INFLAMMAGING: THE SLOW-BURNING INFLAMMATION ACCELERATING SKIN AGING

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ABSTRACT

Inflammaging refers to a chronic, low-level inflammatory process that develops over time and progressively increases with aging. Unlike acute inflammation, which is rapid and self-limiting, inflammaging is low-grade, persistent, and often clinically silent. Despite its low intensity, this ongoing inflammatory activity gradually disrupts skin homeostasis, affecting barrier integrity, immune balance, cellular metabolism, and extracellular matrix turnover. As a result, collagen and elastin organization becomes altered, tissue resilience declines, and skin recovery slows. These changes are associated with wrinkles, loss of firmness, uneven pigmentation, increased sensitivity, and delayed skin recovery. Although more evident with advancing age, inflammaging may begin earlier in life as a result of cumulative environmental and intrinsic stressors and can affect all skin types.

INTRODUCTION

Skin aging is a multidimensional process in which multiple biological changes occur simultaneously and progressively over time. Persistent low-grade inflammation influences skin behavior, reducing adaptability and recovery capacity and contributing to visible aging changes. Skin under chronic inflammatory stress has limited tolerance to additional trauma. Further inflammatory stimulation does not necessarily rejuvenate the skin; it can increase biological stress and may contribute to tissue decline. For this reason, a non-invasive approach is the most coherent strategy to support skin function and long-term stability.

PHYSIOLOGICAL INFLAMMATION

Physiological inflammation is a **normal, short-term repair response**. It can occur at any age and in all skin types after an acute stimulus. Inflammatory signaling is activated to repair tissue and is then reduced once balance is restored. On the surface, the skin may show mild redness or temporary sensitivity that resolves quickly. The person usually reports brief warmth, tightness, or discomfort that disappears as the skin recovers (triggers: minor injury, UV exposure, infection, invasive treatments). This state is **fully reversible** and restores normal skin function. If repeatedly overstimulated, it may shift toward chronic inflammation.

CHRONIC INFLAMMATORY STATE

A chronic inflammatory state may develop when inflammation is **repeated or does not fully resolve**. Inflammatory signaling remains active longer than necessary, barrier stability may decrease, and recovery may slow. The skin appears reactive, easily irritated, slower to calm, and less tolerant to products or treatments. The person often reports persistent sensitivity, burning, dryness, or the feeling that the skin never fully recovers (triggers: overly aggressive or frequent aesthetic procedures, barrier disruption, inappropriate skincare, chronic UV exposure, pollution, smoking, stress, hormonal imbalance, certain medications such as retinoids, corticosteroids, or immunomodulators). This state is **potentially reversible** if inflammatory triggers are reduced and recovery mechanisms are supported. If not addressed, it promotes inflammaging.

INFLAMMAGING

Inflammaging is **persistent, low-grade inflammation integrated into baseline skin function, with progressive severity over time**, especially if not properly managed. Inflammatory activity is inefficiently resolved, repair efficiency declines, and structural renewal progressively weakens. The skin may not look inflamed, yet firmness and elasticity may decrease and recovery may become consistently slow. Often describes the skin as fragile, less resilient, easily stressed, and unpredictable (triggers: intrinsic aging, cumulative UV exposure, pollution, oxidative stress, repeated unresolved inflammation, metabolic imbalance, long-term procedural overstimulation). Inflammaging is **not fully reversible**, but it can be **modulated and slowed**. If untreated, it progressively accelerates functional aging and limits the skin's capacity to respond to treatments.

