PEPTIDE PERSPECTIVES

BEYOND SUPPRESSING INFLAMMATION

Peptides & the Search for Immune Balance

A HEALTHIER IMMUNE SYSTEM IS NOT SIMPLY A QUIETER ONE

- The goal is a well-regulated immune response, not permanent suppression.
- Autoimmune diseases attack healthy tissues, with the immune system mistakenly targeting the body's own cells.
- Immune-mediated conditions include lupus, rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease, psoriasis, and sarcoidosis.
- Influencing one immune marker is not the same as controlling disease.



FOUR BIOLOGICAL PATHWAYS

INNATE AND ADAPTIVE IMMUNITY
- The coordinated actions of innate (rapid, broad defense) and adaptive (specific, memory-based) immune responses.

CYTOKINE SIGNALING
- Key immune messengers that drive or shape inflammation, including TNF-alpha, IL-1 beta, IL-6, IL-17, interferons, and IL-2.

REGULATORY T CELLS (TREGS) AND FOXP3
- Immune "brakes" that help maintain tolerance and prevent harmful self-attack, with FOXP3 as a key regulatory gene.

INTRACELLULAR INFLAMMATORY SIGNALING
- Internal cell pathways, such as NF-kappaB and MAPK, that turn on and amplify inflammatory gene programs.

PRESERVING INFECTION DEFENSE STILL MATTERS.
Immune balance aims to reduce harmful inflammation without leaving the body overly vulnerable to infections.

8 COMPOUNDS & THEIR EVIDENCE

KPV



- Small alpha-MSH fragment (Lys-Pro-Val).
- Targets intestinal inflammatory signaling and mucosal immune responses.
- Promising mechanistic and cell/mouse data (e.g., reduced inflammatory markers).
- No controlled human autoimmune treatment data.

VIP



- Neuroimmune peptide (vasoactive intestinal peptide).
- Modulates dendritic cells and supports Treg-related tolerance; studied in autoimmune models (e.g., MS, IBD).
- Mainly preclinical (cell and animal) evidence.
- Potential blood-pressure lowering and increased secretion (e.g., GI/respiratory) are considerations.

THYMOSIN ALPHA-1



- Regulates dendritic-cell function and T-cell responses.
- Studied for immune tolerance and immune balance in various models.
- Human trials in other conditions (e.g., chronic infections, cancer), but autoimmune disease evidence is limited.

LL-37



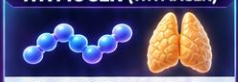
- Antimicrobial peptide with a double-edged role in immunity.
- Can contribute to autoimmune inflammation, such as psoriasis-related self-DNA signaling (innate immune activation).
- May be a pathway to understand or block (e.g., inhibit its harmful signaling), not simply add as a therapy.

ARA-290 (CIBINETIDE)



- Tissue-protective peptide (erythropoietin-derived, non-erythropoietic).
- Studied in sarcoidosis-associated small-fiber neuropathy (clinical research).
- Main interest is tissue protection and nerve support, more than direct autoimmune control.

THYMOGEN (THYMAGEN)



- Short thymic bioregulator (Glu-Trp).
- Limited, older immune studies suggesting modulation of thymic and immune function.
- Not established for autoimmune disease, with minimal modern clinical evidence.

VILON (LYS-GLU)



- Lys-Glu bioregulator (dipeptide).
- Studied in early research on thymocyte maturation and immune differentiation.
- Limited human evidence, with no established role in autoimmune disease.

LOW-DOSE IL-2



- Larger cytokine protein (interleukin-2), rather than a short peptide.
- Expands and supports regulatory T cells (Tregs).
- Notable human signal in lupus (phase IIb study showing reduced disease activity and steroid use).
- Investigational, not FDA-approved for lupus or routine autoimmune treatment.

WHAT GOOD IMMUNE-BALANCE TRIALS SHOULD MEASURE



- **Disease activity** (e.g., validated scores such as SLEDAI, DAS28, etc.).
- **Flare rates** (frequency and severity).
- **Patient symptoms** (e.g., pain, fatigue, function).
- **Quality of life** (validated patient-reported outcomes).
- **Infection risk** (rate and severity of infections).
- **Steroid-sparing effect** (ability to reduce or discontinue steroids safely).
- **Organ/tissue outcomes** (e.g., imaging, organ function, or tissue inflammation where relevant).

WHERE SCIENCE IS HEADING



- **More selective immune tolerance strategies** (targeting specific pathways or immune cell subsets).
- **Tissue-directed delivery** (e.g., gut, skin, lung, or lymphoid tissue) to improve effect and reduce broad immune impact.
- **Preserving antimicrobial defense** (maintaining the ability to respond to real infections).
- **Treg-focused therapies** (e.g., expansion, stabilization, or improved function of regulatory T cells).
- **Better translation from markers to patient outcomes** (linking immune changes to real clinical benefit).
- **Personalized immune phenotyping** (using individual immune profiles to guide who may benefit and which approach).

IMMUNE MARKERS, CELL SIGNALS, AND MOUSE MODELS ARE NOT THE SAME AS PROVEN AUTOIMMUNE DISEASE CONTROL.

Most featured compounds remain investigational for immune balance.

Educational research overview | November 2026 | Not medical advice