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Chronic Pain & Inflammation Protocol

Introduction

Pain is an inherent part of being human and serves a vital purpose beginning at (or even before) birth. It teaches us the boundaries of our bodies and the physical environment and keeps us safe in the physical world. But for many, pain becomes a chronic, daily experience that disrupts quality of life and the ability to live in comfort.

Why does pain become chronic for some but not others? The answer is complex and multifaceted. Typically, chronic pain follows an injury, multiple injuries, or another disease. Pain and inflammation resolve over time for most people, with a return to homeostasis and full function. But for some, pain shifts from a presenting symptom to a diagnosable condition with a distinct medical definition.[1]

Chronic inflammation often underpins chronic pain and is a powerful therapeutic target. Identifying and addressing sources of inflammation in the body is an essential part of a comprehensive treatment strategy. It's particularly important to assess chronic low-grade infections, whether viral, fungal, or bacterial, as well as poly-microbial. They can result in the ongoing production of proinflammatory mediators and the chronic activation of inflammatory pathways throughout the body.

See our Chronic Viruses, Candida, and Lyme protocols for more information.

Physiology/Diagnosis/Clinical Relevance

Pain is a subjective experience involving not only nociception but also emotional, cognitive, and social components.[2] Acute pain functions as an alarm system to protect us from tissue damage. It is primarily mediated by nociception – the process by which thermal, mechanical, or chemical stimuli activate a type of peripheral nerve fibers called nociceptors.[3,4]

Chronic pain, on the other hand, emerges from a sequence or multitude of various events, similar to many other illnesses. Even when there is a single event initiating the onset of chronic pain (e.g., injury), there are multiple factors that impact its duration, intensity, and effects (physical, psychological, social, and emotional).[5]

[1] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6676152/
[2] https://pubmed.ncbi.nlm.nih.gov/19837031/
[3] https://pubmed.ncbi.nlm.nih.gov/31551115/
[4] https://pubmed.ncbi.nlm.nih.gov/19837031/
[5] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6676152/

An important clinical risk factor for developing chronic pain is the presence of another site of acute or chronic pain within the body. With increasing severity and number of pain sites, the likelihood of chronic pain increases. Painful stimuli can alter brain chemistry, which predisposes individuals to develop chronic pain. This process can occur within days of exposure to ongoing painful stimuli and can last for up to a year after the pain has resolved. One of the best ways to reduce the chances of chronic pain development is to prevent acute pain from occurring and manage it well when it does occur.[6]

Pain and Inflammation

Pain and inflammation go hand-in-hand. Just as chronic inflammation plays a central role in almost all chronic diseases, it is also evident in chronic pain. Every pain syndrome has an inflammatory profile that consists of the inflammatory mediators present, and this profile may vary from one person to another or within the same person at different times.[7]

