

Thymus Hormone Linked to Lower Age-Related Inflammation

Analysis by [Dr. Joseph Mercola](#)

October 14, 2026

STORY AT-A-GLANCE

- › Researchers at the Keck School of Medicine of the University of Southern California (USC) identified thymulin, a hormone produced by the thymus, as a regulator of inflammatory immune cells in mice
- › A larger share of myeloid cells, the fast-acting arm of your immune system, produced inflammatory cytokines in aged mice and in healthy human donors aged 60 to 87 than in younger ones
- › Giving thymulin to aged mice was associated with fewer inflammatory cytokine-producing myeloid cells, slower breast tumor growth, and longer survival, while young mice showed no meaningful change
- › A separate review reports that inflammatory cytokines sped up thymus shrinkage in animal studies; zinc status, vitamin D, sex hormones, and chronic stress may also influence how fast the gland declines
- › Practical steps that may support immune adaptability as you age include cutting seed oils, eating zinc- and vitamin A-rich whole foods, prioritizing deep sleep and morning sunlight, staying active, and avoiding tobacco and excess alcohol

Your **thymus** sits behind your breastbone, between your lungs, and it is about the size of a dried apricot. It does its most visible work in childhood, training the T cells that let your immune system recognize infections and abnormal cells. As you age, the thymus

gradually shrinks, and for decades scientists assumed its usefulness shrank along with it. That assumption is now getting a second look.¹

Researchers at the Keck School of Medicine of the University of Southern California (USC)² report that beyond producing T cells, the thymus releases a hormone called thymulin, which appears to regulate cytokines, the signaling proteins your body uses to mount a response against infection. Those same proteins drive inflammation when they stay switched on.³

Thymulin activity fades with age, and the researchers propose that its decline contributes to a pattern known as inflammaging. Inflammaging has been linked to cancer, heart disease, metabolic conditions, and neurodegenerative disease, yet scientists have struggled to explain what keeps the process going. These findings in mice and in donated human cells may offer one piece of that puzzle.⁴

Thymulin Emerged as a Regulator of Inflammatory Immune Cells in Aged Mice

The featured preclinical study, published in the journal Nature Communications, was conducted on mice, cultured cells, and blood donated by healthy volunteers rather than in a trial of patients.* The team also reanalyzed a publicly available gene sequencing dataset covering 26 human breast tumors. Their goal was to identify circulating factors from young animals that dampen inflammaging, and to test whether those factors change how breast tumors progress in older hosts.⁵

- **Inflammatory myeloid cells become more common with age in both mice and people** — Myeloid cells are frontline immune cells, including monocytes, macrophages, granulocytes, and dendritic cells. In aged mice, roughly comparable to people in their 50s to 70s, researchers found a marked increase in myeloid cells producing the inflammatory cytokines IL-1 α , IL-1 β , IL-6, and TNF- α .

People followed the same pattern. In blood samples from 90 healthy human donors, the share of cytokine-producing myeloid cells rose along with age. It was also higher in donors aged 60 to 87 than in donors aged 21 to 33.

- **The same inflammatory signature showed up in human breast tumors** — Working from the published dataset of 26 breast tumors from patients aged 35 to 88, the team compared the eight youngest with the eight oldest, all untreated before surgery. Tumors from the older patients contained a higher proportion of myeloid cells. Those cells were also running different genetic programs.

In younger patients, they favored genes for flagging threats to the rest of the immune system, while in older patients, they favored genes for inflammatory messengers, damage signals that injured cells release, inflammasome components, and regulators of NF- κ B, the master switch that turns inflammatory genes on.

- **Sharing a bloodstream with a young mouse lowered the inflammatory signal** — To test where the difference originated, the researchers used heterochronic parabiosis, a technique in which an older and a younger mouse are surgically joined so they share one bloodstream.

In these pairs, inflammatory myeloid cells in the aged animal dropped to levels comparable to young-young pairs, and aged animals bearing tumors showed delayed tumor progression and improved survival compared with aged-aged pairs.

- **Bone marrow transplant experiments narrowed it further** — Aged bone marrow placed into a young host produced far fewer inflammatory signaling proteins than the same marrow in an aged host. Young marrow placed into an aged host also became more inflammatory.

The host environment, rather than the age of the immune cells, appeared to govern the outcome. The effect persisted in mice engineered to lack T and B cells, indicating it did not depend on adaptive immunity.

- **A database search pointed toward the thymus** — Screening 163 aging-related molecules, the researchers noticed that mice lacking FOXO3, LMNA, or SIRT family genes both overproduce inflammatory cytokines and share one anatomical feature: a shrunken thymus.

That convergence led them to the thymic epithelial cells that secrete several hormones, among them thymosin α 1, thymosin β 4, thymopoietin, and thymulin. Thymulin stood out because prior work had shown it suppresses inflammatory cytokine production in human blood cells, and because its circulating activity declines with age to the point of being undetectable in older mice.

