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THE IMPACT OF PHYSICAL ACTIVITY ON SKIN AGING: THE ROLE OF OXIDATIVE STRESS AND ADAPTIVE MECHANISMS - NARRATIVE REVIEW

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ABSTRACT
Skin aging is a multifactorial process driven by intrinsic mechanisms and environmental exposures, with oxidative stress playing a central role in cutaneous deterioration. Reactive oxygen species (ROS), generated both endogenously and through external factors such as ultraviolet radiation and pollution, contribute to collagen degradation, impaired barrier function, and structural skin damage. This narrative review summarizes current evidence regarding the relationship between physical activity and skin aging, with particular emphasis on oxidative stress–related mechanisms. Regular moderate exercise appears to have beneficial effects through improved cutaneous blood flow, enhanced antioxidant defense, mitochondrial adaptation, and stimulation of collagen-related pathways. Physical activity may also improve skin hydration, elasticity, and dermal structure. However, excessive or poorly adapted exercise may increase ROS production and inflammatory responses, potentially accelerating tissue damage. Outdoor physical activity additionally exposes the skin to environmental stressors, particularly UV, highlighting the importance of adequate photoprotection. Overall, the effects of exercise on skin aging appear dose-dependent and context-specific.

KEYWORDS
Skin Aging, Physical Activity, Oxidative Stress, ROS, Photoaging

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Introduction

The skin constitutes the largest organ of the human body. It has a wide range of functions, which includes protection against harmful organisms, substances and UV radiation (1,2). What is more, it also plays a crucial role in regulating body temperature (3) and maintaining fluid homeostasis by limiting transepidermal water loss (1,3). Overall, skin aging is characterised by changes in pigmentation resulting in uneven skin tone and reduced elasticity, progressive cutaneous atrophy, loss of subcutaneous and supportive tissue along with deterioration of the skin barrier function (4) which is manifested clinically as fine wrinkles, dyspigmentation, thin and transparent skin, dry and itchy skin (2,3,5,6), loss of the fat tissue that leads to hollowed cheeks and eye sockets and when it comes to skin appendages: hair loss or hirsutism, inability of sufficient perspiration and thinning of nail plates (6). What is more, histological studies have demonstrated epidermal hyperplasia, accompanied by disruption of collagen fibrils, as well as a significant accumulation of pathological elastin-rich material within the dermal connective tissue (5).

A wide range of external factors, including pollutants such as ozone and particulate matter (PM), solar ultraviolet (UV) radiation and visible light, contribute to elevated generation of reactive oxygen species (ROS) within the skin (6,7,8,9). Beyond externally induced ROS, certain oxygen radicals are also generated internally as byproducts of mitochondrial oxidative metabolism (9,10,11). Moreover, prolonged psychological stress can promote oxidative stress in the skin by weakening endogenous antioxidant capacity (9,12,13). Another key factor is the skin microbiota, which can be influenced by external factors such as UV radiation and pollutants and result in a disturbance in skin homeostasis and overall cutaneous health (14,15). Additionally, skin function is also influenced by bathing habits, having an outdoor hobby, diuretics use, moisturizer use (16,17) and sleep quality (18).

Objective

The objective of this narrative review is to critically synthesize current evidence on the impact of physical activity on skin aging, with particular emphasis on the role of oxidative stress as a central molecular mechanism linking exercise to cutaneous aging process. The review further aims to elucidate adaptive biological responses induced by regular physical activity, including antioxidant defense activation and cellular hormesis, as well as to examine the modifying influence of environmental factors encountered during exercise (such as UV radiation, air pollution, temperature and humidity) on intrinsic and extrinsic pathways of skin aging.

Methods and materials

The present work was designed as a narrative review based on a structured literature search. The search strategy encompassed the databases: PubMed and Google Scholar with the final update performed on 15th May 2026. Search terms combined concepts related to skin aging, physical activity and oxidative stress including “physical activity”, “exercise”, “training”, “skin”, “skin aging”, “photoaging”, “UV-induced skin aging”, “oxidative stress”, “reactive oxygen species”, “keratinocytes” and “fibroblasts”. These words combined in targeted manner to retrieve studies examining association between physical activity and processes of cutaneous aging, with particular focus on mechanisms mediated by oxidative stress as well as adaptive cellular responses occurring within the skin.