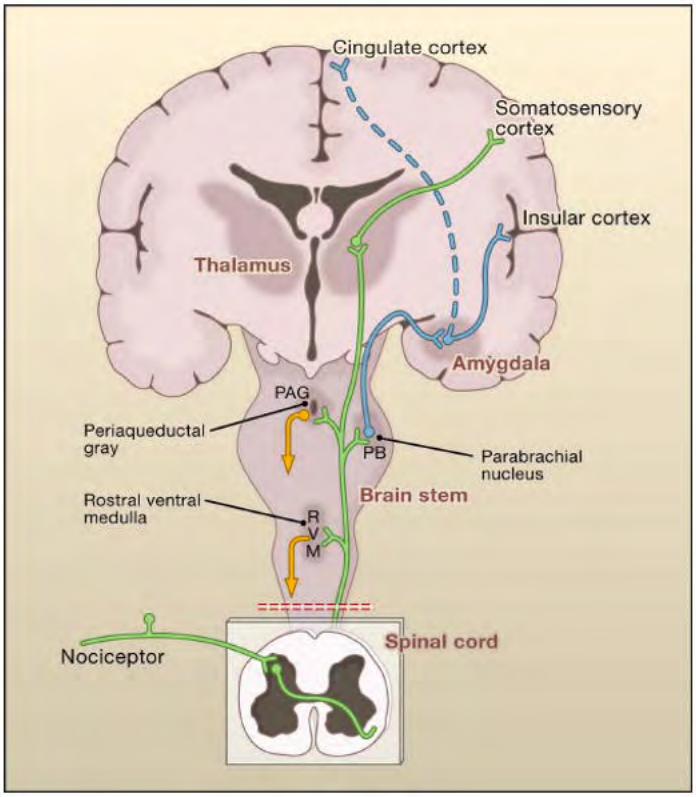

Figure 1. Anatomy of the pain pathway.
https://pubmed.ncbi.nlm.nih.gov/19837031/

Inflammation is unavoidable. It is part of the natural cycle that allows for immune response and healing. However, when inflammation is unchecked or in excess, it can have many detrimental effects, leading to localized and systemic injury as well as chronic disease. To withstand the dangers of inflammation, infection, and injury that we face daily, our immune systems must recognize danger signals and then induce innate and adaptive immune responses. Two such signals include pathogen-associated molecular patterns (PAMPs) and damage-associated molecular pattern molecules (DAMPs).

[6] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6676152/
[7] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2771434/

DAMPS are endogenous stress proteins or molecules released extracellularly and produced as a result of cell or tissue damage. DAMPs are capable of activating pattern recognition receptors (PRRs) to signal danger upon tissue damage and induce both inflammatory and repair processes. Excessive or persistent signaling mediated by such molecules may fuel a stress-inflammation amplification loop that underlies the pathogenesis of several chronic inflammatory disorders.[8] PAMPs are molecular motifs on the surface of microorganisms that are recognized by PRR-bearing cells of the innate immune system and epithelial cells to initiate an immune response against pathogens and ultimately lead to immunity.[9]