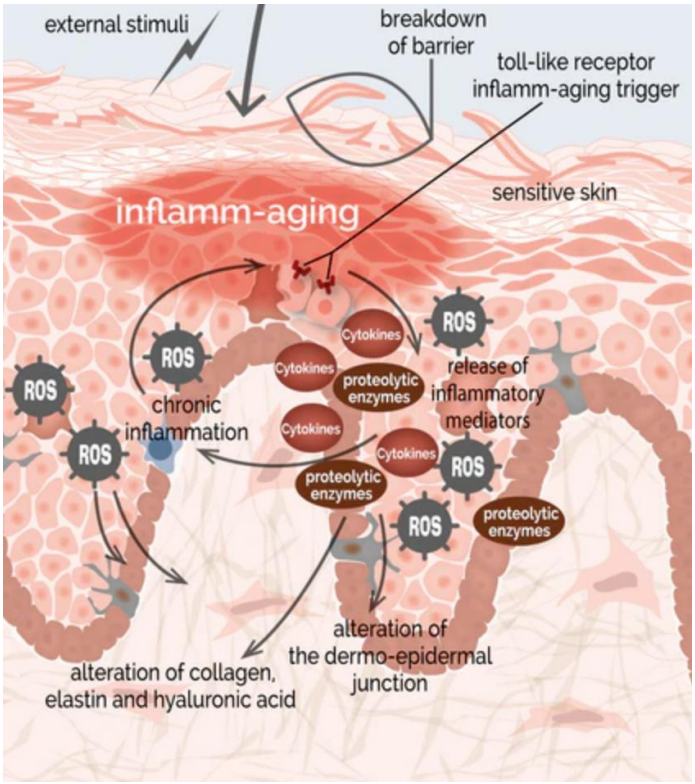

Figure 1. Cutaneous inflammaging: chronic inflammatory amplification and structural skin deterioration

Schematic representation of the biological mechanisms involved in cutaneous inflammaging. Repeated external stimuli contribute to epidermal barrier breakdown, leading to activation of innate immune pathways, including toll-like receptors. This activation initiates and sustains a chronic, low-grade inflammatory state characterized by continuous release of inflammatory mediators, reactive oxygen species (ROS), cytokines, and proteolytic enzymes. These factors amplify oxidative stress and inflammatory signaling, impair resolution and repair processes, and progressively alter skin structure. Over time, chronic inflammation contributes to extracellular matrix (ECM) degradation, disruption of the dermo-epidermal junction, and alterations in collagen, elastin, and hyaluronic acid organization. The cumulative effect is increased skin sensitivity, reduced resilience, impaired recovery capacity, and acceleration of functional skin aging.

Legend:

External stimuli: Environmental, mechanical, chemical, or procedural factors that challenge skin homeostasis

Breakdown of barrier: Disruption of epidermal barrier integrity, increasing permeability and sensitivity

Toll-like receptor inflammaging trigger: Activation of innate immune receptors involved in inflammatory and immune signaling

Sensitive skin: Skin with reduced tolerance and heightened reactivity due to impaired barrier and chronic inflammation

Chronic inflammation: Persistent, low-grade inflammatory state maintained over time

Release of inflammatory mediators: Continuous production of molecules that sustain inflammatory signaling

Cytokines: Pro-inflammatory signaling proteins that regulate immune and inflammatory responses

ROS (Reactive Oxygen Species): Highly reactive molecules contributing to oxidative stress and cellular damage

Proteolytic enzymes: Enzymes involved in the degradation of extracellular matrix components

ECM breakdown and remodeling: Progressive degradation and disorganization of the extracellular matrix

Alteration of collagen, elastin, and hyaluronic acid: Structural and compositional changes affecting firmness, elasticity, and hydration

Alteration of the dermo-epidermal junction: Structural weakening of the interface between epidermis and dermis, impairing tissue stability

Image Source: Adapted from Bare Your Skin®, “How Inflammaging Is Aging Your Skin,” 2021.