The review focused on studies published between 2016 and 2026. Eligible publications included observational studies conducted in human populations across different age groups and levels of physical activity, as well as randomized and non-randomized interventional trials evaluating the impact of exercise and external factors on skin structure and function, along with the molecular markers related to aging and oxidative stress.

Titles, abstracts and, when required, full-text articles were evaluated in relation to the review objectives and formulated research questions. Studies were included if they investigated the impact of the physical activity on skin aging, encompassing molecular, cellular or clinical endpoints related to oxidative stress, antioxidant defense mechanisms, photoaging, as well as the influence of external environmental factors occurring during exercising. Articles were excluded if they did not directly investigate the article’s objective, concentrated exclusively on outcomes unrelated to the skin, showed no relevance to oxidative stress pathways, were unavailable in full text or published in languages other than English.

The selected publications were analysed using a qualitative approach. Due to substantial heterogeneity among the included studies in terms of study design, methods used to assess exposure, characteristics of physical activity and timing of outcome assessments, the results were formed narratively, instead of being statistically aggregated in a new meta-analysis.

Biology of skin aging

Aging results from progressive functional deterioration occurring at both the cellular and systemic levels. Mammalian somatic cells exhibit a finite capacity for division and once this limit is reached, they enter a state of irreversible growth arrest known as cellular senescence. Senescent cells are characterized by permanent loss of proliferative ability, resistance to apoptosis and the release of pro-inflammatory and tissue-damaging mediators (3).

This is a process involving two related mechanisms: intrinsic (natural, chronological) aging and extrinsic (environment-related) aging. Intrinsic aging refers to naturally occurring time-dependent changes in key biological processes, including cellular alterations, genetic factors and hormonal shifts (19) and is caused by cumulative effects of free radical reactions and ROS-mediated damage to the essential cellular macromolecules (20). In contrast, extrinsic aging is driven by exposure to environmental stressors, lifestyle and dietary patterns, oxidative damage and other influences that contribute to the acceleration of physiological aging (2,21).

To systematically analyze the biological aging process, Lopez-Otin et al. (22) (updated by Jin et al.(23)) identified nine key hallmarks of aging, grouped into three categories: primary hallmarks (genomic instability, telomere attrition, epigenetic alterations and loss of proteostasis), antagonistic hallmarks (dysregulated nutrient sensing, mitochondrial dysfunction and cellular senescence) and integrative hallmarks (stem cell exhaustion and altered intercellular communication). According to their functional roles, primary hallmarks are regarded as sources of damage, antagonistic hallmarks act as responses to that damage and integrative hallmarks represent the resulting outcomes of the first two groups.

Embryonic development of the skin

The skin consists of three primary layers: epidermis, dermis and hypodermis. During embryogenesis, these layers arise from different germ layers. The epidermis is derived from the ectoderm, initially forming from an undifferentiated population of progenitor cells, forming the basal epidermal layer, enriched in epidermal stem cells (ESCs). The dermis is derived from the mesoderm located beneath the ectoderm, which is a major source of mesenchymal stem cells, which differentiate into multiple cellular lineages, including collagen-producing fibroblasts, vascular components that maintain cutaneous perfusion, subcutaneous adipocytes and resident immune cells within the skin (2,23).

Skin structure and function

The skin is a highly specialized, multilayered organ that forms the primary barrier between the body and the external environment, performing crucial protective, regulatory and sensory roles. The epidermis composed mainly of keratinocytes, represents the outermost barrier and is essential for limiting transepidermal water loss and shielding the organism from the environmental stressors. The epidermis is connected to the underlying dermis through the dermal-epidermal junction (DEJ). The dermis provides structural support and elasticity, primarily due to fibroblasts and well-developed extracellular matrix (ECM) rich in collagen, elastin and proteoglycanes. Fibroblasts play a key role in preserving dermal architecture through continuous synthesis and remodeling of ECM components, while also engaging in dynamic crosstalk with keratinocytes to maintain cutaneous homeostasis (2,3,24). The hypodermis, which contains abundant adipose tissue, nerves and vessels, contributes to insulation, thermoregulation, energy storage, mechanical protection and cutaneous blood flow. It also contains mesenchymal stem cells involved in wound healing and re-epithelialization. Beyond its structural functions, the skin also acts as an active immunological and metabolic organ, participation in inflammatory processes, antioxidant defense and overall homeostasis. Adipose-derived stem cells are defending the tissues from oxidative stress and inflammatory processes through the secretion of bioactive compounds and antioxidant factors. The proper organization and function of these components are critical for skin health and become progressively disrupted with aging (2,24).

