce a high level of IGF-1 to promote muscle regeneration.

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