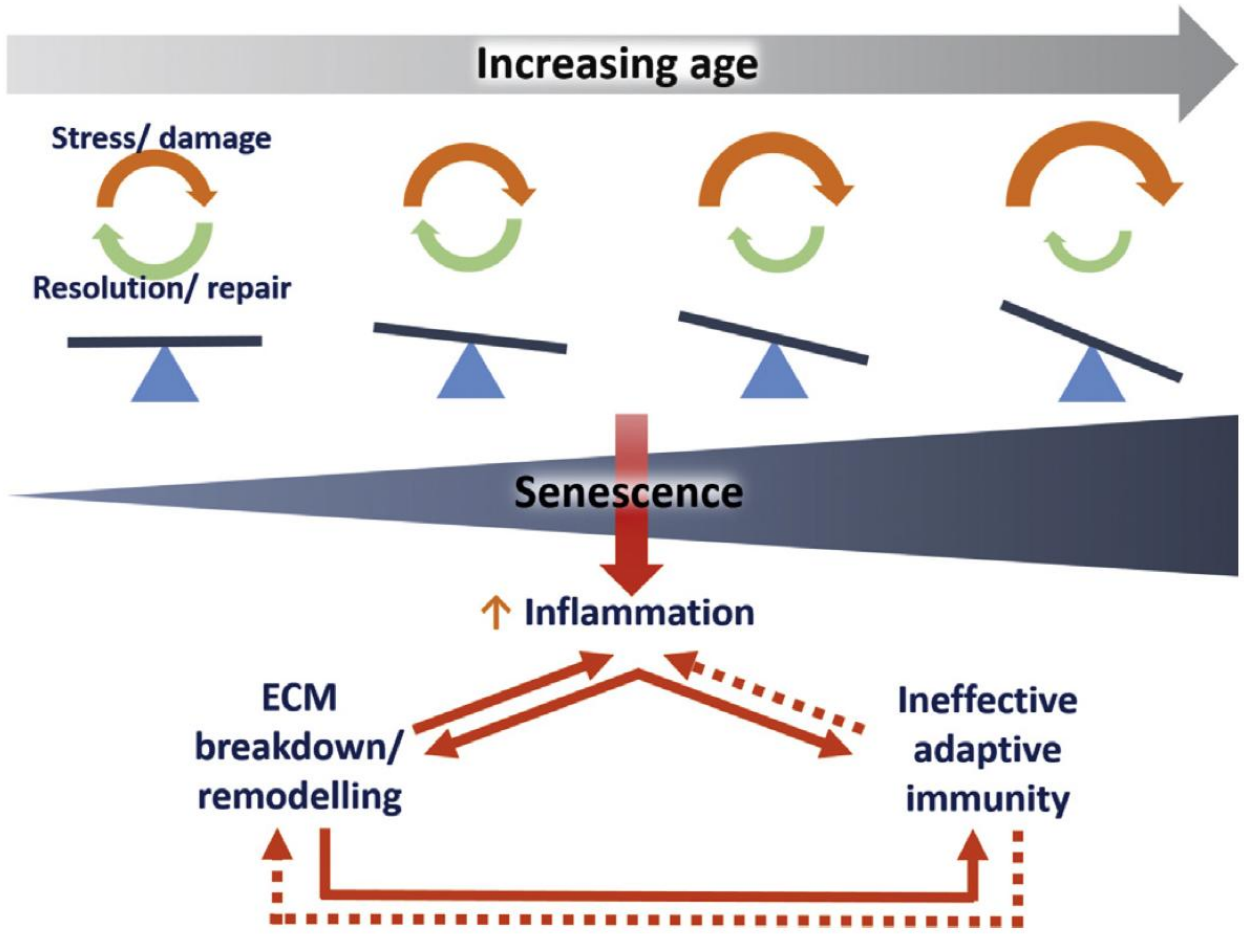

Figure 2. Inflammaging and progressive imbalance between stress and repair in the skin

Schematic representation of the cyclic relationship between repeated stress and damage ((environmental, metabolic, and procedural factors)), inflammatory activation, and resolution and repair processes in the skin over time. With increasing age, the efficiency of inflammatory resolution and tissue repair progressively declines, leading to the accumulation of cellular senescence. This shift is associated with a persistent, low-grade inflammatory state that contributes to extracellular matrix (ECM) breakdown and remodeling, reduced adaptive immune efficiency, and diminished tissue resilience. Over time, this imbalance accelerates functional skin aging and limits structural stability and recovery capacity.