Oxidative stress in skin aging

Mitochondria and ROS

Reactive oxygen and nitrogen species (ROS and RNS), including superoxide anion ($O2^{-}$•), hydroxyl radical (•OH), singlet oxygen ($^{1}O_2$), hydrogen peroxide (H_2O_2), nitric oxide (NO) and peroxynitrite ($ONOO^{-}$), are recognized as harmful to skin integrity. These reactive species can arise as byproducts of intracellular metabolic pathways, particularly in the mitochondria, where electron leakage occurs from the respiratory electron transport chain (10, 11). Other endogenous ROS are generated by cytochrome 450 metabolism (25), xanthine oxidase activity, peroxisomes and activation of inflammatory cells (for example neutrophils, macrophages and eosinophils).

External factors contributing to ROS generation include exposure to UV radiation, electromagnetic fields, ionizing radiation (e.g. γ-rays and X-rays) and environmental pollutants such as polycyclic aromatic hydrocarbons (PAHs), oxides, volatile organic compounds (VOCs), particulate matter (PM), and ozone (O_3) (6,17,25). Collectively, their presence contributes to cellular damage and plays a significant role in skin aging processes (10, 11). The human organism is equipped with multiple antioxidant defense systems that provide protection against ROS. These include enzymatic antioxidants, such as superoxide dysmutase (SOD), catalase and glutathione peroxidase, as well as non-enzymatic antioxidants including glutathione, ascorbic acid (vitamin C) and tocopherol (vitamin E). The main role of these compounds is to neutralize ROS and RNS, thereby preventing oxidative damage to cellular structures (9).

Oxidative stress

Oxidative stress occurs when the generation of oxidants, such as free radicals, ROS, RNS and other reactive metabolites, exceeds the capacity of the body’s antioxidant mechanisms to neutralize them (9, 26).

