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Scientists Uncover a New Way to Pinpoint Inflammation in the Body

Analysis by Dr. Joseph Mercola | September 24, 2026

STORY AT-A-GLANCE

- Chronic inflammation is associated with diseases like heart disease, Alzheimer's, cancer, and autoimmune conditions. However, standard tests only measure generalized inflammation and do not pinpoint the exact tissues affected
- Researchers at Case Western Reserve University have developed a method to track inflammation at its source using antibodies that detect molecular markers left by reactive oxygen species (ROS)
- Unlike traditional markers like C-reactive protein (CRP), this new approach could allow doctors to identify specific organs under inflammatory stress, which the researchers suggest could support earlier and more precise diagnoses
- Emerging research challenges the idea that chronic inflammation is just unresolved acute inflammation. A hypothesis paper proposes that it results from a lack of anti-inflammatory mediators, not just excessive inflammatory signaling
- Key drivers of chronic inflammation include excess linoleic acid (LA), endocrine-disrupting chemicals, endotoxins, and electromagnetic fields (EMFs)

Many chronic diseases, from heart disease and Alzheimer's to cancer, share one thing in common — chronic inflammation. But while inflammation is thought to play a key role in disease progression, pinpointing where it occurs in the body has always been a challenge. Standard blood tests measure broad markers like C-reactive protein (CRP), but they fail to identify specific tissues or organs affected by inflammation.[1]

A study published in the journal Proceedings of the National Academy of Sciences[2] (PNAS) has found a way to detect inflammation in specific areas using antibodies. This innovation could open the door to highly targeted diagnostic tests, allowing earlier detection of inflammatory diseases. These findings come from laboratory work in cell, animal, and human tissue samples, but the approach has not yet been validated as a clinical blood test.

How Does Inflammation Work?

Inflammation is how your body responds to threats, whether from injury or infection. It mobilizes immune cells, increases blood flow, and activates signaling molecules to contain damage and promote healing. But for this process to work properly, the immune system needs to maintain a balance between inflammation and resolution. When this stability is disrupted, inflammation becomes chronic, leading to long-term health problems.[3]

- **Your body's first line of defense —** When you get injured or are exposed to harmful bacteria, your immune system jumps into action. Specialized immune cells called macrophages act like sentries, constantly patrolling your body. They detect distress signals from damaged cells or foreign invaders and respond quickly to keep you safe.[4]

- **Cellular "alarms" trigger an immune response —** When your immune system detects a threat, it sounds the alarm by releasing signaling molecules called cytokines and chemokines. These act like emergency alerts, calling in more immune cells to help. Cytokines also control how strong and long-lasting the inflammation is, making sure your body responds appropriately.[5]

- **Increased blood flow fuels the battle —** When your immune system detects a threat, your blood vessels widen to increase blood flow to the area. This delivers immune cells, including neutrophils, which attack invaders and clear out damaged tissue. Along with oxygen and nutrients, this surge helps fight infection and start the healing process, causing redness, swelling, and warmth, the classic signs of inflammation.[6]

- **Immune cells attack and clear the threat —** When your body detects a threat, neutrophils are the first to arrive. They quickly engulf harmful particles and release antimicrobial substances to kill invaders. If the threat persists, macrophages take over, clearing pathogens and cellular

debris, while T-cells coordinate the immune response and destroy infected cells to prevent further damage.[7]

- **Shutting down inflammation for recovery —** Once the infection or injury is under control, your immune system releases anti-inflammatory molecules to slow down the response. This helps limit unnecessary damage to healthy tissue and helps your body transition from defense to repair.[8,9]

To learn more about the key differences between acute and chronic inflammation, early warning signs, and how to restore balance, check out "**Warning Signs of Acute and Chronic Inflammation in the Body**."

Will This Approach Change How Inflammation Is Diagnosed?

