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Modulation of Skin Extracellular Matrix and Collagen Homeostasis: Differential Effects of Aerobic and Resistance Training on Skin Aging

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Abstract

Background. The structural integrity of the skin relies on the extracellular matrix (ECM) and collagen homeostasis, which degrade during aging. While physical activity is a systemic regulator of cutaneous homeostasis, the molecular divergence between distinct training modalities remains poorly characterized. Specifically, the pathways through which aerobic training (AT) versus resistance training (RT) influence skin health require elucidation.

Aim. To analyze the molecular mechanisms through which physical activity influences skin aging, focusing on ECM remodeling, skin density, and counteracting aging processes.

Material and methods. A narrative review of literature from the last decade was conducted, focusing on mechanotransduction, myokine signaling, and fibroblast gene expression under the influence of AT and RT.

Results. RT induces skin thickening by activating mechanotransduction pathways and stimulating structural genes like biglycan (BGN), HAS2, and CHSY1. This process is supported by the suppression of circulating plasma inhibitors such as CXCL4. Simultaneously, AT serves as a systemic antioxidant filter. By reducing senescence - associated secretory phenotype (SASP) markers and inhibiting matrix metalloproteinase (MMPs) activity, AT creates a protective environment that prevents the degradation of newly synthesized collagen fibers.

Conclusions. A hybrid training model (combining AT and RT) represents the most effective non - pharmacological strategy for skin rejuvenation. Such a dual - pathway approach stimulates the production of structural components and stabilizes the dermal environment against metabolic and oxidative stress. The integration of physical activity into anti - aging protocols offers a biological "reprogramming" of the skin, leading to improved structural integrity and resilience.

Key words: skin aging, extracellular matrix remodeling, mechanotransduction, resistance training, aerobic training, fibroblasts, myokines, IL-15, anti - aging.

1. Introduction

The skin is the largest organ of the human body and serves a key protective barrier function against environmental factors. Its structural integrity depends primarily on the dermal extracellular matrix (ECM), which is mainly composed of type I and III collagen, elastin, and proteoglycans. Among these, the most significant in the context of changes induced by physical exercise is collagen. Collagen constitutes the primary structural protein responsible for the mechanical strength and firmness of the skin, and its organized network determines the resistance of tissues to deformation [1,2].

Skin aging is associated with progressive changes in the structure of the ECM, including a reduction in collagen content, fragmentation of collagen fibers, and decreased synthetic activity of dermal fibroblasts. These processes result from both intrinsic aging and external factors, such as ultraviolet (UV) radiation, as well as poor dietary habits and addictions. An important mechanism of skin aging is the disruption of the balance between collagen synthesis and degradation, involving increased activity of matrix metalloproteinases (MMPs), as well as oxidative stress induced by an excess of reactive oxygen species (ROS), fibroblast senescence, and chronic low-grade inflammation [3–5].

Physical activity serves as a systemic regulator of skin homeostasis. Through the improvement of vascular function, increased tissue oxygenation, and modulation of oxidative stress, physical exercise regulates fibroblast activity and ECM remodeling. Myokines and mechanotransduction processes appear to constitute the key link in this interaction, determining the molecular response of skin cells to training stimuli. Nevertheless, the mechanisms integrating physical exercise with collagen metabolism have not yet been fully elucidated.

Different forms of physical activity may induce distinct biological effects. AT, through the reduction of oxidative stress, promotes the protection of collagen against degradation. In turn, RT, by utilizing mechanical stimuli and activation of anabolic pathways (including IGF-1 and TGF-β), demonstrates the potential to stimulate its synthesis.

The aim of this study is to provide a systematic review of current knowledge regarding the molecular effects of physical activity on skin structure. Particular attention is devoted to the comparative analysis of different forms of exercise in the context of counteracting aging-related processes, such as collagen degradation, oxidative stress, and inhibition of fibroblast activity, thereby highlighting the role of physical activity as a key factor in delaying the biological clock of the skin [6–8].

2. Materials and Methods

This study is based on a narrative review of the literature examining the molecular mechanisms through which physical activity influences skin aging and extracellular matrix (ECM) remodeling. Relevant publications from the last decade were identified through a comprehensive search across major scientific databases, including PubMed, Google Scholar, and Nature Portfolio.