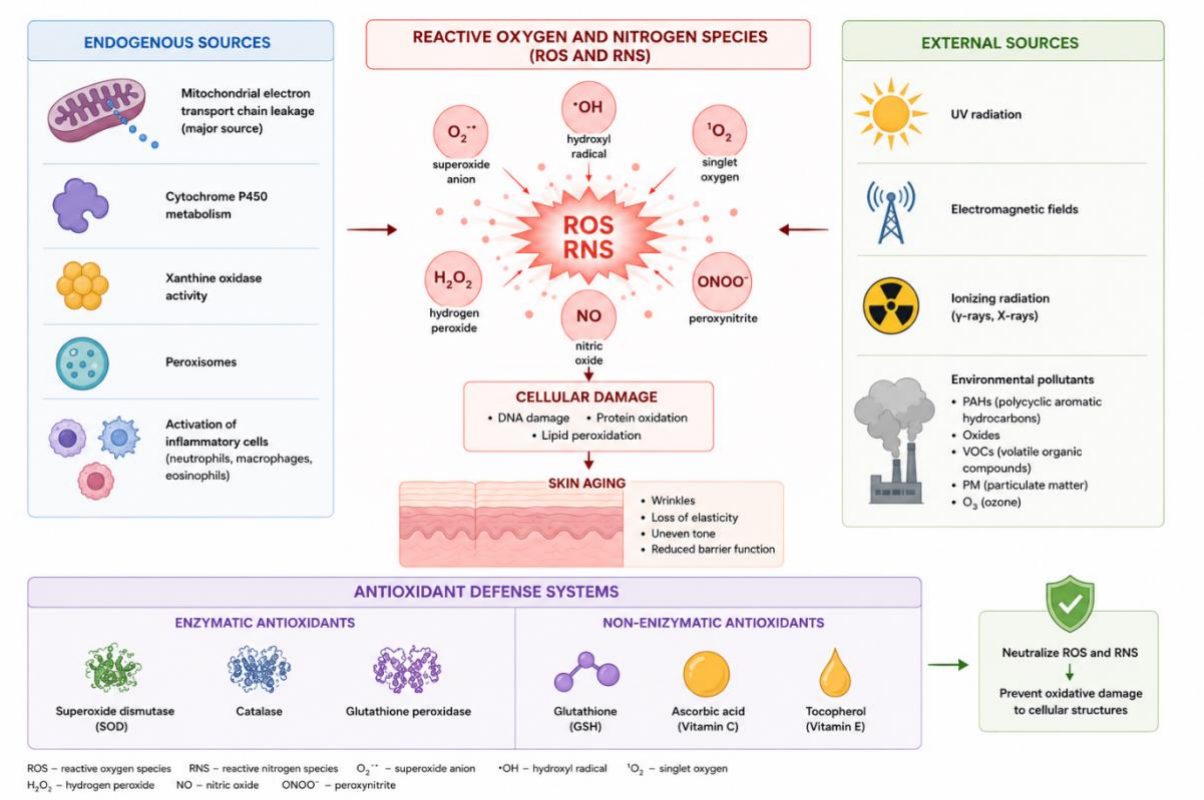

Fig. 1. *Endogenous and exogenous ROS and RNS sources.*

Photoaging

UV radiation, which originates both from natural sunlight and artificial sources such as mercury vapor, water-cooled and air-cooled lamps used for diagnostic and therapeutic purposes, can cause significant skin damage. UVR from sunlight is divided into three types based on wavelength: UVC (100-280 nm), UVB (280-320 nm) and UVA (320-400 nm) (8,27). Even low, chronic exposure to UVR can lead to tanning and contribute to accelerated skin aging, known as photoaging (28). Prematurely photoaged skin is commonly characterized by epidermal thickening, uneven pigmentation and damage to the dermal connective tissue, including characteristic solar elastosis, reduced elasticity, dull appearance, increased roughness and vascular alterations (2).

Most UVC radiation and part of UVB are absorbed by the ozone layer and therefore do not reach the skin (29, 30). The remaining UVB penetrates the epidermis and is responsible for erythema (sunburn), presenting with edema, pain and skin peeling (8,31). Prolonged exposure to UVB may damage DNA in skin cells, which elevates risk of photodamage, solar elastosis, photocarcinogenesis, actinic keratoses and epithelial tumors (8,32). Moreover UVB induces melanocyte activity, resulting in increased melanin production, which may cause hyperpigmentation and pigmented lesions, thereby negatively impacting skin's aesthetic appearance (9,33). Whereas UVA contributes to carcinogenesis and photoaging due to its high abundance in solar radiation and its ability to penetrate the dermis (5,28), where it damages collagen and elastin fibers (8). These components are crucial for preserving skin firmness and elasticity, and their degradation leads to formation of wrinkles and skin sagging (17). Both UVB and UVA radiation induce oxidative stress in the skin, leading to both transient and permanent genetic damage (5, 9). UVA radiation stimulates the production of pro-inflammatory mediators, including interleukin-1 (IL-1), IL-6, tumor necrosis factor alpha (TNF-α), platelet-activation factor (PAF), prostaglandins, insulin and nuclear factor kappa β (NF-κβ) (28). These processes are associated with upregulation of activator protein-1 (AP-1) activity and increased expression of matrix metalloproteinases (MMPs), which collectively contribute to extracellular matrix (ECM) degradation, inflammation and photoaging (5,7,8,27). MMPs are zinc-dependent endopeptidases with broad substrate specificity, enabling the breakdown of multiple components of the ECM. They are synthesized by keratinocytes and dermal fibroblasts in response to various stimuli, not only the UV radiation, but also oxidative stress and cytokines. To date, at least 28 different MMPs have been identified, which are involved

in numerous processes such as wound healing, skeletal development and remodeling, arthritis, inflammation, angiogenesis, carcinogenesis and the previously mentioned photoaging (5). What is more, MMPs upregulation increases the synthesis of collagenases, gelatinase and stromelysin-1 in fibroblasts and keratinocytes, what leads to degradation of collagen, elastin and other components of the matrix (28).