The featured study, conducted by researchers at Case Western Reserve University,[10] highlights a new antibody-based method to track traces of inflammation, pinpointing it at its source. This approach could offer a more precise way to detect inflammation-driven conditions.[11]

- **A unique chemical reaction enables detection —** Researchers found that when reactive oxygen species (ROS) — highly reactive molecules that damage DNA, proteins, and lipids — interact with certain compounds, they create a distinct chemical reaction. This reaction leaves a detectable marker, allowing antibodies to track inflammation at its source.[12]

- **The role of reactive oxygen species —** During inflammation, your immune cells release ROS to kill bacteria and fight infections. These molecules also come from environmental sources like UV light, pollution, radiation, and smoking. While ROS helps protect you, too much damages your cells and tissues and contributes to disease.[13]

- **How ROS interact with fats in your cells —** Researchers found that ROS reacts with linoleic acid (LA), a type of omega-6 fat in all cell membranes. This interaction creates compounds called epoxyketooctadecenoic acids (EKODEs), leaving behind markers that can be used to detect inflammation.[14]

- **A breakthrough in biomarker detection —** The study also found that EKODEs form a unique bond with cysteine, an amino acid. These compounds build up in tissues under oxidative stress, including the brain, heart, and liver. By raising antibodies against the EKODE-cysteine

adduct in rabbits, the team detected these markers in human cells and brain tissue as well as in mouse tissue, paving the way for more precise inflammation detection.[15]

- **Linking EKODEs to disease —** The researchers aim to map EKODEs to specific diseases by identifying which organs and conditions these markers are most strongly associated with. One area of focus is the eye, where the team plans to examine EKODEs produced in response to age-related macular degeneration and diabetic retinopathy, both of which can lead to vision loss.[16]

The authors believe these findings could pave the way for a simple blood test that detects inflammation in specific organs. "This research opens up an amazing number of pathways for future studies," said Greg Tochtrop, Ph.D., professor of chemistry at Case Western Reserve and senior author of the study. "It will lead directly to better understanding inflammation and detecting diseases, as well as to discovering new drugs."[17]

How Lipid Peroxidation Products Fit into the Bigger Picture of Inflammation

While the groundbreaking inflammation detection method highlighted in this article offers new insight into chronic disease, understanding the molecular players driving inflammation will empower you to take control of your health at a deeper level. As biochemical reviews describe,[18] EKODEs are just one of several lipid peroxidation products formed when polyunsaturated fats (PUFs), particularly LA, oxidize.

Two others that have been studied extensively are 4-hydroxy-2-nonenal (4-HNE) and malondialdehyde (MDA), which also exert wide-ranging effects on your biological systems.[19] These are signaling molecules that appear to protect or damage depending on how they interact in your cells. Their activity may help explain why inflammation can spiral out of control and why spotting them matters:

- **EKODEs are double-edged signalers —** EKODEs (epoxy-keto fats) feature both an epoxide ring and a keto group, giving them distinct bioactive properties. At low concentrations, EKODEs activate pathways that influence antioxidant defenses, inflammation, and

metabolism. In animal studies, oxidized linoleic acid products have also been shown to influence pain signaling, though this work is early and has not been established for EKODEs specifically.[20]

However, when levels spike during oxidative stress, like in chronic disease, they appear to become damaging — modifying proteins, disrupting membranes, and amplifying cellular dysfunction. In this model they act as mediators in inflammation, tipping the balance between adaptation and damage.

- **4-HNE is a potent messenger of stress —** 4-HNE is a highly reactive aldehyde that activates Nrf2 to boost detoxification enzymes. It can also increase cytokines, your body's inflammatory alarm system. At low levels, 4-HNE acts as a signaling molecule that promotes cellular adaptation.

  At high levels, 4-HNE shifts to a destructive role, binding to mitochondrial proteins, and promoting apoptosis in laboratory studies. Reviews of lipid peroxidation report elevated 4-HNE in conditions ranging from Alzheimer's[21] to atherosclerosis, though these are associations rather than demonstrated causes.[22]

  Its electrophilic nature lets it "tag" proteins on cysteine or lysine residues, altering their function in ways that either support survival or accelerate cellular dysfunction. In inflammation, 4-HNE appears to shape both the magnitude and trajectory of the response.

- **MDA is the silent cross-linker —** Malondialdehyde, the dialdehyde cousin, plays a less overt — but equally damaging — role in oxidative stress. It excels at cross-linking proteins and DNA, leaving a trail of damage. It's less a direct signaler and more a saboteur, quietly amplifying inflammation by compromising your cellular machinery.

  Although MDA influences stress pathways indirectly, its real danger lies in chronic accumulation — elevated MDA has been observed in diabetes, aging, and cancer, where it is thought to erode structural integrity. MDA's presence is treated as a marker of unresolved long-term oxidative stress.