The selection process evaluated studies based on their methodological quality and explicit focus on cellular signaling pathways, specifically mechanotransduction, myokine pathways, and fibroblast gene expression. Non - English publications, conference abstracts, and studies failing to address cutaneous biological processes were excluded. Due to the narrative nature of this review, findings regarding the distinct impacts of aerobic and resistance training were analyzed qualitatively.

3. Architecture of the Dermal ECM and Aging

3.1. Structural Components of the ECM

ECM constitutes the fundamental scaffold of the dermis, determining its structural integrity, elasticity, and mechanical resistance. The principal components of this complex network are collagen fibers, encoded primarily by the COL1A2 gene (type I collagen) and the COL3A1 gene (type III collagen), which cooperate with auxiliary collagens stabilizing the structure of larger fibers [6,9].

Tissue elasticity is ensured by networks of elastin fibers integrated with fibrillin. Oxytalan fibers are of particular importance in the upper layer of the dermis. These are microfibrillar bundles that run perpendicular to the skin surface and form a stable connection with the basement membrane, thereby preventing the detachment of the epidermis from the dermis [4,10].

In addition to fibrous proteins, proteoglycans such as decorin (DCN), versican (VCAN), and biglycan (BGN) play a key role in the architecture of the ECM. The latter, although traditionally regarded as a structural element, in the light of modern regenerative medicine serves as a superior organizer of collagen fibers. Its deficiency results in disrupted matrix organization and marked thinning of the dermis. Biglycan is currently recognized as a key marker of tissue sensitivity to mechanical stimuli because - importantly in the context of anti-aging prevention - resistance training directly induces an increase in its expression [6,11].

The overall structure is complemented by hyaluronic acid, which provides essential hydration and appropriate matrix volume [4,12].

3.2. Fibroblasts as the "Command Center"

The principal command center within the dermis consists of fibroblasts, whose biological activity is closely dependent on mechanical signals. In healthy skin, fibroblasts utilize specialized connectors in the form of integrins to attach to intact collagen fibers, which stimulates the reorganization of the intracellular cytoskeleton. This also maintains fibroblasts in a state of optimal activity focused on synthesis [13].

The mechanical tension generated within the cell in this manner constitutes an essential stimulus for the activation of new collagen production. Beyond their structural function, fibroblasts also play an important regulatory role with respect to the epidermis through the release of insulin-like growth factor (IGF-1). This factor is crucial not only for the proper proliferation of keratinocytes, but also for their effective protection against UV-induced damage [4].

Fibroblasts also exhibit high responsiveness to systemic signals originating from the body, including the myokine IL-15 (secreted by skeletal muscles during physical activity). IL-15 has been shown to improve mitochondrial energy efficiency in fibroblasts, which directly translates into enhanced production of structural skin proteins [6,8,14].

3.3. Molecular Mechanisms of Aging

The evolution of the skin with age is associated with the progressive degradation of this precise architecture. Aging leads to the accumulation of ROS, which, by acting as signaling molecules, activate key transcription factors such as AP-1 and nuclear factor kappa B (NF-κB). These molecular switches drastically increase the expression of MMPs, which directly impair skin integrity through the degradation of collagen and elastin. These enzymes act as molecular scissors, destroying collagen and elastin fibers. Simultaneously, fibroblasts accumulate in a state of so-called senescence (cellular aging). As a result of these processes, these cells cease to build the matrix and instead secrete pro-inflammatory factors that further inhibit the production of type I collagen. It is worth mentioning, however, that the levels of these enzymes also increase in naturally aging skin, while pro-inflammatory processes accelerate this phenomenon. Although the MMPs family is very broad, MMP-1 can be distinguished as the principal protease initiating collagen fragmentation within the skin [4,15,16].

The collagen fragmentation induced by MMP-1 leads to a situation in which fibroblasts lose their mechanical support. Deprived of stable anchorage, these cells cease to respond to anabolic signals (such as TGF-β), resulting in a rapid decline in the synthesis of new fibers [2,13].

The aforementioned molecular processes are reflected in the clinical appearance of the skin, manifesting as disorganization of the papillary layer of the dermis and progressive loss of tissue density. The loss of integrity of the protein network deprives fibroblasts of a stable scaffold, ultimately resulting in visible skin laxity, loss of elasticity, and altered skin texture [4,6].