- **Thymulin lowered cytokine output, but only in aged samples** — Human blood cells treated with thymulin in the lab for 24 hours showed reduced cytokine expression in an age-dependent manner. Aged mice given daily injections for one week also showed a marked drop in the share of circulating cytokine-producing myeloid cells, while young mice showed no significant change.

In tumor-bearing aged mice, thymulin delayed tumor growth and improved survival across three separate breast cancer models, again with minimal effect in young animals. The researchers interpret this age dependence as thymulin restoring a signal that had gone missing rather than broadly revving up immunity.

- **Thymulin acted on NF- κ B, and the effects carried into T cells and drug response** — NF- κ B is a transcription factor that acts as an on-switch for inflammatory genes. It stays restrained until a signal frees it, at which point it travels to the DNA and turns those genes on. In immune cells from aged mice, thymulin interfered at both points: less NF- κ B reached the DNA, and less of the release signal got through.

To confirm the effect ran through NF- κ B rather than something else, the researchers used cells engineered to report NF- κ B activity as a readable output. Thymulin dampened it. When they disabled the exact spot on the DNA where NF- κ B attaches, thymulin stopped working altogether.

Inside the tumors of aged mice, thymulin increased T cell production of IFN- γ , a sign that those T cells were mounting an attack. In the same animals, tumors that had shown no response to anti-PD-L1 immunotherapy also became responsive once thymulin was given alongside it.

The authors note that the findings come from breast cancer models and how well they generalize to other cancers remains undetermined. They also caution that mouse-to-human age comparisons are not linear.⁶

What Drives Thymic Decline Over Time

To understand why thymulin declines, it helps to look at what happens to the thymus itself with age. A February 2026 narrative review published in Science Advances examines the broader process of age-related thymic involution,* the gradual loss of thymus tissue and function that begins much earlier in life than most people realize.⁷

- **Shrinkage begins young and reshapes the organ** — Involution starts as early as 6 weeks of age in mice and within the first years of life in humans, reaching its peak at puberty, when sex hormone levels rise. The gland loses the clear boundary between its outer and inner zones, develops regions empty of epithelial cells, accumulates fibroblasts, and in humans fills with fat.
- **Losing the gland surgically tracked with worse long-term outcomes** — Adults who had their thymus removed (thymectomy) showed reduced T cell production and narrower T cell receptor diversity, and they carried higher risks of death and cancer than control patients and the general U.S. population.

Risk of cancer relapse was also elevated, while autoimmunity risk was transient and modest. Young adults who underwent thymectomy in early childhood carry a T cell profile resembling that of middle-aged and older individuals.

- **Inflammation and thymic decline appear to reinforce each other** — Inflammatory signals rise inside the aging thymus and can speed up its shrinkage. As the gland loses function, it may also release less thymulin, leaving inflammation less restrained.

Together, the findings suggest a self-reinforcing cycle in which inflammation accelerates thymus decline, and that decline allows inflammation to build further. However, no single experiment has followed the full sequence.

- **Stress hormones can shrink the thymus quickly** — Psychological stress, infection, fasting, and other stressors raise glucocorticoid hormones, which can cause developing T cells in the thymus to die. The gland may recover once stress hormone levels fall, but repeated or prolonged exposure could place added strain on thymic function.
- **Low zinc is linked to reduced thymulin activity** — Zinc levels tend to decline with age, and the review links this drop to lower thymulin activity and thymic involution. In aged mice, zinc supplementation restored the gland's structure and its network of epithelial cells, although whether it can rejuvenate the thymus in generally healthy older adults remains uncertain.
- **Vitamin D deficiency accelerated thymic aging in mice** — Mice unable to produce the active form of vitamin D developed signs of premature thymic aging. Their thymic epithelial cells did not mature normally, and the gland became less effective at screening out potentially self-reactive T cells.

**These findings are from laboratory or animal research. Results may not directly apply to human health.*

How to Support Your Thymus as You Age

These findings do not translate into a single thymus-rejuvenation protocol, but they do point to several conditions that may influence how well the gland holds up with age. Inflammation, nutrient status, metabolic health, sleep, and toxin exposure all shape the environment in which your immune system operates. The practical goal is to preserve immune adaptability — your ability to keep producing fresh, diverse T cells well into adulthood. These five steps support that goal:

1. **Lower your inflammatory burden** — Your immune system functions better when it's not stuck in low-grade defense mode. Start with the biggest inflammatory triggers. Cut **seed oils** such as soybean, canola, sunflower, safflower, corn and grapeseed, along with the restaurant meals and packaged foods that often contain them.

These oils deliver large amounts of **linoleic acid** (LA), a polyunsaturated fat that, in excess, gets incorporated into your cell membranes and mitochondria, where it may oxidize and contribute to chronic inflammation. Replace them with grass fed butter, ghee, or tallow. Reducing your exposure to plastics and heavily fragranced products also cuts down on xenoestrogens, hormone-disrupting chemicals that interfere with cellular signaling.

