Understanding Inflammation and Pain

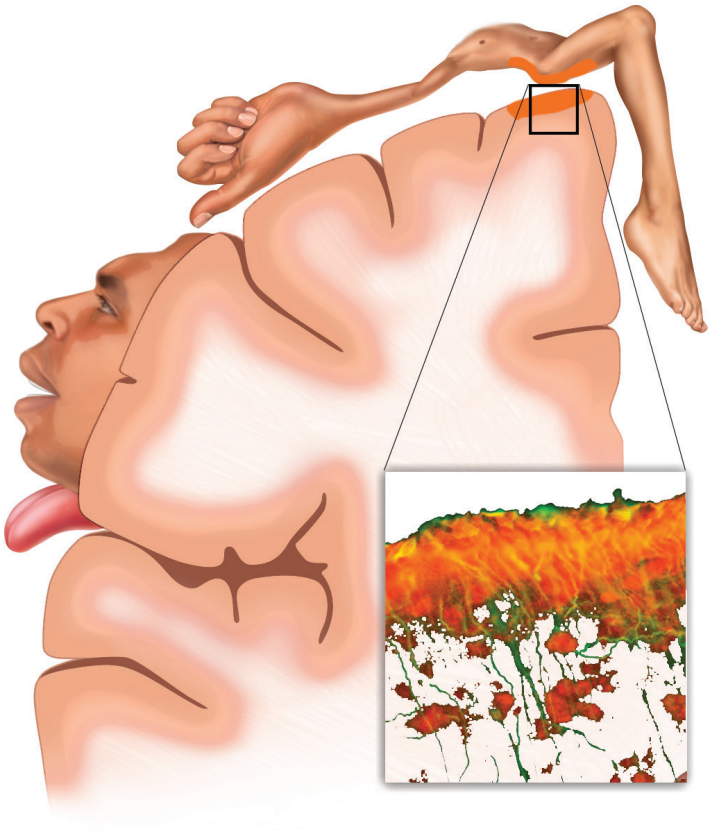
By Til Luchau

Chances are very good that if your client has pain or discomfort, inflammation is to blame; if not wholly, then at least in large part. Inflammation is directly involved in the pain conditions ranging from sciatica to arthritis, whiplash to tennis elbow, and from migraines to postexercise muscle soreness. So how exactly does inflammation trigger pain?

Pain (like inflammation itself) is biologically and behaviorally necessary (see "Understanding Inflammation's Progression," *Massage & Bodywork*, November/December 2018, page 98). Pain protects against tissue damage by shaping our behavior, while inflammation protects against tissue damage by mobilizing our immune system's physiological safeguarding and repair functions. Both of these kinds of protection—behavioral and immunological—make sense when danger or damage are actually present; but both pain and inflammation can become problematic when they chronically persist after the threat is no longer around.

Also, keep in mind that pain and nociception are not the same thing. Nociception is a nerve signal indicating potential mechanical, thermal, or chemical threat to tissues; pain is the experience the nervous system generates in response. Many times, when your clients' pain is prolonged, it can be because there is continued nociceptive input (for example, from ongoing mechanical or inflammatory irritation). But pain can persist even with little or no tissue damage; or, even when there is obvious damage or degeneration, there can be little or no pain experience at all.

And while your clients' pain may not be as related to physical tissue damage as we might have thought, there turns out to be a strong relationship between pain and expectations, fears, context, memory, and social influences. These psychosocial factors play a role in inflammation, as well as in pain. For example, high hostility scores have been correlated with increased inflammation, while openness (hostility's flip-side) has been associated with decreased inflammatory markers.[1] But the relationship between pain, inflammation, and psychosocial factors is nuanced and complex: while depressed people often have stronger inflammatory responses, turning *down* inflammation with immunosuppressive drugs can also trigger depressive mood changes. So even though pain, inflammation, and reactive emotions are deeply interconnected, they don't move in lockstep with one another. Instead, they could be thought of as different modes of physical, psychological, and behavioral adaptation that we use in varying ways in the face of perceived threat.



1 Musculoskeletal pain inflames both local tissues and the brain. In a recent study of sciatic pain (Loggia et al., 2015), inflammation wasn't limited to the locally painful tissues of the low back and leg. Glial cells (inset), which play a key role in both immunity and chronic pain, also respond with inflammatory activation (orange) in the corresponding regions of the brain's sensory and motor cortexes. This has implications for the use of both therapeutic sensation (such as produced by touch) and active client movement when working with musculoskeletal inflammation. *Motor cortex image adapted from artwork by Giovanni Rimasti, used by permission of Joseph E. Muscolino (www.learnmuscles.com). Glial cell micrograph adapted from H. Peluffo, L. Acarin, M. Faiz, B. Castellano, and B. Gonzalez, used under CCA 2.0G.*

THE NEUROIMMUNE SYSTEM

In inflammation, the nervous and immune systems work together. Tissue injury or irritation triggers the release of pro-inflammatory molecules into the surrounding interstitial environment (Image 1). Very quickly, the resulting "inflammatory soup" chemically excites and sensitizes nearby peripheral nerves, triggering behavior-altering soreness and pain (as well as initiating the cascade of the inflammatory progression, discussed in "Understanding Inflammation's Progression"). Pain *sensitization* is a key link between the immune and nervous systems: sensitization amplifies the actual nociceptive signals (not just the experience of pain), either peripherally at the tissue receptors themselves, or centrally, within the spinal cord and brain.