The molecular picture of ECM degradation is further complemented by the effects of chronic exposure to cortisol, which acts as a systemic inhibitor of collagen synthesis. At the cellular level, cortisol directly suppresses the transcriptional activity of genes responsible for the production of type I and III collagen in fibroblasts and enhances the activity of metalloproteinases. This phenomenon is of key importance in the context of physical activity; while chronically elevated glucocorticosteroid levels lead to matrix atrophy, physical exercise optimizes the hormonal profile of the body, which constitutes an essential condition for the efficient progression of reparative processes within skin architecture [8,17].

4. Mechanisms of Muscle – Skin Crosstalk

Contemporary aging science defines skeletal muscles as an active secretory organ of an endocrine nature which, through the secretion of myokines and modulation of systemic signals, influences the condition of peripheral tissues, including the dermis [6,18,19].

This communication, referred to as muscle–skin crosstalk, constitutes a key mechanism through which physical activity modulates the ECM and collagen homeostasis.

This process is based on three pillars: the hormonal influence of myokines on cellular metabolism, the systemic activation of anabolic pathways, and the direct mechanical response of fibroblasts to the tension generated during muscle activity [6,18–20].

Key evidence supporting the existence of this axis was provided by studies based on the comparative analysis of plasma collected from participants before and after a training intervention. These studies demonstrated that it is the alteration in the profile of circulating factors, rather than solely local mechanical stimulation, that determines the rejuvenation of the skin phenotype.

Verification of this hypothesis under laboratory conditions confirmed that aging skin cells exposed to “active” plasma regain metabolic efficiency and exhibit characteristics of significantly younger tissues. This demonstrates that skeletal muscles, during activity, release specific signaling substances into the bloodstream that are capable of influencing fibroblast biology and reversing the effects of their aging. This mechanism is based on the action of myokines, the release of growth factors, and the response to mechanical tension [6,14].

4.1. The Endocrine Role of Muscles (Myokines): Analysis of the Effect of IL-15 on Mitochondrial Rejuvenation in the Skin

IL-15, whose secretion is directly stimulated by muscle fiber contractions induced by both AT and RT, constitutes a key mediator in the muscle - skin axis. This is confirmed by the human studies cited in the analysis, which demonstrate that acute physical exercise generates a rapid release of IL-15 into the bloodstream, reaching measurable elevated concentrations within as little as 10 minutes from the onset of activity. Although these levels return to baseline values after approximately 3 hours of recovery, repeated exposure to this myokine induces lasting adaptive changes in the skin [14,19].

The restorative mechanism of IL-15 within the dermis is based primarily on the stimulation of mitochondrial biogenesis and efficiency. Through the activation of the AMPK pathway and the transcriptional regulator PGC-1α in fibroblasts, energy deficits typical of senescent cells are reversed [14].

At the molecular level, IL-15 induces activation of AMPK kinase, which serves as a signal for metabolic reprogramming of the cell. The key effect of this cascade is an increase in the expression of subunits of respiratory chain complex IV (cytochrome c oxidase), which directly restores the efficiency of oxidative phosphorylation. As a result, fibroblasts shift from inefficient glycolytic metabolism to efficient ATP production. The increase in cellular energetic capacity enables the activation of protective mechanisms, which subsequently reduce the accumulation of mutations in mitochondrial DNA (mtDNA) and limit the levels of ROS. Restoration of energy homeostasis removes the biosynthetic block, allowing cells to resume collagen production and extracellular matrix regeneration [6,19].

The durability and stability of the described effects were confirmed in a 16 - week training protocol, in which participants performed regular exercise sessions twice per week. After this period, morphological analyses of the skin (performed using high-frequency ultrasonography and biopsy) demonstrated that such consistency ensures the necessary continuity of metabolic stimulation of fibroblasts. As a result of long-term exposure to IL-15, a significant thickening of the dermis and a marked improvement in its structural density were observed. This demonstrates that regular myokine release induced by physical exercise drives persistent adaptive changes that effectively inhibit age - related skin atrophy [6].

4.2. Anabolic Signaling and Growth Factors: The Role of the IGF-1 Axis and the TGF-β Pathway

Physical exercise induces an anabolic response that modifies the skin microenvironment through the activation of metabolic pathways crucial for the biosynthesis of structural proteins, including collagen proteins [6].

The central element of this response is IGF-1, whose secretion increases in response to post-exercise growth hormone (GH) release. IGF-1 acts not only as a circulating hormone but also as a local autocrine and paracrine factor, binding to the IGF-1 receptor (IGF-1R), which has tyrosine kinase activity [21,22].