Exposure to UV radiation and the resulting oxidative stress also lead to local and systemic immunosuppression. This process is associated with activation of cytokines such as Il-4, Il-10, Il-1β, TNF-α and prostaglandin E2 (PGE2), as well as alterations in the morphology, function and number of Langerhans cells and T lymphocytes. Additionally, UV exposure contributes to increased local steroidogenesis within the skin (28).

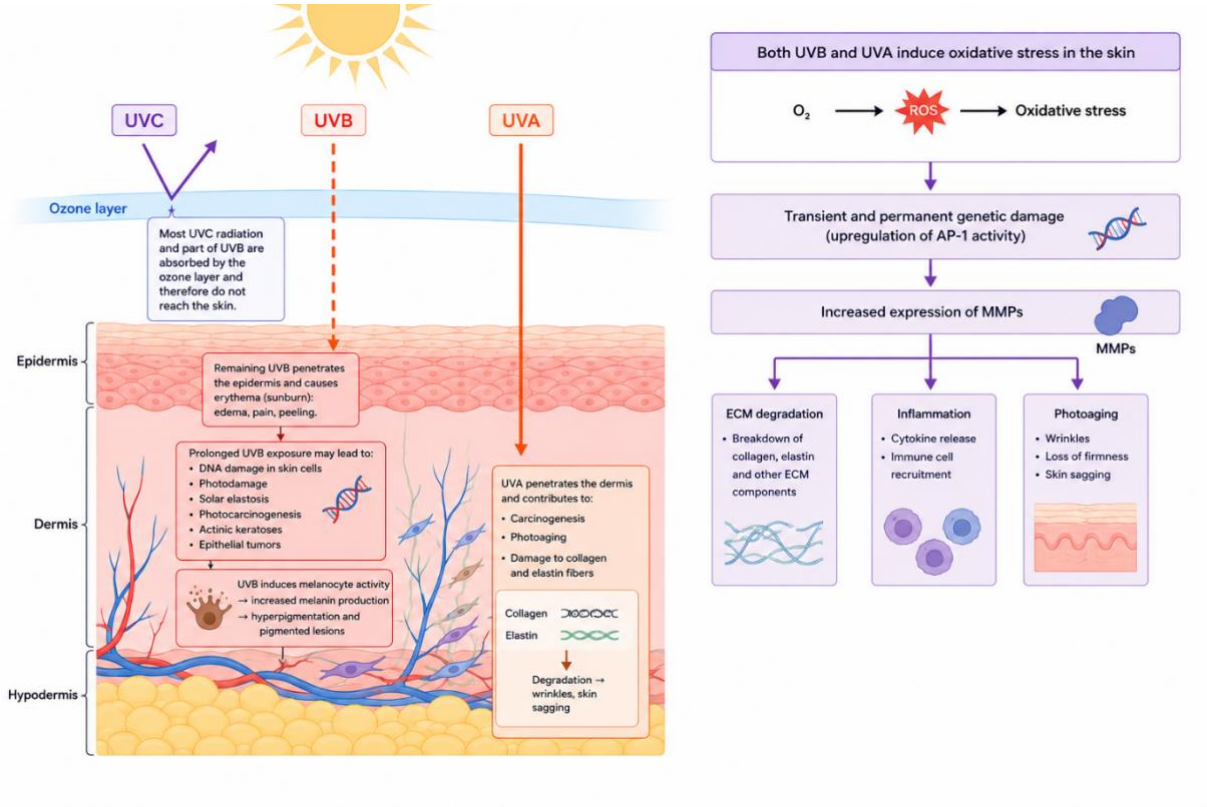

Fig. 2. *The effect of UV radiation on the skin.*

Photoprotection strategies

Many individuals engage in outdoor physical activity, therefore adequate protection against UV radiation is essential. First, outdoor activities should be avoided during peak UV radiation hours, which can be practically identified when shadows are shorter than the objects casting them. Additional photoprotective measures include the use of protective clothing, wide-brimmed hats and sunglasses. Exposed areas of the skin should also be protected with broad-spectrum sunscreen. Although the detrimental effects of UVA radiation have been known since the 1990s, more recent studies have highlighted the role of visible light (VL), especially blue light and long UVA (380-400 nm), in contributing to hyperpigmentation and skin aging (7,8,34).