Interestingly, inflammation-related sensitization isn't limited to the directly injured areas. Inflammatory markers have also been found within related dorsal root ganglia along the spine (associated with processing chronic pain), neural pathways within the spinal cord, and perhaps most intriguingly, even in immune cells in parts of the brain associated with both sensation and movement of the painful area (glial cells in the sensorimotor cortex, Image 2).[2]

In the short term, this neural co-inflammation protectively inhibits muscular activity related to a painful (and potentially injured) area. But if prolonged, inflammatory sensitization can lead to numerous neurological and myofascial changes, including muscle size reduction (within days), reduced fatigue resistance, contraction speed change, and infiltration of fibrous and fatty tissues (within weeks or months, Image 3). Curiously, these inflammatory changes have been observed in muscles and fascia far from an injury, such as in spinal multifidus not adjacent to the site of a disk injury.[3]

In other words, inflammation doesn't just affect the fascia and connective tissues of the locally painful area. Over time, local pain can also inflame:

- Other myofascial structures far from the injury site;
- The neurons that connect the injured areas with the brain;
- And strangely, the corresponding area of your brain's body map.

As bad as this sounds, the body and brain have a tremendous capacity for adaptation. This means that all these things can also get better, and hands-on work has repeatedly been shown to help.[4]

There is a lot more to say about hands-on work's relevance to the inflammation/pain relationship before we get into actual techniques. Other important concepts include the ways local pain and inflammation are affected by systemic (whole-body) inflammation, the role of the vagus nerve and autonomics, stress, the controversies around ice, diet, and much more. For now, you can learn more about these topics in the "Working with Inflammation" webinar (available in ABMP's member library (www.abmp.com/members/continuing-education/online-learning/courses/webinar-working-inflammation, or via my website). In future editions of this column, I look forward to describing the specific ways that hands-on approaches can help with both inflammation and pain.

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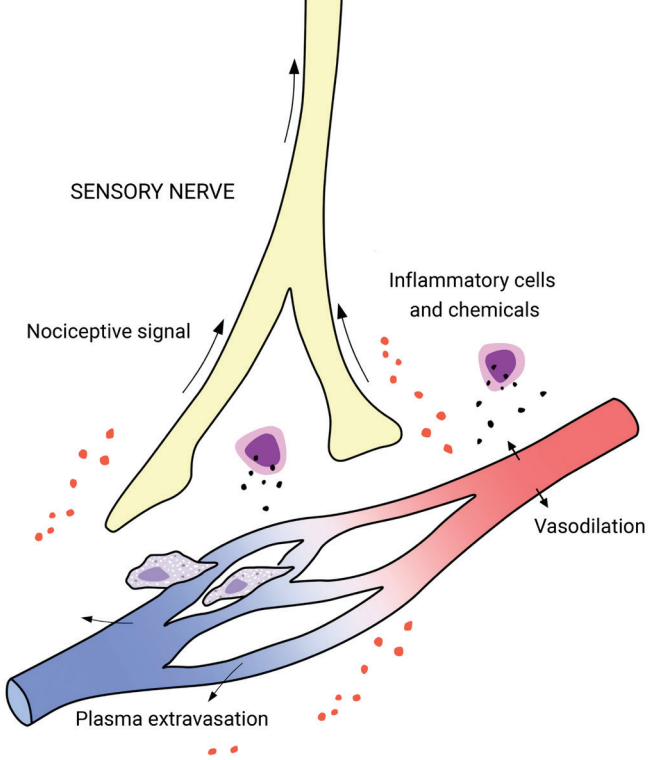
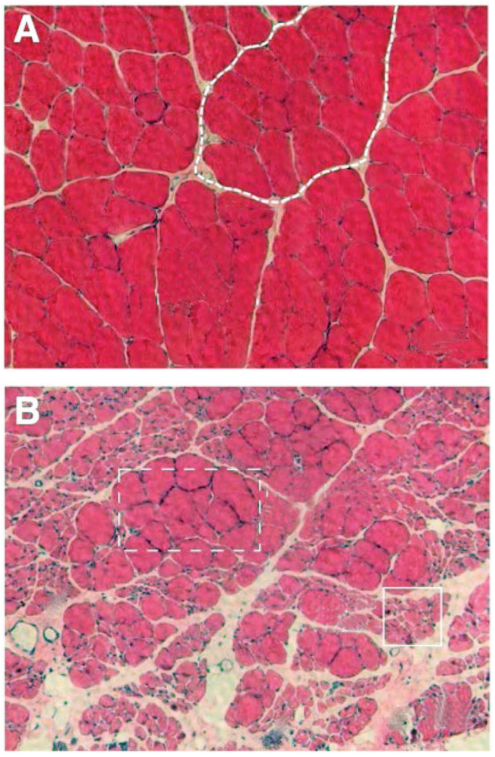