Activation of IGF-1R initiates the recruitment of IRS-1/2 adaptor proteins, which triggers two key cascades: the PI3K/Akt/mTOR pathway, responsible for global protein synthesis and inhibition of senescence-associated autophagy, and the MAPK/ERK pathway, which stimulates keratinocyte proliferation. This mechanism is crucial for the thickening of the stratum spinosum of the epidermis and the increase in the number of metabolically active fibroblasts, which directly counteracts the loss of skin density and volume typical of the aging process [4,23].

In parallel, physical exercise reactivates the age-silenced transforming growth factor beta (TGF-β) pathway. In a young organism, binding of TGF-β to the TβRII receptor leads to phosphorylation of Smad2/3 proteins, which, after forming a complex with Smad4, translocate to the nucleus, where they act as transcription factors for collagen genes (COL1A1, COL1A2) [4,13].

In older individuals, this process is inhibited by the overexpression of the matricellular protein CCN1 (CYR61), which through interaction with αvβ3 integrins reduces the levels of TβRII receptors, rendering fibroblasts “deaf” to biosynthetic stimulatory signals. It has been shown that regular physical training markedly reduces CCN1 levels, thereby “unlocking” the cellular transcriptional machinery and restoring the capacity for type I collagen production as well as proper organization of elastin fibers [2,24].

Protection of the newly formed matrix is ensured by the reduction of systemic inflammatory status, manifested by a decrease in the level of the chemokine MCP-1 (CCL2). Lower circulating MCP-1 levels reduce the chemotaxis of monocytes into the dermis and their differentiation into M1-type macrophages, which are the primary source of MMPs [6].

Silencing of the MCP-1/MMP axis results in inhibition of collagen and elastin degradation. Consequently, physical exercise acts in a bidirectional manner: it promotes the molecular assembly of new protein structures via the IGF-1/TGF-β pathways and stabilizes ECM

architecture through the reduction of activity of enzymes responsible for dermal tissue degradation [4,6].

4.3. Mechanotransduction: Tissue Mechanical Tension as a Trigger for Chemical Cascades

Mechanotransduction constitutes a direct bridge linking muscular work to the remodeling of skin structure. This is of key importance, as both AT and RT affect skin tissue in distinct ways. During RT, loads and tension generate stretching forces that act directly on skin cells. This forces changes in cell shape and the activation of integrins - specialized receptor proteins functioning as connectors - which initiates proper incorporation of the actin cytoskeleton, referred to as the cell's internal scaffold, as well as activation of focal adhesion kinase (FAK). Additionally, correlation analysis showed that positive structural changes after RT were strongly associated with decreased levels of 24 different blood metabolites, highlighting the systemic nature of this response and demonstrating that physical tissue tension triggers a cascade of changes extending beyond mechanics alone [6].

In the case of AT, this mechanism is based to a greater extent on the mechanical effects of accelerated blood flow and increased microcirculation. The hemodynamic dynamics within dermal capillaries exert pressure on endothelial cells and adjacent fibroblasts. This mechanism activates mechanosensitive ion channels, which stimulates the release of nitric oxide (NO) and pro-angiogenic factors [6,25].

Additionally, the increase in body temperature during AT induces the expression of heat shock proteins (e.g., HSP70), which function as molecular chaperones, protecting collagen proteins from degradation [4,6,26].

This biochemical signaling cascade induced by physical exercise provides fibroblasts with an impulse to intensify biosynthetic processes, which is associated with a specific decrease in the blood concentration of markers such as CXCL4, CCL28, and N,N-dimethylglycine. Detailed plasma analyses have shown that the reduction of CXCL4 is particularly critical, as this chemokine acts antagonistically to growth factors that stimulate fibroblasts. The reduction of these inhibitors "unblocks" skin cells, enabling increased expression of BGN, which leads to strengthening of the extracellular matrix [6].

BGN, as a key proteoglycan, is responsible for fiber organization and promotes thickening of the dermal scaffold under mechanical loading. Through mechanotransduction, fibroblasts that have undergone morphological collapse during aging due to collagen fragmentation receive a stimulus to return to an active state. This process is crucial for restoring dermal homeostasis and enables the reactivation of anabolic pathways (such as TGF-β) and the reconstruction of

tissue mechanical integrity, ultimately leading to a visible reduction in signs of skin aging [6,27].

