

Inflammation

The body's inflammatory process is designed to protect us from invading microbes, chemicals and allergens; physical trauma; and other threats both real and imagined. Inflammation occurs at the peak of the immune response when conditions bring in specialized immune cells to help repair and remove damaged tissues.

An acute (short-term) response is the basis of a successful immune system. However, when inflammatory stressors become chronic (long-term) and accumulate, trouble begins.

The immune system is divided into two types of responses: *natural* and *specific*. The *natural response* is an all-purpose first line of defense comprising cells that identify and attack a number of different invaders in a short time frame. They initiate inflammation by releasing toxic substances that damage and then consume the invader or damaged tissue.

The second component of the immune system is the *specific response*. The specific response recognizes a specific invader, like bacteria or an allergen, and then mounts a defense against it. This response relies on the first, natural response to fend off invaders or damaged cells.

Physical & Mental Stressors

Significant research has indicated that mental stressors provide as great a challenge to homeostasis as physical conditions like influenza or muscle tears.

The term *allostatic load* refers to the method by which the body adjusts to the combined effect of many physical and mental stressors. A high allostatic load is essentially an overload of those different stressors, creating what one researcher dubbed "a cascade of cause and effect".

HPA Axis. The connecting factor among physical, emotional and mental stressors occurs in an area of the brain known as the HPA axis. Consisting of the hypothalamus, pituitary gland and adrenal gland, the HPA axis serves as the body's emergency alert system that responds to a variety of stressors by releasing hormones, such as epinephrine and norepinephrine, which prepare different body systems for action. The HPA axis also releases growth hormone (GH) and cortisol; GH helps repair tissue and promotes growth, whereas cortisol fuels the body by maintaining proper glucose and fatty acid levels.

When faced with a lot of different stressors (i.e., a high allostatic load), the intricately tuned immune system can get caught in a stress hormone–inflammation loop. Hormones are released at higher than normal levels, leading to an increase in proinflammatory cytokines, which, in a vicious circle, then restimulates the HPA axis. Cortisol levels also increase and alter the immune/ inflammation system, resulting in higher levels of inflammatory factors in the body.

Physical conditions such as postural and joint malalignments play a major role in inflammatory conditions. Clients who spend long periods of time sitting each day are especially vulnerable when performing certain upright movements that can create excessive friction in joint complexes. Excessive shoulder internal rotation, thoracic flexion and hip external rotation are classic seated malalignment issues that result in pain or damage to areas that then become inflamed.

Excess weight and biochemical imbalances have also been linked to inflammation caused by excess intra-abdominal fat. Beyond a certain level, excessive intra-abdominal fat produces a cortisol response, which tells the body to store fat; this begins a never-ending feedback loop.

Advanced age is another contributing factor. As we age, interleukin levels increase dramatically, which plays a role in the development of many diseases of aging, including heart disease, osteoporosis, Alzheimer's disease and other cognitive impairment diseases.

The Link Between Inflammation & Disease

Over the past 10+ years, advances in measuring immune system responses have allowed scientists to measure specific hormones and chemical agents that contribute to the cascade effect of inflammation. Researchers have found links between different inflammatory conditions, such as the relationship of gum disease to atherosclerosis, depression to allergic reactions and chronic fatigue syndrome to the common cold.

Behavioral scientists have linked stress loads to asthma, skin and gastric disorders, and sexual dysfunction. Neuroscientists have shown that specialized Nerves close to the skin, airways, urogenital tract and gastrointestinal tract play a significant role in the inflammatory process in those areas. Other studies have found a link between repetitive-use injury in an isolated limb and tissue damage and inflammation in nonrelated limbs.

The Mental Element

A key element when dealing with chronic inflammation is creating a positive mental environment around the client's session. Emphasize that it is perfectly acceptable to give less of an effort during times of high stress. Sometimes the best session is restorative in nature.

VNS

Typically, doctors prescribe medications to combat inflammation. However, there's growing evidence that another way to combat inflammation is by engaging the Vagus Nerve and improving "vagal tone" (ability of the Vagus Nerve to respond). This can be achieved through daily habits such as Tai Chi, Ai Chi, Yoga, and Meditation—or in more extreme cases of inflammation, such as rheumatoid arthritis (RA)—by using an implanted device for Vagus Nerve Stimulation (VNS).

An international team of researchers conducted a clinical trial which demonstrates that stimulating the Vagus Nerve with a small implanted device significantly reduced inflammation and improved outcomes for patients with rheumatoid arthritis by inhibiting cytokine production. (Cytokines are substances, such as interferon, interleukin, and growth factors, which are secreted by certain cells of the immune system and have an effect on other cells that regulate immune and inflammatory responses.) The neuroscientists and immunology experts involved in this study used state-of-the-art technology to map the neural circuitry that regulates inflammation.