- **The interplay between these three compounds —** These three lipid-derived compounds don't act independently. During oxidative stress, they interact within the same biochemical environment, competing for nucleophilic targets — cysteines and lysines on proteins — and

diluting each other's effects. For example, if 4-HNE tags a key enzyme first, it blocks subsequent interactions by EKODEs.

In high-stress states (say, a heart attack or chronic infection), they are proposed to act together and overwhelm antioxidant defenses like glutathione — 4-HNE compromising mitochondria, MDA cross-linking DNA, and EKODEs destabilizing membranes.

At lower levels, EKODEs have been shown to activate antioxidant-response pathways, which may curb 4-HNE's and MDA's effects and offer a protective counterbalance. Note that this interplay is described from laboratory work and has not been mapped out in people.

- **Why this matters to you —** The behavior of these compounds serves not just as a scientific explanation but as a roadmap. These lipid peroxidation products are measurable (via assays like TBARS for MDA or mass spectrometry for 4-HNE), and their levels reflect your oxidative load.

  The diagnostic approach discussed in this article could, in theory, spotlight their activity indirectly. Think of it as a window into their influence on your inflammatory response. That said, talk to your health care provider about whether this testing is appropriate for you should you be interested in it.

  It's also worth noting that what presents as oxidative stress often traces back to a deeper problem in how your cells produce energy — a reductive shift in the mitochondria that leaves reactive oxygen species with nowhere to go.

  Seen this way, lowering lipid peroxidation isn't just about neutralizing damage; it's about restoring the cellular energy production that keeps these products in check in the first place.

The good news is that these lipid peroxidation products are factors you can influence. Understanding how they interact helps explain where inflammation begins. Research in this area points toward familiar levers: Dietary patterns that support the body's own antioxidant response, and reducing omega-6 load by removing vegetable oils from the diet.

Is Our Understanding of Chronic Inflammation Flawed?

While researchers at Case Western Reserve University[23] have identified biomarkers that reveal inflammation in specific organs, a new hypothesis-and-theory paper published in Frontiers in Immunology[24] — a single-author framework proposal rather than new experimental data — challenges the long-held belief that chronic inflammation is simply unresolved acute inflammation. Instead, it suggests that chronic disease stems from a loss of anti-inflammatory mediators, not just excessive inflammatory signaling.

- **Inflammation versus unalamation —** Experts have long believed that chronic inflammation happens because the immune system stays too active. However, the author proposes that it results from a loss of anti-inflammatory mediators, disrupting a balance called unalamation. This challenges the conventional idea that suppressing inflammation is the ideal treatment.[25]

- **The role of unalamation in health and disease —** Your body uses inflammatory mediators like prostaglandins and cytokines to fight infections, heal wounds, and maintain tissues. Unalamation is the balance between these inflammatory and anti-inflammatory signals. When anti-inflammatory mediators drop too low, inflammation persists, even without an injury or infection, leading to long-term health problems.[26]

- **Why standard anti-inflammatory drugs fall short —** NSAIDs and other inflammation-blocking drugs help with short-term inflammation but do not, in this framework, correct chronic conditions like arthritis, heart disease, or neurodegenerative disorders. This is because they block inflammatory signals without restoring the missing anti-inflammatory ones, leaving the underlying imbalance unaddressed.[27]

- **Cancer and the unalamation connection —** The author also revisits the idea that chronic inflammation drives cancer. Tumors contain both inflammatory and anti-inflammatory signals, which suggests that they grow in a state of heightened unalamation, not just inflammation.

  This may explain why blocking inflammation alone doesn't stop cancer and, the author proposes, why unalamation may be worth investigating as a therapeutic direction. Again, keep in mind that this is a hypothesis, not a demonstrated treatment approach.[28]

This emerging research reshapes how we approach inflammation-related diseases. Instead of merely shutting down inflammatory pathways, future therapies may need to restore the body's natural balance of pro- and anti-inflammatory mediators, offering a more sustainable path to

healing.

What Are the 'Four E's' That Drive Inflammation?

