

Effects of psychological stress on skin aging: A literature review

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ABSTRACT

Skin aging is a multifactorial biological process driven by the interplay of intrinsic and extrinsic influences. Progressive deterioration of the dermal collagen fiber network represents a central structural hallmark of this process. Both the quantity and functional quality of cutaneous collagen are modified by numerous factors, including autoimmune disease, chronological aging and psychological stress. This narrative review synthesizes current evidence on the neuroendocrine, molecular and cellular pathways through which psychological stress may influence dermal collagen and elastin remodeling, thereby contributing to premature skin aging. Available evidence suggests that chronic activation of the hypothalamic–pituitary–adrenal axis and sympathetic nervous system leads to increased glucocorticoid and catecholamine signaling, oxidative stress, extracellular matrix degradation and impaired epidermal barrier function. Understanding these mechanisms may support the development of integrated therapeutic strategies targeting stress-responsive pathways to preserve skin structure and function. Future studies that track patients over time and examine molecular changes are needed to turn these findings into ways to prevent or treat stress-related skin aging.

KEY WORDS: collagen, epidermal barrier, oxidative stress, hypothalamo-hypophyseal system

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INTRODUCTION

Genetic programming, hormonal changes and cellular senescence are the main causes of intrinsic aging, whereas environmental stressors such as UV radiation, pollution, smoking and nutrition are the main causes of extrinsic aging. [1] Psoriasis, atopic dermatitis, pathological impairment of barrier function and wound healing are among the cutaneous dermatoses that may be triggered by psychological stress. [2] Chronic stress is frequently associated with poor sleep, dietary imbalance and decreased self-care, all of which worsen oxidative and inflammatory damage. [1] There is currently only one publication that discusses obvious aging symptoms brought on by psychological stressors. This study was carried out on participants who were subjected to occupational stress brought on by a particular kind of professional job under very particular circumstances. [3] The effects of psychological stress on the autonomic nervous system, renin-angiotensin system (RAS) and hypothalamus-pituitary-adrenal (HPA) system are widely known, despite the fact that direct

evidence connecting psychological stress to aging is still developing. These systems are recognized to play a role in DNA damage, oxidative stress and inflammation all of which are recognized aging processes in all tissues, including the skin. [4] Chronic psychological stress is an additional potential element in aging that accelerates telomere shortening. It manifests as indications of skin aging in both people and rats. [5,6] According to recent studies, psychological stress may also affect skin aging by interfering with the integrity of the dermal extracellular matrix and the homeostasis of the epidermal barrier. Stress hormone signaling has been shown in both experimental and clinical studies to decrease the production of structural proteins and lipids in the epidermis. It also hinders stratum corneum hydration and increases transepidermal water loss. These changes not only compromise cutaneous defense but may also make the skin more susceptible to premature aging. [7]. In the dermis, stress-responsive molecular pathways have been shown to alter collagen and elastin networks through modulation of fibroblast activity, enzymatic

remodeling and oxidative injury. This weakens the biomechanical resilience and contributes to wrinkle formation and loss of elasticity [8-10]

AIM

The aim of this narrative review is to evaluate the current evidence on the relationship between psychological stress and skin aging. It seeks to summarize the underlying neuroendocrine and molecular mechanisms involved, examine clinical and epidemiologic findings.

MATERIALS AND METHODS

A literature search was conducted in PubMed and Google Scholar for articles published between 2016 and 2026. The following search terms were used in various combinations: "psychological stress," "mental stress," "perceived stress," "skin aging," "dermal aging," "wrinkles". Searches were limited to studies published in English and involving adult human subjects.

REVIEW

The database search yielded 44 records on PubMed and 17,800 on Google Scholar. After removal of duplicates and screening of titles and abstracts, 18 full-text articles and 6 abstracts were assessed for eligibility, of which 13 met the inclusion criteria. Included studies were published between 2016 and 2026.

Several studies reported that exposure to psychological stress activates the hypothalamic–pituitary–adrenal axis, resulting in increased production of stress hormones both systemically and within the skin. This neuroendocrine response was associated with impaired epidermal barrier function, including reduced synthesis of lipids and structural proteins, lower stratum corneum hydration and elevated transepidermal water loss. [7] Integrative studies highlight that persistent neuroendocrine activation enhances inflammatory signaling, induces oxidative damage and disrupts matrix metalloproteinase control, hence expediting the destruction of collagen and elastin. [11, 12]

Studies commonly assessed skin aging through clinician-based visual inspection, focusing on wrinkles, pigmentation changes and reduced elasticity. According to the literature, psychological stress contributes to skin aging through multiple interconnected mechanisms. It promotes cellular senescence and alters extracellular matrix remodeling. Stress also activates neuroendocrine pathways and increases oxidative damage within skin tissues. In addition, it influences subjective perceptions of aging, further linking psychological burden to visible cutaneous changes.

General reviews of skin aging describe the combined effects of intrinsic and extrinsic influences. Intrinsic factors include genetic predisposition, telomere shortening, immunological decline, hormonal changes, and cumulative oxidative stress. These internal processes interact with environmental exposures such as ultraviolet radiation, pollution, smoking, diet, and sleep disturbances. Together, they contribute to the development of wrinkles, skin laxity, pigmentary alterations, and surface irregularities. [9, 10, 13]

Clinical investigations provide human evidence supporting these observations. In a clinical study involving 36 participants, 18 were exposed to mild psychological stress and 18 to moderate psychological stress. Adults in the moderate stress group showed reduced antioxidant capacity. They also exhibited impaired epidermal integrity. In addition, this group demonstrated more pronounced microrelief changes, including increased fine lines and surface irregularities, compared with individuals experiencing lower levels of stress.