The optimal sunscreen formulation should include a combination of UVB filters, such as PABA derivatives or cinnamates, together with UVA2 filters (e.g. avobenzone) and agents capable of protecting against UVA1 wavelengths, which have only recently been introduced into Europeans sunscreens several years ago (34).

Studies indicate that the use of sunscreen on the face and exposed skin is higher among women than men (30% vs. 15%) and is considerably lower among individuals with skin of colour as well as those who are not prone to sunburn (35).

Athletes commonly implement basic photoprotective measures such as sunscreen use, protective clothing and avoidance of outdoor activity during midday hours. However, sunscreen reapplication is still frequently neglected, which is particularly important during prolonged training sessions and in conditions

associated with excessive sweating. Despite the generally high public awareness regarding the harmful effects of UV radiation, athletes continue to experience sunburns relatively frequently, with the highest prevalence reported among kitesurfers, reaching up to 84,5% (36).

Effects of physical activity on skin aging

Physical activity has been shown to augment skin blood flow, with acute maximal exercise leading to an approximately eightfold increase in cutaneous perfusion, as indicated by total spectral power density, and in the population of older and postmenopausal women regular physical activity leads to enhancing cutaneous vasodilatation by roughly 1.5 times (1).

This represents a physiological response involving skin vasodilatation, accompanied by a rise in skin temperature, which facilitates the dissipation of heat generated during exercise. Cutaneous vasodilatation is diminished by sedentary lifestyle behaviours and aging and is also influenced by the level of skin hydration. However, there are no significant differences between male and female (1). That is to say, maintaining a regular exercise routine not only enhances cutaneous blood flow during physical activity but also improves overall vasodilatory function of the skin. Concerning skin hydration, it depends on a moisture gradient between the deeper layers and the skin surface. That's why maintaining sufficient blood flow is crucial for preserving skin hydration (1). Although direct evidence that exercise improves skin hydration is lacking, cross-sectional studies have found that regularly active adults and hospitalized patients tend to have better skin hydration compared to those who are inactive (37,38). Aerobic exercise and resistance training improves skin structure, leading to improvement of the elasticity of the upper skin layers due to enhanced ECM integrity. Additionally, in the resistance training group an increased skin thickness was noted, despite this parameter typically decreasing with age (39,40).

Physical activity and hormones secretion

Exercise influences hormonal secretion, including the increased secretion of growth hormone and estrogen (39). Both of these hormones play a role in regulating cutaneous collagen synthesis, which is responsible for skin elasticity and thickness (4), and its loss contributes to thinning and wrinkling of the skin. What is more it can be suggested that this may also influence elasticity of the skin and other related parameters.

Among women, stress may lead to hormonal imbalance, resulting in reduced levels of estradiol, free estradiol and luteinizing hormone (LH), accompanied by elevated concentrations of follicle-stimulating hormone (FSH) (42). This may contribute to skin roughness, decreased metabolic activity and dull complexion. Physical activity such as jogging, rope jumping and running promote the secretion of endorphins and dopamine, which not only helps reduce stress but also positively influences hormonal regulation (17). Additionally, prolonged psychological stress results in increased levels of adrenocorticotropic hormone (ACTH). Excessive secretion of this hormone may impair immune function, reducing the skin's defence capacity and potentially contributing to the development of inflammatory processes such as hyperpigmentation (13).

Physical activity as a modulator of oxidative stress

Regular moderate exercise reduces oxidative stress. First, it increases the production of antioxidants such as SOD and glutathione and enhances cellular resistance to free radical-induced damage. What is more, low concentrations of ROS stimulate the expression of antioxidant enzymes and anti-inflammatory factors (17,26). However, it is not solely a preventive strategy against aging, as physical activity also covers beneficial effects in older individuals. Physical activity modulates mitochondrial ROS production. With aging, mitochondrial dysfunction results in elevated ROS generation, which contributes to skin aging. Regular exercise can mitigate this dysfunction and stimulate mitochondrial biogenesis, thereby supporting the prevention of systemic functional decline (43). Another aspect is age-related thickening of the stratum corneum. Regular exercise performed twice weekly over a 12-week period has been shown to reverse these changes and decrease its thickness (43). In addition, facial exercises performed daily for 20 weeks have been associated with measurable aesthetic improvements in facial appearance in middle-aged women, resulting in a marked enhancement in fullness of both the upper and lower cheeks (17).