Source: Adapted from Pilkington SM et al., Inflammaging and the Skin, Journal of Investigative Dermatology, 2021.

WHERE INFLAMMAGING OCCURS IN THE SKIN

Inflammaging develops when inflammation is not efficiently resolved and instead becomes part of the skin's everyday biology. It may settle quietly into the tissue and slowly change how the skin functions over time. In the **epidermis**, keratinocytes may remain under constant stress and stay in a defensive state rather than a regenerative one. Barrier turnover may slow, lipid organization may become less efficient, and the surface may lose cohesion. At the **dermal–epidermal junction**, persistent inflammation may weaken the structures that anchor and connect the layers. Communication between epidermis and dermis may become less effective, repair signals may be delayed, and the skin may lose resistance and

stability. In the **dermis**, chronic inflammation can affect both structure and circulation. Blood vessels may remain chronically stimulated, which may contribute to altered microcirculation and less efficient oxygen and nutrient exchange. Resident immune cells may remain continuously active and keep releasing low levels of inflammatory mediators. This constant inflammatory environment can alter fibroblast behavior: collagen and elastin may be produced with less organization, while matrix-degrading enzymes may remain active because tissue breakdown is continuously stimulated and repair cannot fully complete. Structural proteins may therefore be degraded faster than they are rebuilt, and the extracellular matrix gradually weakens.

WHY ADDRESSING INFLAMMAGING MATTERS

Skin aging is inevitable, but inflammaging represents an additional, potentially modifiable inflammatory burden. Physiological aging is characterized by a gradual decline in regenerative capacity, while chronic low-grade inflammation can shift the balance toward reduced repair efficiency and increased cumulative structural loss. Managing inflammaging does not stop aging, but it can help preserve functional balance, support recovery capacity, and mitigate the pace and depth of long-term tissue deterioration.

HOW TO MANAGE INFLAMMAGING IN THE SKIN

Managing inflammaging is not based on stimulation, but on reducing inflammatory burden, restoring tissue homeostasis, and supporting incomplete repair. In inflammaged skin, biological balance is altered, and the priority is to calm the system before asking it to rebuild. This skin often benefits from decongestion and drainage to reduce fluid stagnation, support microcirculation, and lower inflammatory pressure. It also requires renewal without inflammation, supporting epidermal turnover while preserving barrier integrity and avoiding additional stress. Repair must remain protective and supportive, reinforcing the barrier and stabilizing tissue structure rather than overstimulating it. JetPeel by TavTech provides a non-invasive method to combine microcirculation, gentle exfoliation, and active infusion without needles or tissue trauma. When paired with JetCare Med by TavTech infusion products, this approach invigorates collagen and elastin, improves skin density, elasticity, and firmness, and may help limit the progression of chronic inflammation that contributes to tissue degradation and structural decline, supporting skin function over time.

Figure 3. JetPeel treatment performed with JetCare Med infusion products

Image illustrating a JetPeel procedure performed without needles and without direct contact with the skin surface. The technology creates a controlled jet stream used for trans-epidermal infusion of active ingredients into the skin without needles or physical contact, while preserving epidermal barrier integrity. By avoiding direct mechanical friction, tissue trauma, and unnecessary inflammatory stress, this contact-free approach is suitable for addressing inflammatory skin conditions within a non-invasive and safe skin care strategy.

Image Source: Courtesy of TavTech Ltd., manufacturer of JetPeel technology. Image used for educational purposes.

CONCLUSION

In inflammaging, the priority shifts from overstimulation to the reduction of inflammatory load and the preservation of tissue homeostasis. These elements are essential to maintain skin resilience and recovery capacity over time. JetPeel by TavTech represents a coherent clinical approach by supporting microcirculation, oxygenation, skin renewal, and delivering targeted active ingredients into the skin through a non-invasive trans-epidermal delivery approach, without inducing additional inflammatory stress, thereby supporting skin function and long-term stability.