The July 2016 study, "Vagus Nerve Stimulation (VNS) Inhibits Cytokine Production and Attenuates Disease Severity in Rheumatoid Arthritis," was the first human study designed to reduce symptoms of rheumatoid arthritis by stimulating the Vagus Nerve with a small implanted device which triggered a chain reaction that reduced cytokine levels and inflammation. Although this study focused on rheumatoid arthritis, the trial's results may have implications for patients suffering from other inflammatory diseases, including Parkinson's, Crohn's, and Alzheimer's.

This was the first study to evaluate whether stimulating the inflammatory reflex directly with an implanted electronic device can treat RA in humans. They have previously shown that targeting the inflammatory reflex may reduce inflammation in animal models and in vitro models of RA . . . which might be relevant for other immune-mediated inflammatory diseases as well.

These findings suggest a new approach to fighting diseases that are currently treated with relatively expensive drugs that have a host of side effects. VNS gives healthcare providers a more effective way, potentially, to improve the lives of people suffering from chronic inflammatory diseases.

How to Activate the Vagus Nerve on Your Own

There are many ways to activate the Vagus Nerve and turn on the relaxation response. When you take a deep breath and relax and expand your diaphragm, your Vagus system is stimulated. With this breath you instantly turn on the parasympathetic nervous system, your cortisol levels are reduced, acetylcholine is produced, inflammatory response decreases, and your brain heals.

Activating the Vagus Nerve can stimulate stem cells to become cells that can repair and rebuild organs. Research has found that acetylcholine is a major brake on inflammation in the body. In other words, stimulating your Vagus Nerve sends acetylcholine throughout your body, not only relaxing the body but also turning down the fires of inflammation.

To practice deep diaphragmatic breathing, inhale through your nose with your tongue behind your top front teeth, and then exhale through your mouth.

Consider using the breath alone or use techniques that use diaphragmatic breathing – see if it helps your clients with inflammatory issues.

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American Dietetic Association (ADA). 2007. Position of the American Dietetic Association and Dietitians of Canada: Dietary fatty acids. *Journal of the American Dietetic Association*, 107 (24), 1599–1611.

Appleton, N. 2004. *Stopping Inflammation*. Garden City Park, NY: Square One.

Barr, A.E., Barbe, M.F., & Clark, B.D. 2004. Systemic inflammatory mediators contribute to widespread effects in work-related musculoskeletal disorders. *Exercise and Sport Sciences Reviews*, 32 (4), 135–42.

Borer, K.T. 2003. *Exercise Endocrinology*. Champaign, IL: Human Kinetics.

Butler, D.S. 2006. *The Sensitive Nervous System*. Adelaide, Australia: NOI Group.

Dement, W.C. 2000. *The Promise of Sleep*. New York: Dell.

Enwonwu, C.O., & Ritchie, C.S. 2007. Nutrition and inflammatory markers. *Journal of the American Dental Association*, 138 (1), 70–73.

Ivy, J., & Portman, R. 2004. *Nutrient Timing*. North Bergen, NJ: Basic Health.

Katz, A.E., et al. 2005. Zyflamend, a unique herbal preparation with nonselective COX inhibitory activity, induces apoptosis of prostate cancer cells that lack COX-2 expression. *Nutrition and Cancer*, 52 (2), 202–212.

Kreider, R. B., Fry, A.C., & O'Toole, M.L. 1998. *Overtraining in Sport*. Champaign, IL: Human Kinetics.

Ley, K. 2001. *Physiology of Inflammation*. New York: Oxford University Press.

Linden, W. 2004. *Stress Management: From Basic Science to Better Practice*. Thousand Oaks, CA: Sage.

Lipton, B. 2005. *The Biology of Belief*. Santa Rosa, CA: Mountain of Love.

Lovallo, W.R. 2004. *Stress and Health*. Thousand Oaks, CA: Sage.

McEwen, B.S., & Seeman, T. 1999. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896, 30–47.

Medinfo. 2008. *Non steroidal anti-inflammatory drugs*. www.medinfo.co.uk/drugs/nsaids.html; retrieved June 1, 2008.

Meggs, W.J. 2004. *The Inflammation Cure*. New York: McGraw-Hill.

Ratey, J.J. 2008. *Spark: The Revolutionary New Science of Exercise and the Brain*. New York: Little, Brown.

Sahrmann, S.A. 2001. *Diagnosis and Treatment of Movement Impairment Syndromes*. St. Louis: Mosby.

Sears, B. 2005. *The Anti-Inflammation Zone*. New York: Collins Living.

Seegerstrom, S.C., & Miller, G.E. 2004. Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130 (4), 601–30.

Smith, L.L. 2004. Tissue trauma: The underlying cause of overtraining syndrome? *Journal of Strength and Conditioning Research*, 18 (1), 185–93.

Talbott, S. 2002. *The Cortisol Connection*. Alameda, CA: Hunter House.

Tiidus, P.M. 2008. *Skeletal Muscle Damage and Repair*. Champaign, IL: Human Kinetics.