

Understanding Inflammation's Progression

By Til Luchau

Unless you've really been out of the loop, you've surely heard inflammation is to blame for almost every known malady. Or at least,

that's what the diet, supplement, and self-help ads claim. But, even if the health-care marketplace has overhyped and oversold inflammation in its relentless search for the next big thing, it's no exaggeration to say inflammation plays a key role in a wide range of conditions—from depression to tendon pain, fibromyalgia to cancer, and joint problems to aging itself.¹

For massage and manual therapists, inflammation can be puzzling with its many different types, manifestations, and mechanisms; its impossibly intricate physiology (Image 1); and the numerous (and often conflicting) views on how we should (or shouldn't) work with it.

For example, beginning massage therapists are usually taught that inflammatory conditions (such as acute injuries or swollen joints) are strict contraindications to direct massage. But many experienced practitioners will quietly confirm that careful, pressure-based work, sometimes directly with inflamed areas, can often be beneficial. (However, I'm not recommending you try this based solely on what you're reading here; don't risk worsening your client's pain and inflammation without specific training and supervision.)

There are also bigger questions about the nature and function of inflammation that might influence the way we think and

work. What roles do the brain and nervous system play? How do psychosocial factors (such as stress or depression) play in to inflammation? Could pain itself be a type of inflammatory response?

Let's start by reviewing some inflammation basics. In later columns, I'll dive deeper into specific questions, including how we, as manual therapists, can work with inflammation in helpful ways.



INFLAMMATION IS GOOD

Inflammation is simply the immune system's tissue protection and repair response. It can be triggered by injury, infection, or irritation. Inflammation is necessary and good: though it can be uncomfortable, without inflammation, the body can't protect or heal itself. Normally, inflammatory responses are self-limiting, and wind down when their protection and repair tasks are complete.



WHEN INFLAMMATION TURNS BAD

Problems happen when there is either:

- Hypoinflammation, such as when a compromised immune system can't fight an infection or heal an injury; or
- Hyperinflammation, which can happen when either immune reactions are out of proportion to the threat (as in allergic reactions or autoimmune disorders) or when normal inflammatory reactions don't resolve and become chronic.

Chronic or unresolved inflammation is implicated in an ever-lengthening list of conditions, including asthma, Alzheimer's, depression and mental illness, hypersensitivities, rheumatoid arthritis, metabolic syndrome, and certain cancers. Inflammation is not only a factor in nearly

all chronic diseases, but also in nearly all musculoskeletal complaints, including chronic pain.²

PROGRESSION OF INFLAMMATION

Healthy inflammatory responses progress over time, moving through acute, repair, and adaptation phases (Image 2). Though many of these functions happen simultaneously (for example, resolution processes begin even while inflammatory processes are in their early development), each step sets the stage for the next.