Endurance exercises have been shown to mitigate age-related alterations in the skin, partly through the action of IL-15, which contributes to the regulation of mitochondrial function in aging skin (43,44). Increased IL-15 expression is believed to result from activation of muscle 5' adenosine monophosphate-activated protein kinase (AMPK), a key metabolic regulator, whereas loss of muscle AMPK activity leads to deterioration of skin structure. This pathway is naturally stimulated by muscle contraction and nutrient deprivation, both of

which occur during physical exercise (44). An increased IL-15 concentration has been observed in many, although not all, studies. One possible explanation for these discrepancies may be the time interval between physical activity and sample collection; however, this issue clearly requires further investigation (43).

Unbalanced physical activity may exert negative effects on skin health. Insufficiently adapted or overly intense exercise may induce inflammatory responses in tissues, leading to cellular damage and potentially contributing to the development of skin diseases (17,26). This refers for example to prolonged endurance training, high intensity anaerobic physical activity and resistance exercise (26). This effect is primarily related to the marked increase in oxygen consumption during exercise, which may rise by approximately 10-20 times compared with resting conditions. As a consequence, the elevated metabolic demand promotes enhanced generation of ROS (17).

Limitations

The existing literature investigating the relationship between physical activity and skin aging remains relatively limited, especially in terms of clinical studies directly evaluating cutaneous outcomes. An additional challenge is the lack of standardized methods for the assessment of skin aging parameters, which reduces comparability across the studies. Moreover, considerable heterogeneity in study design, participant characteristics, types and intensity of physical activity and outcome measurement further complicates the interpretation of findings and precludes the formulation of definitive conclusions. Therefore, further well-designed studies are warranted to better elucidate the relationship between physical activity and skin aging.

Discussion

Physical activity emerges as a significant, yet multifaceted modulator of skin aging, influencing cutaneous biology through interconnected systemic and local mechanisms. The available evidence suggests that regular, appropriately dosed exercise promotes adaptive responses that may counteract key processes involved in skin aging, particularly those driven by oxidative stress. These adaptations include upregulation of endogenous antioxidant systems, improved mitochondrial efficiency, and activation of cellular stress-response pathways that collectively enhance the skin's resilience to damage.

At the same time, the impact of physical activity on skin cannot be interpreted in isolation from its physiological and environmental context. Exercise transiently increases oxygen demand and reactive oxygen species generation, and when excessive or poorly adapted, may exceed the capacity of antioxidant defenses, thereby contributing to molecular and cellular stress. Moreover, outdoor physical activity introduces additional external stressors, including ultraviolet radiation and environmental pollutants, which may amplify oxidative mechanisms implicated in photoaging.

Importantly, the relationship between exercise and skin aging appears to be dose- and context-dependent rather than strictly beneficial or harmful. Moderate, regular physical activity is likely to support skin homeostasis and delay certain features of aging, whereas extremes of intensity, duration, or inadequate adaptation may shift this balance toward increased oxidative burden.

Overall, physical activity should be considered a dynamic regulator of skin aging processes rather than a uniformly protective intervention. A deeper understanding of optimal exercise parameters and their interaction with environmental exposures is required to fully define its role in maintaining skin health across the lifespan.

However, the current body of evidence remains limited, particularly with regard to well-controlled clinical studies directly assessing skin-specific outcomes in relation to defined exercise modalities, intensity, and duration. There is also a lack of standardized methodological approaches for evaluating cutaneous aging parameters, which hampers direct comparison between studies and limits the ability to draw firm conclusions.

Further research is therefore warranted to better characterize dose–response relationships, elucidate underlying molecular mechanisms, and clarify the interaction between physical activity and environmental exposures in the context of skin aging.

Conclusions

Physical activity plays a complex role in skin aging by influencing both oxidative stress levels and adaptive antioxidant defenses. Regular moderate exercise may improve skin appearance and support its homeostasis through improved mitochondrial function, enhanced microcirculation, and activation of protective cellular mechanisms. However, excessive or poorly adapted physical activity, especially when combined with environmental stressors, may increase oxidative burden and contribute to cutaneous damage. Overall, its effects on skin aging are context-dependent and require further investigation.

Conflict of interests: The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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