To break free from inflammation, it would be wise to address its root causes, not just treat symptoms. Today's diets, daily habits, and environmental exposures create a constant inflammatory burden and overwhelm your body's ability to maintain balance. I believe there are four primary drivers of inflammation — what I call the "Four E's":

1. **Excess LA —** An omega-6 polyunsaturated fat (PUFA), LA is found abundantly in vegetable oils and ultraprocessed foods. LA is essential in small amounts and required for mitochondrial function — the problem is quantity, not the fat itself.

   In excess, LA is one of the most harmful components of the Western diet, and has been reported to negatively affect your metabolic health and gut microbiome,[29] which are two of the most important factors for maintaining proper inflammatory responses and overall health. I recommend keeping LA intake between 2 and 5 grams per day.

2. **Electromagnetic fields (EMFs) —** EMFs are generated by everyday electronic devices such as cell phones, Wi-Fi routers, and microwaves, causing unseen harm to your health. In a mechanism laid out in Pall's review of EMF effects,[30] EMFs activate voltage-gated calcium channel (VGCC) receptors in your cells, leading to an influx of calcium ions. This surge in calcium is thought to catalyze the production of peroxynitrite, a potent oxidant that contributes to cellular stress and inflammation.

3. **Endocrine-disrupting chemicals (EDCs) —** Exposure to EDCs significantly impacts your health by over-activating estrogen receptors in your body. Microplastics are alarmingly prevalent in our environment, with research suggesting that the average person ingests the equivalent of a credit card's weight in plastic each week.[31]

   Plastic is often laden with harmful substances like phthalates and bisphenol A (BPA), both of which bind to estrogen receptors and disrupt normal hormonal functions. Elevated estrogen has been shown to raise intracellular calcium levels in cell studies,[32] which is thought to drive the generation of peroxynitrite — a mechanism proposed to exacerbate inflammation and contribute to various chronic health conditions.

4. **Endotoxins —** Consuming ultraprocessed foods loaded with vegetable oils and high-fructose corn syrup (HFCS), as well as exposure to EDCs, disrupts your gut microbiome, increasing endotoxin production and systemic inflammation. Endotoxins are toxic substances released from the cell walls of certain bacteria, particularly gram-negative bacteria.

   These bacteria are facultative anaerobes, meaning they thrive in both aerobic (with oxygen) and anaerobic (without oxygen) environments. This adaptability allows them to colonize various areas in the body, including the gut, where they contribute to inflammation.

   When endotoxins from these bacteria enter the bloodstream — often due to a compromised gut barrier (leaky gut) — they trigger a strong inflammatory response known as endotoxemia. In review articles, this condition has been linked to various health issues, including metabolic syndrome and autoimmune diseases.[33,34,35]

Reducing these four inflammatory stressors lowers the burden on your system and supports the conditions your body needs to restore its natural balance of pro- and anti-inflammatory mediators. Learn how to address these root causes of inflammation in "Cellular Health Revolution — Unveiling Hidden Threats and Empowering Solutions."

Frequently Asked Questions (FAQs) About Inflammation and Chronic Disease

Q: Why is it hard to detect the exact location of inflammation?

A: Standard blood tests only measure broad inflammatory markers like CRP, which indicate inflammation but don't reveal where it's happening in the body. The new antibody-based test could change that by identifying inflammation in specific organs.

Q: What is unalamation, and why does it matter?

A: Unalamation refers to the functional balance between inflammatory and anti-inflammatory mediators. Chronic inflammation may not be caused by too much inflammation but rather by too little anti-inflammatory activity.

Q: Why do NSAIDs fail to treat chronic inflammation?

A: NSAIDs and other anti-inflammatory drugs block inflammatory mediators but do nothing to restore missing anti-inflammatory signals. Without correcting the underlying imbalance, inflammation persists.

Q: Is chronic inflammation really the cause of cancer?

A: One Frontiers in Immunology hypothesis paper proposes that tumors exist in a state of heightened unalamation, not just chronic inflammation. This means both inflammatory and anti-inflammatory mediators are present in excess, creating an environment the author argues may favor tumor growth. This is a proposed framework, not an established finding.

Q: What are the best ways to restore balance and fight chronic disease?

A: Researchers working in this area propose that, rather than blocking inflammation alone, future therapies may need to replenish anti-inflammatory mediators, reduce oxidative stress and support mitochondrial function. These are research directions rather than established treatments.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified health care provider before making changes to your health regimen.

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