[11]. Cellular analyses from the same cohort indicated that elevated cortisol and epinephrine levels were associated with reduced extracellular matrix synthesis, DNA damage, delayed wound closure and altered expression of genes regulating collagen production and barrier maintenance [11]. Additionally, experimental research demonstrated that stress mediators directly affect cutaneous fibroblasts. A molecular role for sympathetic signaling in speeding up dermal aging is supported by the findings that epinephrine exposure caused premature cellular senescence, elevated pro-inflammatory pathways and reduced wound-healing capacity. [5] Complementary molecular reviews have explained that stress-responsive signaling pathways disrupt collagen and elastin networks. These pathways alter fibroblast activity and promote enzymatic remodeling of the extracellular matrix. As a result, the biomechanical integrity of the skin is weakened. This structural decline contributes to wrinkle formation and tissue laxity. [8]. Population-based surveys and observational studies reinforce these biological findings. Among young Asian women, higher levels of perceived psychological stress were linked to increased self-reported signs of early skin aging. These included a dull complexion, rough skin texture, and a perception of slower skin metabolism. In parallel, dermatologists and psychologists frequently identified dryness, acne, and irritation as common cutaneous features associated with psychological stress [14].

DISCUSSION

Chronic activation of stress-responsive systems, particularly the sympathetic nervous system and the

hypothalamic–pituitary–adrenal axis, appears to play a central role in stress related skin aging. Persistent stimulation of these pathways is associated with reduced extracellular matrix integrity and impaired epidermal barrier function. Across narrative, clinical, and epidemiologic studies, this neuroendocrine activation has been linked to the development of visible cutaneous aging features.[2,7,15]. These findings extend the current understanding of intrinsic and extrinsic skin aging by situating psychological stress as a physiologically reasonable and clinically significant modification of the aging trajectory [11,13].

A substantial amount of research links stress to changes in skin collagen. Stress's duration is a key distinguishing factor. Dysregulation of the circadian cortisol/corticosterone rhythm is a key sign of chronic stress's negative consequences. [16] The disturbance of epidermal homeostasis by catecholamines and glucocorticoids is one of the most often documented mechanisms. Stress-related decreases in structural protein expression, lipid synthesis and stratum corneum hydration, along with elevated transepidermal water loss, may weaken the barrier's integrity and increase susceptibility to environmental aggressors such as contaminants and UV light [7,15]. This breakdown of the barrier may accelerate age-related decrease in the dermal and epidermal compartments by sustaining low-grade inflammation and oxidative damage. [1,10] These findings lend credence to the idea that stress not only causes aging-related alterations but may also intensify other external factors that cause skin damage [13]. Chronic psychological stress raises the level of reactive oxygen species (ROS) and causes lipid peroxidation in keratinocytes and fibroblasts. Elevated ROS levels damage cellular membranes and DNA, causing mitochondrial malfunction and decreasing dermis flexibility [1].

Another important way that psychological stress seems to affect skin aging is through changes in dermal extracellular matrix remodeling. Long-term exposure to stress mediators may affect the skin's structural support and mechanical resilience, according to experimental evidence showing stress-induced fibroblast senescence and poor wound healing as well as molecular analyses of collagen and elastin network disruption. [2,10]. These changes are consistent with biomechanical modeling studies showing that collagen degradation lowers the threshold for wrinkle formation and contributes to laxity and textural changes [9]. Together, these results give the clinical relationships between stress and outward signs of aging a mechanical foundation [5, 7].

These molecular findings are supplemented by human observational and survey-based studies that show correlations between self-reported or clinician-assessed indicators of aging and perceived stress, even in very young populations [8, 5]. The case for a clinically significant connection between psychological stress and cutaneous aging processes is strengthened by the convergence of subjective sensations with objective biophysical and cellular measurements [7]. However, a large portion of the human evidence is still cross-sectional, which restricts the ability to draw conclusions about causality and increases the likelihood that observed links could be influenced by reverse relationships, such as distress brought on by skin problems [5, 8].

Future research should emphasize longitudinal cohort studies to clarify temporal links between stress exposure and progressive skin aging, as well as interventional trials assessing whether stress reduction can meaningfully modify biological age indicators in the skin [5, 7]. Standardizing stress measurements, including objective biomarkers like hair cortisol and integrating high-resolution imaging and genetic profiling would increase causal inference [7, 15]. Additionally, expanding research across diverse populations and age groups will be essential to ensure generalizability and to identify vulnerable subgroups [8].

CONCLUSIONS

Psychological stress should be acknowledged as a biologically significant modulator of skin aging, functioning via interrelated neuroendocrine, cellular and extracellular systems rather than as a peripheral or solely subjective effect. Its effects extend beyond obvious skin changes, influencing barrier function, extracellular matrix integrity and cellular resilience, as well as interacting with other inherent and extrinsic aging factors. This viewpoint reframes skin aging as a multifaceted phenomena in which psychological and physiological systems interact, emphasizing the significance of incorporating mental health into dermatological research and clinical procedures.

Furthermore, current research suggests that unexplored intervention opportunities exist: addressing stress-responsive pathways pharmacologically, nutritionally or behaviorally may supplement established treatments targeted at protecting collagen, elastin and barrier function. Conceptually, these findings show that skin aging is not only the result of chronological or environmental variables, but also a dynamic reflection of systemic stress exposure, stressing the need of viewing the skin as both a biological endpoint and a visible indication of holistic health.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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