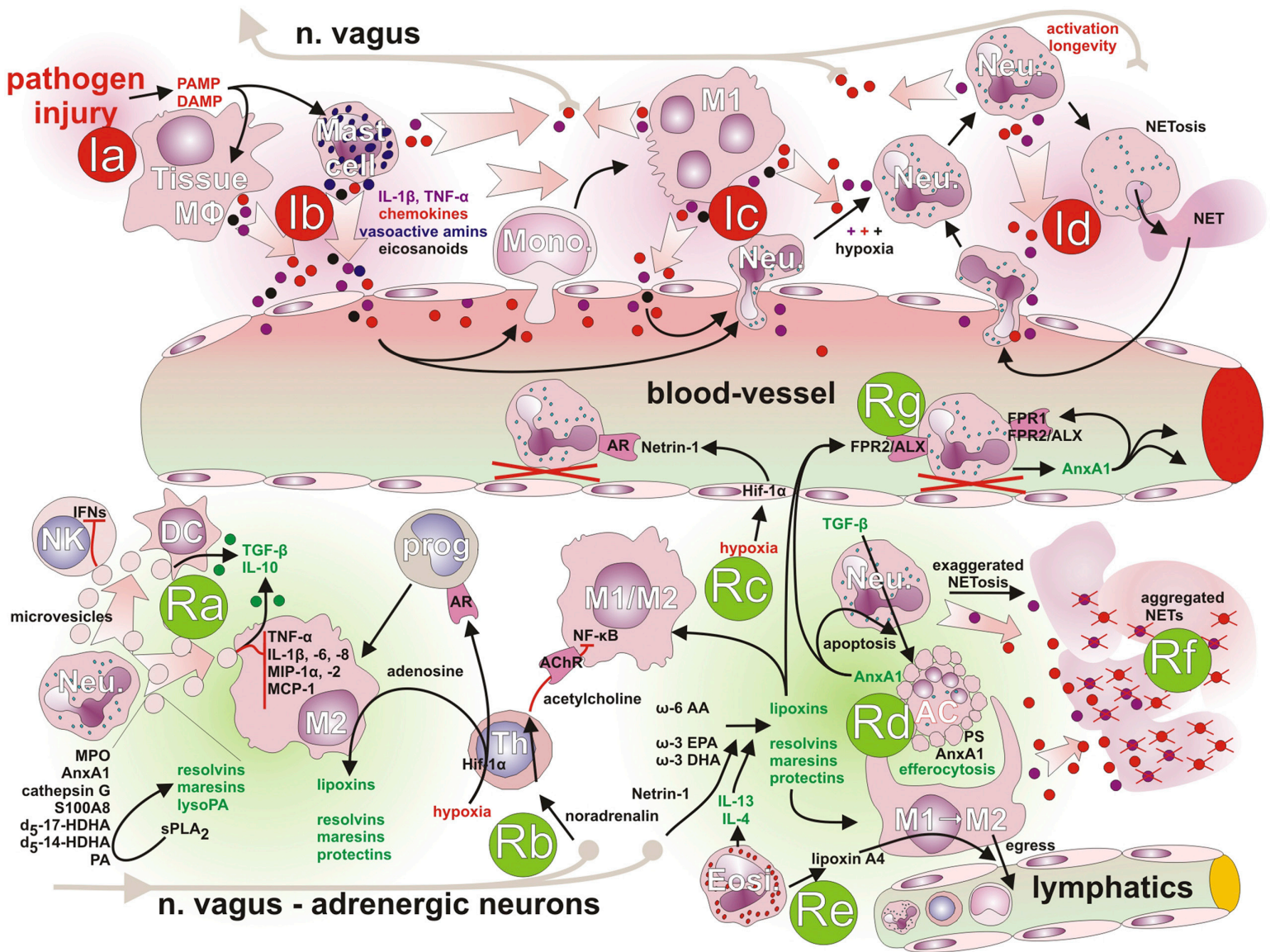
Acute Response

Within seconds or minutes after damage, danger, or pathogen detectors trigger an acute inflammatory response, proinflammatory histamine and kinins increase vascular and cell permeability, and the affected tissues swell with plasma and immune cells (such as leukocytes). Cytokines (signaling proteins) recruit roaming immune cells; local warmth and redness (Image 3) indicate that the active inflammatory responses are at work; and pain and tenderness (from nociceptor irritation by proinflammatories, pressure from swelling, and protective reactions) help guard against further strain or mechanical distress during this initial phase.



Repair

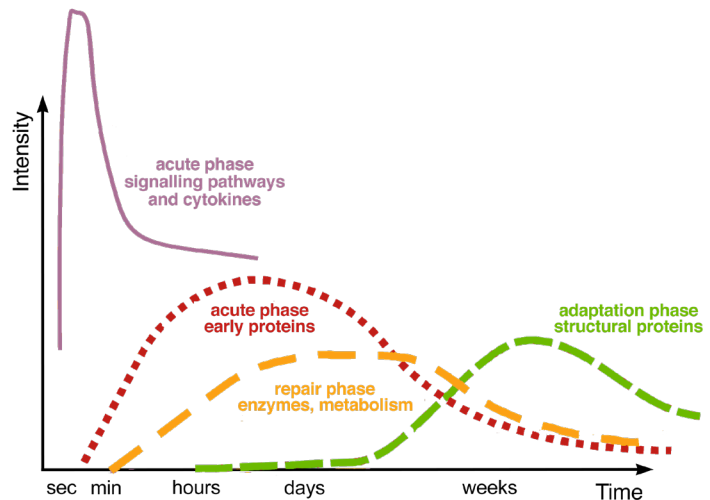
Within hours or days, specialized cells (such as mononuclear cells and macrophages) begin the process of degrading and removing damaged or diseased cells. Once unleashed, these destructive processes are kept in check by innate anti-inflammatory and pro-resolution mechanisms to minimize "bystander" damage to other tissues.³ Simultaneously, fibroblasts (which



1 There's a lot to know about inflammation, and this intimidating graphic will keep even hard-core geeks busy for a while. For the rest of us, the simplified version is that a pathogen or injury (upper left) initiates a sequence of inflammatory processes (upper half, in red) that release proinflammatory substances and move plasma and specialized cells from blood vessels into the tissues, where they are monitored and regulated by the peripheral nervous system (the vagus nerve in this instance, top). Meanwhile, anti-inflammatory and pro-resolution processes (lower half, in green) temper and balance the inflammatory progression. The vagus nerve (bottom) plays a direct role in controlling systemic inflammation via its release of anti-inflammatory compounds. As the inflammatory progression matures (from the left to right of the graphic), the resulting large proteins are processed by the lymphatic system and other mechanisms (Re, lower left). If you want even more detail about this illustration, go to <http://bit.ly/luchau-gallo2017>. Image: Gallo et al., 2017, used under CC BY-NC-ND 4.0.

