Mini Review
Volume 9:7, 2022

Journal of Dermatology and Dermatologic Diseases

ISSN: 2684-4281 Open Access

Aging Skin: Pathophysiology and Cutting-Edge Treatments

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Abstract

The fight against aging, which is defined as a set of physiological mechanisms that alter the physical and intellectual capacities of humans, is one of the major challenges of the 21st century. This process includes more than just skin aging. Major healing defects are linked in part to changes in the biomechanical properties of skin cells, primarily dermal fibroblasts, that are associated with this condition. The immune system, which is also crucial for wound healing and maintaining skin homeostasis, is affected by time as well:Skin immunosenescence would result, promoting a pro-tumor environment while limiting the anti-infectious and vaccine responses. Before listing the effective anti-aging strategies to combat age-related dermal and epidermal stigmas, the primary skin damages caused by aging—whether intrinsic or extrinsic—will be discussed in detail.

Keywords: Skin aging • Senescence • Natural polyphenols

Introduction

The skin acts as a barrier that separates the human body from the environment and has the largest area of contact with the outside world. In addition to protecting the body from damage from the outside world and preventing water loss, it also has a cosmetic effect. Throughout our lives, organ aging occurs. The skin, the largest organ in the human body, clearly ages as a result of aging, exposure to ultraviolet radiation (UVR) and chemical pollution. People are trying to learn more about skin aging and pay more attention to it as science and technology advance and human living standards rise. Cosmetics and medications for the treatment and prevention of skin aging account for a significant portion of many people's daily expenses, particularly women. Research into the prevention and treatment of aging skin continues to be pushed forward by this enormous demand [1].

Description

There are three layers to animal skin: the epidermis, dermis and subcutaneous tissue. Skin epidermal cells rapidly differentiate into the stratum corneum, granular, spinous and basal layers during development. Undifferentiated organisms (SC) and transient intensification cells (TA) situated at the base layer advance the recovery of human skin epidermis. External signaling pathways, like the Wnt signaling pathway, control epidermal regeneration and SC behavior. The connective tissue that is made up of fibroblasts and is located below the epidermis is referred to as the dermis. It is the part of the skin that is above the subcutaneous fat and below the epidermis. The dermis is where collagen and other matrix proteins (like fibronectin, elastin and glycans) are made and secreted to the extracellular environment, giving the skin elasticity, strength and the ability to withstand external interference. Skin aging, carcinogenesis, wound healing, fibrosis and other pathological processes all involve fibroblasts. The fat layer immediately below the dermis layer, which surrounds the hair follicles and is responsible for connecting the skin to the muscles and bones, storing energy, secreting hormones and keeping the body warm, is referred to as the subcutaneous layer. Adipose tissue in the subcutaneous layer also plays a role in regulating the rate at which hair grows back, keeping the skin's internal environment in balance and encouraging skin repair after infection or damage [2,3].

In 1969, it was proposed that sun exposure contributes to skin aging in addition to intrinsic factors. The primary cause of extrinsic skin aging is UV exposure; Approximately 80% of facial aging is caused by it. UV-radiated epidermis thickens, in contrast to the thinner epidermis of naturally aged skin. Because corneocyte desmosomes are unable to be broken down, the stratum corneum, the epidermis' outermost layer, thickens. In accordance with the fact that UV irradiation impairs the differentiation process of epidermal keratinocytes, there is an increase in the expression of the differentiation marker involucrin in the stratum corneum. One of the epidermal stem cell markers, the expression of the cell-surface protein 1-integrin, which interacts with proteins in the extracellular matrix, is significantly reduced in basal cells. This indicates that basal keratinocyte proliferation is also impaired in aged basal cells.

The statement of type VII collagen in keratinocytes diminished in UV-transmitted skin regions. At the dermal–epidermal junction, the anchoring fibrils are made of Type VII collagen. Due to the weaker connection between the dermis and epidermis, its decreased production contributes to wrinkles. Collagen type I decreases in photoaged skin as a result of increased collagen degradation, according to studies. This degradation activity is carried out by a variety of MMPs, serine proteases and other proteases. In point of fact, it is extremely uncommon to observe p16INK4a-positive keratinocytes in normal skin, highlighting the continuous proliferation and differentiation of skin keratinocytes. However, senile lentigo skin has been found to have senescent keratinocyte changes. Feeble lentigo skin is portrayed by a thickened epidermis, perhaps because of the expansion of individual keratinocytes and not to changes in their numbers. The thickened epidermis may have senescent keratinocytes because anti-p16 antibody staining is more intense in the lesional epidermis [4].

Since the increased expression of p16 in seborrheic keratosis indicates that keratinocytes are in a senescent state, it has been hypothesized that senile lentigo shares a genetic basis with seborrheic keratosis. However, it is still unclear how senescent keratinocytes and their SASPs contribute to pigmentary changes caused by aging. SASPs like TNF-α, IL-1α, IL-1β and IL-6 are additionally emitted from UV-prompted senescent keratinocytes corresponding to the expanded degree of NF-κB and a decline in Y-box-restricting protein. Additionally, skin-aging pigmentation may result from autophagy impairment. Melatonin-induced autophagy activation protects keratinocytes from oxidative stress through the silent information regulator 1 (SIRT1) pathway and accelerated keratinocyte senescence impairs autophagy and induces intracellular protein aggregation by reducing melanosome degradation in hyperpigmentation. Hypopigmentation, on the other hand, can result from a defect in autophagy caused by mutations in the tuberous sclerosis

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Date of submission: 30 June, 2022, Manuscript No JPD-22-80933; **Editor Assigned:** 01 July, 2022, PreQC No P-80933; **Reviewed:** 15 July, 2022, QC No Q-80933; **Revised:** 20 July, 2022, Manuscript No R-80933; **Published:** 27 July, 2022, DOI: 10.37421/2684-4281.2022.9.359

complex (TSC) or the ectopic P-granules autophagy protein 5 homolog (EPG5) [5].

Conclusion

The relationship between UVR-mediated skin aging and inflammatory molecules was outlined in this review. External factors, most notably ultraviolet radiation (UVR), which damages DNA directly or indirectly through the production of radicals, can accelerate natural aging. It has been demonstrated that DNA damage causes the release of inflammatory molecules from epidermal keratinocytes (IL-1, IL-3, IL-6, IL-8, GM-CSF, M-CSF, G-CSF, TGF-, TGF-, TNF- and PDGF) and dermal fibroblasts (MMP-1, MMP-2, MMP-3, MMP-9, MMP-11, MMP-17 and MMP-27) in theSASP factors can be secreted by dermal fibroblasts and melanocytes as a result of chronic inflammation, which can make aging worse. Many age-related diseases, such as atherosclerosis, type 2 diabetes, obesity, cardiovascular disease, sarcopenia, neurodegenerative diseases and Alzheimer's disease, are caused by chronic SASP factor secretion. New strategies that target these inflammatory molecules are needed because it has been demonstrated that UV-induced inflammatory responses can accelerate aging and age-associated pathophysiology.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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How to cite this article: Zhang, Shoubing. “Aging skin: Pathophysiology and Cutting-Edge Treatments" J Dermatol Dis 9 (2022): 359.