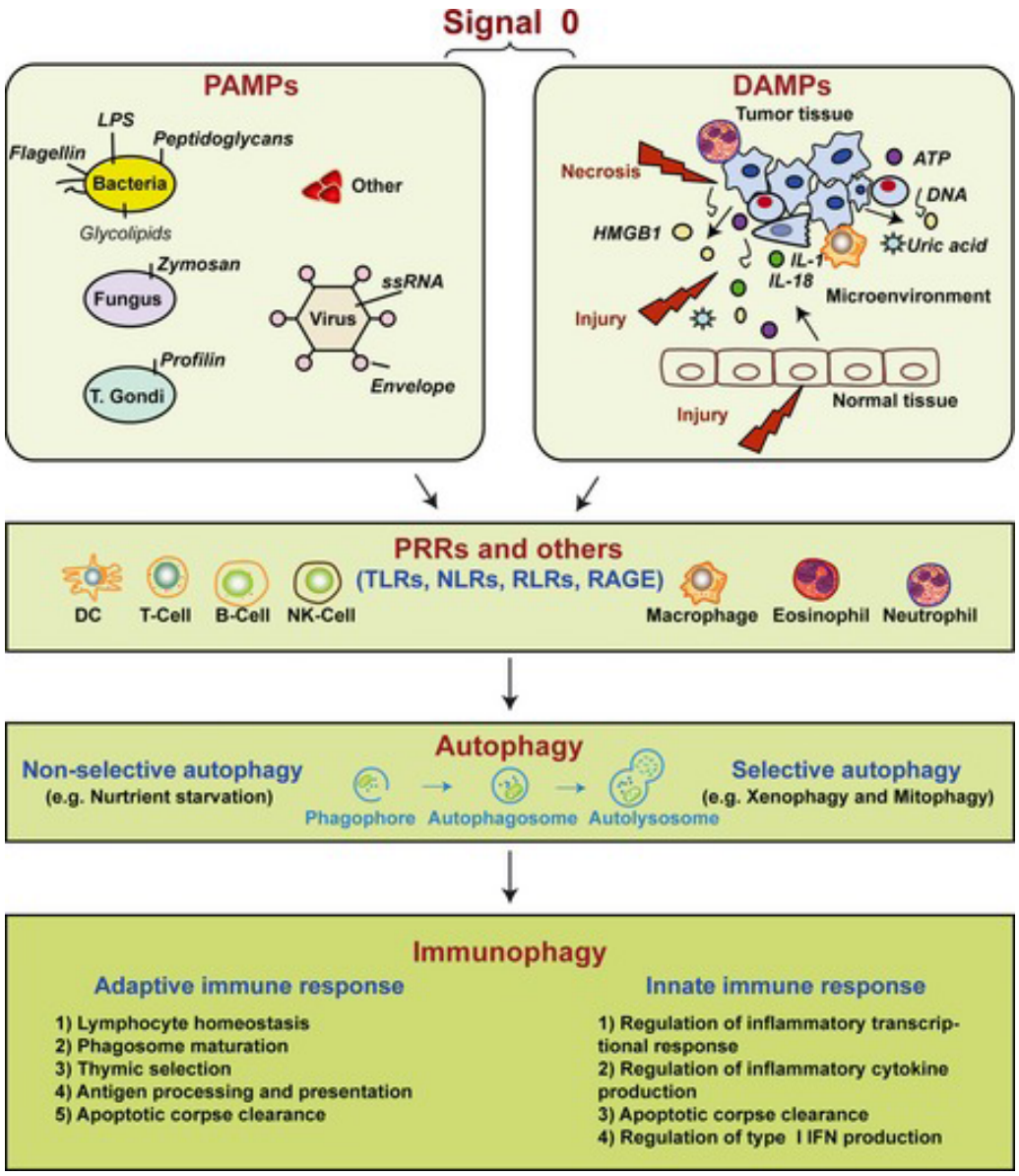

Figure 2. PAMPs and DAMPs and their role in autophagy and immunity.
https://pubmed.ncbi.nlm.nih.gov/22889221/

[8] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6224468/
[9] https://pubmed.ncbi.nlm.nih.gov/22889221/

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Risk Factors for Developing Chronic Pain[10]	
Demographic	• Advanced age • Gender – females report more chronic pain • Ethnicity – non-Caucasians report more pain • Low socio-economic status • Unemployed status • Occupational stressors
Lifestyle and behavior	• Smoking • Excessive alcohol • Sedentary lifestyle • Poor nutrition • Colder climates/lack of sunshine
Clinical	• Pain in multiple locations • Comorbid chronic illness/disease (e.g., cardiovascular, cancer, pulmonary, autoimmunity) • Mental health (e.g., depression, anxiety, PTSD, history of abuse) • Attitudes and beliefs about pain • Obesity • Sleep disorders • Genetic predisposition (pain tolerance and sensitivity are partially inherited) • Surgical/medical interventions (postoperative pain)

The Microbiome's Role in Inflammation and Chronic Pain

The microbiome plays a critical role in chronic inflammation and pain through these mechanisms:

- Immune regulation
- Production of microbial metabolites
- Activation of the gut-brain axis
- Modulation of neuroinflammation
- Regulation of intestinal permeability

Dysbiosis and disruption of the gut barrier can lead to increased intestinal permeability (leaky gut), allowing for translocation of microbial mediators and bacterial products (e.g., LPS) into systemic circulation, activating immune cells and promoting systemic inflammation. Chronic, low-grade inflammation – driven by dysbiosis and microbial-derived factors – has been implicated in various chronic pain conditions, including fibromyalgia, migraine, and neuropathic pain.[11]

[10] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6676152/
[11] https://pubmed.ncbi.nlm.nih.gov/27231050/

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Bacterial Components and Inflammation[12]		
Metabolite	**Location**	**Inflammation Receptors**
Lipopolysaccharides (LPS)	Outer membrane of gram-negative bacteria	Toll-like receptor (TLR-4) which can release TNF-α, IL-1β, and IL-6
Peptidoglycans	Component of bacterial cell walls, particularly in gram-positive bacteria	Nucleotide-binding oligomerization domain-containing protein (NOD1) and (NOD2)
Lipoteichoic acid (LTA)	Component found in cell walls of gram-positive bacteria	Stimulates production of proinflammatory cytokines
Bacterial lipoproteins	Lipid-anchored proteins found in cell membranes of gram-negative and gram-positive bacteria	TLR-2
Flagellin	Component of bacterial flagella	TLR-5
Bacterial DNA	Product of bacterial die-off	TLRs, stimulation of inflammasome complex
Bacterial toxins (exotoxins, endotoxins)	Released by bacteria	Direct damage to host cells and tissues