2. **Eat for nutrients that support your thymus and immune function** — Aside from zinc, vitamin A may help maintain thymus structure and immune coordination,⁸ and vitamin C may support the gland and T cell production.^{9,10} Collagen-rich proteins also support tissue repair and immune recovery. You can obtain these nutrients from whole foods:

- **Zinc** — Oysters and ruminant meats
- **Vitamin A** — Egg yolks and pasture-raised, grass fed dairy
- **Vitamin C** — Citrus fruits, berries, and kiwi
- **Collagen** — Bone broth and slow-cooked ruminant cuts such as oxtail and shanks

3. Use movement, heat, and light to support your body's repair pathways — Your body relies on built-in signals that help maintain tissue health and cellular renewal. Several lifestyle strategies may help stimulate these processes without relying on hormone-based products:

- **Blood flow restriction training** — Also known as BFR or KAATSU, this method briefly limits blood flow to working muscles, creating a low-oxygen environment that can help build strength and muscle with lighter weights.
- **Exercise followed by sauna use** — This combination may support circulation, heat adaptation, and the body's natural elimination processes.
- **Whole-body vibration training** — Using a high-quality vibration platform, this method has been studied for its potential effects on muscle, bone, and metabolic health.
- **Red and near-infrared light therapy** — These forms of light have been studied for their ability to support mitochondrial energy production and cellular recovery.

4. Look into compounds that have been studied for thymic signaling — Certain natural substances have a long history of use for immune support. Astragalus, known as Huangqi in Traditional Chinese Medicine, is one example and has also been used to support endurance. Early research suggests that some of its compounds may help lower inflammatory stress, increase antioxidant activity, and improve immune signaling.¹¹

Researchers have also examined thymic peptides such as thymalin, thymosin β 4, and thymosin α 1 for roles in immune signaling, tissue repair, and antiviral activity, along with thymic protein A derived from calf thymus tissue.¹²

Most of these compounds are still considered experimental. Some require a prescription, and many are not FDA-approved for general use. They have been explored in regenerative and integrative medicine for chronic infections, immune

dysfunction, and age-related immune decline. Consult a qualified health care provider if you're considering any of these compounds.

- 5. Avoid the lifestyle factors that burden your immune system** — Smoking appears especially harmful, as tobacco exposure continuously floods your body with oxidative stress and abnormal cell damage, so eliminating it removes one of the largest ongoing burdens.

Beyond that, prioritize deep, restorative sleep and maintain a consistent sleep-wake schedule. Get morning sunlight to help regulate immune signaling, circadian timing, and mitochondrial function, and avoid excessive alcohol, which disrupts gut integrity and immune regulation.

Keeping chronic stress in check matters too, since it keeps inflammatory hormones elevated. Daily movement that preserves muscle mass as you age supports your metabolic health alongside it.

Frequently Asked Questions About Thymus Health and Inflammation

Q: What is thymulin, and what does it do?

A: Thymulin is a hormone released by the thymus. The featured study suggests it helps keep inflammatory myeloid cells from overproducing cytokines by restraining NF- κ B, a major switch for inflammatory gene activity. Thymulin activity declines with age, which may contribute to inflammaging.

Q: What does "inflammaging" mean?

A: Inflammaging describes chronic, systemic, low-level inflammation that increases as you get older, without an infection driving it. It has been linked to chronic diseases, including cancer, heart disease, metabolic conditions, and neurodegenerative diseases. Scientists have struggled to explain what causes it.

Q: Can thymulin lower age-related inflammation in people?

A: Researchers found that thymulin helped lower inflammatory signaling in donated human blood cells and aged mice, but it has not yet been tested as a treatment for inflammaging in human clinical trials.

Q: Why does the thymus shrink with age?

A: Thymic shrinkage begins early in life and peaks at puberty. Aging, rising sex hormones, chronic inflammation, stress hormones, nutrient deficiencies, and changes in the gland's epithelial cells may all contribute to the gradual loss of thymic tissue and function.

Q: What can I do to support immune health as I age?

A: Focus on the broader conditions that shape immune function: lower chronic inflammation, get enough zinc and other essential nutrients, exercise regularly, sleep consistently, avoid smoking and excessive alcohol, manage chronic stress, and maintain metabolic health and muscle mass. These steps may support immune resilience, though none has been shown to reverse thymic aging on its own.

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.

Sources and References

- ^{1, 3} Scientific American, July 21, 2026
- ^{2, 4, 5, 6} Nat Commun 17, 6534 (2026)
- ⁷ Sci Adv. 2026 Feb 13;12(7):eaeb2970
- ⁸ J Clin Med. 2018 Sep 6;7(9):258
- ⁹ Eur J Microbiol Immunol (Bp). 2024 Mar 6;14(2):67-74
- ¹⁰ Nursing Times, January 29, 2024
- ¹¹ Food BioscienceVolume 78, April 2026, 108531
- ¹² Int J Pept Res Ther 31, 10 (2025)