5. Comparative Analysis of Exercise Modalities

The type of physical exercise performed not only defines cardiovascular adaptations of the organism but also actively modifies tissue structure, including the dermal architecture. The detailed comparison of AT and RT presented in this chapter focuses on findings from the most recent literature, with particular emphasis on a key 2023 study (Nishikori et al.). As one of the first studies to demonstrate this in a comprehensive manner, it shows that despite a shared anti-aging objective, each of these modalities stimulates distinct molecular pathways, thereby producing complementary structural effects [6].

5.1. Aerobic Exercise as a Modulator of Metabolic Processes and Epidermal Quality

The process of skin architecture remodeling induced by AT can be defined as a cascade of events in which the key element is the connection between muscular activity and tissue condition through the circulatory system. This mechanism is initiated within active muscle fibers, which under endurance-type contractions activate their secretory potential, releasing myokines into the bloodstream, primarily IL-15. Simultaneously, the body's adaptation to regular exercise is manifested by an increase in peak oxygen uptake (VO_2 peak), which constitutes the foundation for improved peripheral microcirculation and increased capillary density within the dermis [6].

An improved circulatory system functions in this process as an efficient transmission channel, which not only delivers IL-15 produced in the muscles to fibroblasts but also modifies plasma composition through the reduction of systemic inflammatory markers such as interferon gamma (IFN-γ). This multidirectional interaction results in fibroblasts receiving a dual regenerative stimulus: on one hand, IL-15 stimulates mitochondrial biogenesis, enriching cells with ATP energy necessary for biosynthesis, while on the other hand, a metabolically "cleaner" microenvironment suppresses MMP activity, protecting newly formed collagen fibers from premature degradation [6,8,14,28,29].

The final stage of this cycle is a visible layered remodeling of the tissue. Improved perfusion and energy delivery to fibroblasts result in increased synthesis of hyaluronic acid (HAS2) and collagen, which clinically manifests as a reduction in low echogenicity areas (LEP) and improved elasticity. These changes are complemented by epidermal remodeling, where increased oxygenation induces a desirable thinning of the stratum corneum alongside a thickening of the stratum spinosum. In this way, systemic circulatory adaptation to exercise

completes the renewal process, restoring the skin's youthful structure and strengthening its barrier integrity [6,8,30,31].

Additionally, AT acts as a systemic antioxidant filter - by reducing circulating inflammatory cytokine levels (IFN-γ, IL-6), it creates an environment that slows collagen degradation induced by chronic metabolic stress. This process is supported by the stimulation of endogenous enzymes such as superoxide dismutase (SOD) and glutathione peroxidase, enabling effective neutralization of ROS and protection of extracellular matrix structure [32–35].

5.2. Resistance Training as an Architect of Dermal Density and Volume

The most groundbreaking differentiating conclusion between the two forms of activity is the finding that only RT possesses the capacity for the physical volumetric restoration of the dermis. While AT improves elasticity, RT genuinely increases skin thickness [6].

Analysis of the effects on skin structure allows the conclusion that a key condition for stimulating fibroblasts to rebuild the dermis is the application of progressive and sufficiently high mechanical load. In the analyzed study, a real effect in the form of a significant thickening of the dermis was achieved using a method based on increasing intensity: the protocol began with a load of 50% of 1RM (one-repetition maximum), and after six weeks of adaptation progressed to the target phase of 75–80% of 1RM. [6,36]

The remodeling to which the skin is subjected results from the ability of RT to suppress circulating plasma inhibitors of fibroblast growth (such as CXCL4 and CCL28), thereby allowing fibroblasts to produce biglycan without interference [6,37].

In contrast to AT, RT generates a unique cytokine profile in plasma, including an increase in CXCL8 levels. This molecule acts as a direct signal for fibroblasts to intensify the biosynthesis of ECM components. In parallel, there is increased expression of genes responsible for the production of key proteoglycans, such as chondroitin sulfate synthase 1 (CHSY1). This process significantly improves tissue hydration, which directly translates into increased resistance of the skin to mechanical deformation and improved skin tension [6].