Gut microbes produce a wide array of metabolites that can be either beneficial and protective to the host or disruptive. Some examples include short-chain fatty acids (SCFAs), bile acids, and neurotransmitters, which can influence immune function, gut barrier integrity, and pain sensitivity. For example, SCFAs such as butyrate have anti-inflammatory properties and help maintain gut barrier function, while bile acids can activate immune cells and modulate inflammation.[13]

[12] https://pubmed.ncbi.nlm.nih.gov/33086688/
[13] https://pubmed.ncbi.nlm.nih.gov/27231050/

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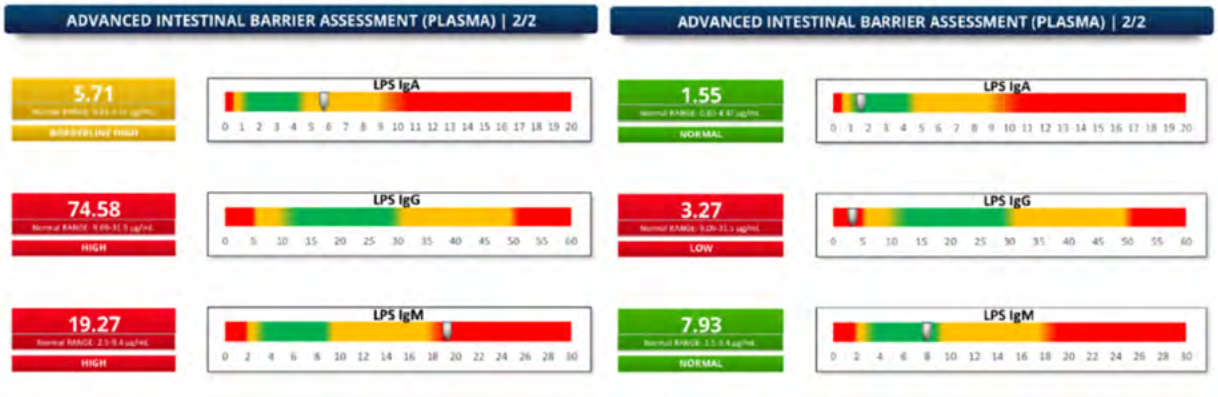
Bacterial Metabolites and Effects[14]		
Metabolites	**Beneficial Effects**	**Detrimental Effects**
SCFAs (butyrate, propionate, acetate)	• Anti-inflammatory properties • Protect gut barrier function • Improve memory and learning; neuroprotective	• Propionate can be inflammatory, leading to damaged mitochondrial DNA, and influencing neurotoxicity
Bile acids (secondary bile acids)	• Antimicrobial and anti-inflammatory • Maintain microbial balance • Support lipid metabolism	• Elevated levels can increase inflammation, cytotoxicity, and gut permeability
Neurotransmitters (serotonin, dopamine, GABA)	• Support mood, sleep, metabolism, and cognition • Protection from neurodegeneration	• Elevated or decreased levels can affect mood, cognition, metabolism, etc.
Amino acids and vitamins	• Support nutritional needs for producing neurotransmitters and hormones, as well as support cellular metabolic pathways	• None known
Trimethylamine N-Oxide (TMAO)	• None known	• Promotes atherosclerosis and is associated with many cardiovascular and metabolic diseases • Increases cognitive impairment through oxidative stress • Disrupts blood-brain barrier
Indole	• None known	• Associated with impaired motor function, depression, and anxiety
Quinolinate	• None known	• Excitotoxic properties • Neurotoxic • Promotes neurodegeneration
Kynurenine	• NMDA receptor antagonist that can reduce neurotoxic effects	• Elevated levels linked to cognitive impairments

[14] https://pubmed.ncbi.nlm.nih.gov/36399245/

Pilot research shows that Biocidin®, combined with G.I. Detox®+, can profoundly affect LPS production and subsequent immune reactivity. The test results below are taken from a participant after 12 weeks of use.