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Normally, inflammation progresses through acute, intermediate (repair), and later (adaptation) stages, each with its own mechanisms and processes (see text). When any stage's progression is inhibited or prolonged, inflammation can become chronic. Resolution timelines vary tremendously, depending on the individual, the tissue, or the organ involved, etc., with full recovery in some cases taking months or years. After Steinacker et al., 1999, courtesy Advanced-Trainings.com.





3 Swelling, warmth, redness, and pain of acute inflammation. Though uncomfortable, these are signs of the healing processes at work.

arthritis; fibromyalgia; and “inflamm-aging,” the low-grade inflammation-like changes that accompany aging.

In prolonged inflammation:

- Fibroblasts, which usually have strong anti-inflammatory effects, can instead play an inflammatory role. Together with myofibroblasts, their unregulated activity over time can cause tissue fibrosis,

stiffening, and pathologic scarring.

- Treatment-resistant depression is more common, supporting the idea that some depression has inflammatory causes.⁵
- Pain can be persistent and difficult to treat. A recent study found that pain sufferers who didn't respond to other forms of therapy had higher levels of chronic inflammation, suggesting (though not confirming) that inflammation can directly trigger persistent pain responses.⁶ I'll talk more about the link between pain and inflammation in the next issue.

NEXT

There is a lot more to know about inflammation, of course. Later, I'll be writing about:

- Local and systemic inflammation
- Pain, inflammation, and the neuroimmune system
- Ways to use what we know about inflammation in our hands-on work. **m&b**

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Notes

1. E. Bullmore, *The Inflamed Mind: A Radical New Approach to Depression* (Australia: Simon & Schuster, 2018); F. R. Balkwill and A. Mantovani, “Cancer-Related Inflammation: Common Themes and Therapeutic Opportunities,” *Seminars in Cancer Biology* 22, no. 1 (February 2012): 33–40, <https://doi.org/10.1016/j.semcancer.2011.12.005>; B. Kennedy et al., “Geroscience: Linking Aging to Chronic Disease,” *Cell* 159, no. 4 (November, 2014): 709–13, <https://doi.org/10.1016/j.cell.2014.10.039>.
2. I. R. Klein-Wieringa et al., “Inflammatory Cells in Patients with Endstage Knee Osteoarthritis: A Comparison Between the Synovium and the Infrapatellar Fat Pad,” *Journal of Rheumatology* 43 (2016): 771–8.
3. C. N. Serhan and J. Savill, “Resolution of Inflammation: The Beginning Programs the End,” *Nature Immunology* 6 (2005): 1191–7, <https://doi.org/10.1038/ni1276>.
4. N. N. Burke et al., “Psychological Stress in Early Life as a Predisposing Factor for the Development of Chronic Pain: Clinical and Preclinical Evidence and Neurobiological Mechanisms,” *Journal of Neuroscience Research* 95, no. 6 (July, 2016): 1257–70, <https://doi.org/10.1002/jnr.23802>.
5. E. Bullmore, *The Inflamed Mind*.
6. J. Lasselin et al., “Low-Grade Inflammation May Moderate the Effect of Behavioral Treatment for Chronic Pain in Adults,” *Journal of Behavioral Medicine* 39, no. 5 (October 2016): 916–24, quoted in P. Ingraham, “Chronic, Subtle, Systemic Inflammation: A Possible Insidious Cause of Mysterious Chronic Pain,” last updated February 19, 2018, accessed September 2018, www.painscience.com/articles/inflammation-chronic-subtle-systemic.php.

6 Til Luchau is the author of *Advanced Myofascial Techniques* (Handspring Publishing, 2016), a Certified Advanced Rolfer, a practice coach, and a member of the Advanced-Trainings.com faculty, which offers online learning and in-person seminars throughout the United States and abroad. He invites questions or comments via info@advanced-trainings.com and Advanced-Trainings.com's Facebook page.

themselves have strong anti-inflammatory capacities) and other cells begin new tissue formation. Local warmth and redness diminish, as does pain—though pressure or stretch can still provoke protective tenderness as the new tissues form.

Adaptation

In the days, weeks, or months that follow, the proinflammatory process gradually matures and diminishes as the first-responder cells themselves become the target of degradation and cleanup via phagocytic, lymphatic, and other resolution mechanisms. Tissue strengthening continues, as fibroblasts cross-link collagen structures. Sensation normalizes and tenderness diminishes as the adapting brain gradually reevaluates and relinquishes the need for protective guarding.

CHRONIC INFLAMMATION

Problems arise when any of these stages fails to resolve, and the inflammation becomes ongoing or chronic. This can happen when the immune system is overwhelmed by pathogens or damage, or when its resilience is taxed by stress, inactivity, diet, lack of sleep, etc. Factors such as adverse childhood experiences⁴ or genetics can also predispose us to chronic inflammation. But the causes of many chronic inflammatory conditions are not fully understood. These include autoimmune disorders, such as rheumatoid



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