The skin structural rejuvenation effect is closely correlated with an increase in lean body mass (LTM). Intensely working skeletal muscles, by increasing their volume and strength during regular resistance activity, release specific signaling factors into the bloodstream. This mechanism goes beyond the mere removal of inhibitory factors affecting fibroblasts - cells are actively stimulated to synthesize new structural components. In this context, the development of muscle mass ceases to be merely a body composition goal and becomes a biological foundation necessary for the physical thickening and improvement of dermal condition [6,38].

6. Discussion

The analysis of the collected evidence supports the hypothesis that the most complete anti-aging protection is provided by a hybrid model. Its effectiveness is based on a unique correlation: RT provides structural anabolic stimuli, while AT optimizes the environment in which regeneration occurs.

At the molecular level, RT acts as an initiator of tissue anabolism. Through the generation of mechanical tension and the suppression of plasma inhibitors (CXCL4, CCL28), it directly stimulates fibroblasts to express genes responsible for dermal structural density, such as CHSY1 (involved in chondroitin biosynthesis) and HAS2 (crucial for hyaluronic acid production). The result of these changes is a real thickening of the dermis and improved tissue hydration.

In this model, the role of AT extends beyond simple fat oxidation - it functions as a protective shield. Through the reduction of inflammatory state markers (SASP), including the chemokine MCP-1, aerobic exercise minimizes MMP activity. This is a critical mechanism for preserving aesthetic outcomes: AT prevents enzymatic degradation of newly formed collagen fibers, which without this protection would be broken down by chronic metabolic stress [6,39,40].

Only such a dual-pathway approach reprograms skin biology toward a full regenerative mode. The synergy between these two types of physical activity ensures that muscle mass gain becomes a permanent biological scaffold for rejuvenated skin, while the reduction of systemic inflammation associated with excess adipose tissue eliminates metabolic factors that degrade collagen.

As a result, the hybrid model makes the skin not only thicker but, above all, more resistant to mechanical stress and the destructive effects of time [6].

Nevertheless, despite the documented benefits for the ECM, the implementation of exercise recommendations requires consideration of various variables that may modify the final aesthetic outcome. A key element of this adaptation is the role of adhesion proteins, in particular β1-integrin, whose expression increases under the influence of regular mechanical loading [40]. As evidenced by studies, it is precisely these receptors that enable skin cells to properly respond to mechanical tension, converting physical stimuli into anabolic signals. However, it should be noted that in older individuals the efficiency of this system may be reduced. Improperly selected exercise intensity, lacking adequate recovery periods, risks a predominance of increased oxidative stress over repair processes [6,40].

Analysis of modifying factors also indicates a significant role of external barriers. While endogenous processes at the molecular level aim to strengthen dermal structure through the activation of structural components, exogenous factors associated with exercise may act in an antagonistic manner [6].

Changes in skin pH induced by sweat secretion and uncontrolled exposure to UV radiation during outdoor physical activity may weaken the epidermal barrier and degrade newly formed collagen fibers. In this context, the benefits derived from the reduction of SASP may be offset by superficial tissue damage. Therefore, a key recommendation is the protection of the skin's hydrolipid barrier and support of epidermal integrity, which allows full utilization of its regenerative potential [6,8].

7. Summary and Conclusions

The conducted analysis of scientific evidence confirms that regular physical activity represents an effective non-pharmacological method of remodeling the skin ECM and, consequently, influencing aging processes. The most comprehensive effects are achieved with a hybrid model based on the combination of resistance and aerobic training. While resistance training, through mechanotransduction, provides cells with a direct anabolic stimulus to increase tissue density, aerobic exercise optimizes the metabolic environment. By strengthening the antioxidant barrier and reducing systemic inflammation, it protects newly formed structures from premature degradation.

Despite evidence of exercise-induced fibroblast plasticity, contemporary science still faces the challenge of precisely determining the long-term durability of these changes. A key limitation of current studies is their short duration and focus on narrow demographic groups. Future research directions should focus on defining the optimal exercise intensity threshold that yields measurable structural benefits without the risk of overload. It is also worth investigating the interactions between physical activity and supplementation supporting connective tissue.

In summary, physical training extends beyond the scope of general health prevention, becoming a targeted biological intervention with anti-aging properties. Understanding that exercise "reprograms" the metabolic profile of the skin allows it to be regarded as a foundation of modern rejuvenation strategies. This approach not only improves aesthetic parameters such as thickness and hydration but, above all, durably increases the mechanical resistance of the dermis to the effects of time and environmental factors.

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