Tissue injury releases inflammatory molecules and cells into the surrounding interstitial environment. The resulting "inflammatory soup" chemically excites and sensitizes nearby sensory nerves, generating a nociceptive (pain-triggering) signal. *Adapted from Donaldson 2009; image courtesy Advanced-Trainings.com.*



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Over time, inflammation and pain can cause myofascial infiltration of fibrous and fatty tissues. A: Healthy skeletal muscle (rat tibialis anterior) with ~5 percent extracellular material. B: Fibrotic changes six months after inflammatory injury, showing extracellular increase to 20 percent of cross section. In another recent study (Bove et al., 2018), rats with repetitive strain injuries receiving modeled manual therapy (bilateral mobilization, skin rolling, and stretching) showed a reduction in nociceptor activity, neural inflammation, and fibrosis compared to unmassaged rats. *Image from Lieber et al., 2013, used under license from The American Physiological Society to Advanced-Trainings.com.*

"Pain & Inflammation"

Watch Til Luchau's technique videos and read his past articles in *Massage & Bodywork's* digital edition, available at www.massageandbodyworkdigital.com, www.abmp.com, and on Advanced-Trainings.com's YouTube channel. Watch Til's ABMP video playlist where all his videos have been compiled.

TIPS FOR WORKING WITH INFLAMMATION AND PAIN

Expand Your Tool Set

Inflammation typically needs a different hands-on approach than you'd take with tight muscles, undifferentiated fascia, or tender trigger points. Fortunately, there are many effective methods for working with local pain and inflammation, reflecting the fact that inflammation has many forms and responds differently from person to person. I'll be discussing specifics from our Advanced Myofascial Techniques approach in later articles, but there are many effective modalities available, and having a variety of options is helpful.

Find the Right "Dose"

Though skilled hands-on work can clearly help with inflammation-related pain, more is seldom better: if too deep, too long, or too frequent, massage or bodywork can further sensitize, rather than relieve. Remember that light pressures can be extremely effective for sensation normalization, calming, fluid-flow benefits, and more.

Use Movement

Active movement, both on the table and in clients' daily lives, can provide a powerful way to reduce both inflammation and pain. Movement helps by de-threatening and de-sensitizing, by "turning on" inflammation-inhibited muscles, by engaging and calming co-inflamed sensorimotor brain maps, and by enhancing interstitial circulation and inflammatory resolution. As with pressure, the movement "dose" is key; encourage your client to find the right movement type, intensity, duration, and frequency that's best for them. Don't hesitate to refer to a rehabilitative specialist, such as a physical therapist.

Don't Forget About Emotional Support

Because both pain and inflammation are linked to mood and outlook, your work will be made even more effective by your clients simply knowing you care. Listening and empathy can be potent tools.

Basic Heath and Self-Care

In addition to exercise, the health basics of good sleep, diet, managing stress, and addressing any medical issues are often the most powerful ways to help with inflammation. Receiving regular bodywork, in addition to the documented physiological effects on pain and inflammation, has intrinsic benefits as a self-care ritual, bookmarking a place for health in your client's life. **m&b**

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Notes

1. D. Girard et al., "Trait Hostility and Acute Inflammatory Responses to Stress in the Laboratory," *PLoS One* 11, no. 6 (2016); M. Luchetti et al., "Five-Factor Model Personality Traits and Inflammatory Markers: New Data and a Meta-Analysis," *Psychoneuroendocrinology* 50 (2014): 181–93.
2. M. L. Loggia et al., "Evidence for *Brain* Glial Activation in Chronic Pain Patients," *Brain* 138, no. 3 (March 2015): 604–15.
3. G. James et al., "Dysregulation of the Inflammatory Mediators in the Multifidus Muscle After Spontaneous Intervertebral Disc Degeneration SPARC-null Mice is Ameliorated by Physical Activity," *Spine* 43, no. 20 (2018): E1,184–94. doi:10.1097/BRS.0000000000002656.
4. G. M. Bove et al., "Manual Therapy Prevents Onset of Nociceptor Activity, Sensorimotor Dysfunction, and Neural Fibrosis Induced by a Volitional Repetitive Task, *Pain* (November 16, 2018): doi: 10.1097/j.pain.0000000000001443 [Epub ahead of print].

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