The microbiome is foundational for addressing inflammation and chronic pain due to its pivotal role in regulating the immune system and modulating inflammatory responses. The gut microbiota, consisting of trillions of microorganisms, influences various physiological processes, including digestion, nutrient absorption, and immune function. Imbalances in the gut microbiome can lead to increased intestinal permeability, allowing harmful substances to leak into the bloodstream and trigger inflammatory pathways. This chronic low-grade inflammation contributes to the development and exacerbation of chronic pain conditions. By restoring balance to the microbiome through dietary interventions, probiotics, and lifestyle modifications, practitioners can help mitigate inflammation, alleviate pain, and promote overall health and well-being.

Clinical Pearl #1: Chronic Pain is Connected to Oral Health

Poor oral health and oral dysbiosis are associated with an increased risk of systemic inflammation.[15] Systemic inflammation can increase the risk of chronic pain; therefore, an assessment of oral health and oral dysbiosis should be considered in every patient presenting with chronic pain. Include a basic oral exam and questionnaire as part of your physical exam and address oral dysbiosis.

See our Oral Health protocol for more information.

Clinical Pearl #2: Consider Inflammatory Foods and Food Sensitivities

Dietary factors can be direct or indirect causes of inflammation and are primarily important in chronic pain conditions. Functional abdominal pain disorders have been associated with food intolerance/malabsorption. Similarly, food sensitivities can activate an immune response, increasing the production of inflammatory cytokines, which may contribute to chronic pain in the body. Food additives can further act as inflammatory mediators.

Food reactivity can be identified using an elimination diet and symptom tracking or food sensitivity testing. Following an elimination diet for a period of time will often lead to an improvement in symptoms, and foods may eventually and gradually be reintroduced. The most common food sensitivities are lactose/dairy, gluten, FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols), soy, corn, eggs, coffee, yeast, wine, food colorings, preservatives, sulfites, and fructose.

[15] https://pubmed.ncbi.nlm.nih.gov/37623290/

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Lifestyle Recommendations

Support your treatment with simple yet effective lifestyle recommendations.

- Check out the tips listed in the Bioclear® Microbiome Detox Lifestyle Guide.
- Adopt a low inflammation diet (Modified Paleo, Mediterranean, etc.) that includes plenty of non-starchy vegetables.
- Increase fiber intake.
- Increase exercise to support the movement of the intestines.

Scan here to go to the Bioclear® Microbiome Detox Program Lifestyle Guide

Therapeutic Plan Suggestions

Bioclear® Microbiome Detoxification Program		
Choose Biocidin® Liquid, Capsules, or LSF to use in your program.		
Biocidin® Liquid Titrate to 10 drops 2x/day per booklet instructions	Biocidin® Capsules Titrate to 2 capsules 2x/day per booklet instructions	Biocidin® LSF Titrate to 2 pumps 2x/day per booklet instructions
G.I. Detox®+	2 capsules at bedtime. 1 hour away from food, supplements, and medications. Temporarily increase dose to 2 capsules 2-3x/day if Herxheimer reaction observed/worsens.	
Proflora™ 4R	1 capsule any time of day	
ADDITIONAL SUPPORT (sold separately)		
G.I. InnerCalm®	1 stick pack mixed in water, 1-2 times daily, taken any time	
Dentalcidin®	Brush 1-3x/day	
Dentalcidin® LS	1-2 pumps 3x/day, swish for 1-2 minutes and spit after brushing	
Dentalflora®	Dissolve 1 tablet in mouth 1-2x daily, at least 30 minutes away from other oral care, food, or drinks	

Additional Therapeutics/Supplements

- Turmeric/Curcumin, Ginger, Devil's claw, Cat's claw, St. John's wort, Meadowsweet
- Systemic enzymes, papain, bromelain
- Pro-resolving mediators (SPMs)

Questions?

For clinical questions, email clinical@biocidin.com or call 800-775-4140, x3.

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*These statements have not been evaluated by the Food and Drug Administration. These products are not intended to diagnose, treat, cure, or prevent any disease.