Photoaging

Anne Han, MD[a], Anna L. Chien, MD[a], Sewon Kang, MD[b,*]

KEYWORDS

• Photoaging • Dermatoheliosis • Photodamage • Rhytids • Skin rejuvenation

KEY POINTS

- Photoaging is caused by chronic ultraviolet exposure leading to a complex process of skin changes that occur predominately on the sun-exposed cutaneous surfaces.
- Photoaging is more pronounced in fair-skin individuals and is characterized by subtle differences across ethnicities.
- Clinical manifestations of photoaging include rhytids, lentigines, telangiectasias, mottled pigmentation, coarse texture, laxity, and loss of translucency.
- Patients are concerned about their appearance related to photoaging and are influenced by society, culture, and personal values.
- A variety of modalities exist to prevent and treat photodamage, including sun protection, topical retinoids, cosmeceuticals, chemical peels, neuromodulators, soft tissue fillers, and light sources such as lasers.

INTRODUCTION

Photoaging is characterized by a complex process of skin changes induced over time by ultraviolet light exposure. It results in premature aging of the skin and is superimposed on the changes caused by chronologic aging. Not all photoaging is equal. The process is influenced by skin type and ethnicity. The degree of photoaging also depends on geographic location (ie, latitude and altitude), extent of sun exposure in relation to occupation and lifestyle, and photoprotective practices, including using sunscreens and photoprotective clothing, and seeking shade. Many patients use antiaging products or have corrective procedures.

Generally, patients are concerned about appearance and are influenced by society, culture, and personal values. Aesthetic ideals of beauty vary, yet the appearance of youthfulness remains a constant benchmark. Photoaging plays an important role in the degree to which youthfulness is retained despite advancing age.[1]

This article discusses the clinical features, epidemiology, histopathology, pathogenesis, and management of photoaging.

HISTORY

The term photoaging was first coined in 1986 and has been used interchangeably with the term dermatoheliosis.[2] However, the latter is a faulty neologism that implies a pathologic condition (osis) of the sun (helio).[3] Thus, photoaging is more accurate and is used exclusively throughout this article.

EPIDEMIOLOGY

Photoaging is more prevalent among populations with fair skin. Fitzpatrick skin types I, II, and III are more prone to photoaging than skin types IV, V, and VI. Ethnic origin, in particular Northern European descent, also plays an important role. In an Australian study of participants younger than the age of 30, moderate to severe photoaging was

Disclosures: None.

[a] Department of Dermatology, Johns Hopkins Medical Institutions, 601 North Caroline Street, 6th Floor, Baltimore, MD 21287, USA; [b] Department of Dermatology, Johns Hopkins Medical Institutions, 1550 Orleans Street, 2nd Floor, Baltimore, MD 21287, USA
* Corresponding author.
E-mail address: swk@jhmi.edu

Dermatol Clin 32 (2014) 291–299
http://dx.doi.org/10.1016/j.det.2014.03.015

observed in 72% of men and 47% of women.[4] In populations with darker skin, wrinkling is not readily apparent until the age of 50 and the severity is not as marked as in fairer skinned populations of similar age.[5] One study found that the onset of wrinkles in Chinese women occurred on average 10 years later than in French women.[6]

Photoaging is directly associated with cumulative sun exposure and, by extension, increasing age. Other factors include geographic location, such as high altitude and proximity to the equator where the harmful effects of ultraviolet light from the sun are most severe. Lifestyle practices, including outdoor occupations and outdoor recreational activities, increase cumulative sun exposure. For example, farmers, sailors, construction workers, and truck drivers frequently show severe effects of sun exposure over a lifetime. Indoor tanning is a practice that is also responsible for accelerated photoaging.[7]

Factors that diminish the features of photoaging include rigorous sun-protection practices. In Asian culture, in particular, women fastidiously avoid exposing their face to the sun.[8] They may wear large brimmed hats, carry parasols, and avoid the beach or other outdoor activities. These practices are highly influenced by societal ideals of beauty and attractiveness.

PATHOGENESIS

Both UV-A (320–400 nm) and UV-B (290–320 nm) seem to be implicated in the photoaging process, although UV-A is emerging as the major contributor because it penetrates deeper into the dermis and reaches the earth at least 10-fold more abundantly than UV-B.[9] UV-B radiation is mainly absorbed in the epidermis by cellular DNA, inducing damage with formation of cyclobutane pyrimidine dimers. UV-B is responsible for sunburn, photocarcinogenesis, and immunosuppression.[10]

Cumulative UV-A radiation causes damage to the dermal extracellular matrix and blood vessels. UV-A also indirectly damages DNA, as well as lipids and proteins, through the generation of reactive oxygen species (ROS). ROS cause oxidative damage to cellular components such as cell membranes, mitochondria, and DNA. Mitochondria are the main endogenous source of ROS and are produced during the conversion of ADP to ATP. Endogenous ROS, including superoxide anion, hydrogen peroxide, and singlet oxygen, activate cytokine and growth factor receptors, which in turn induce transcription factor activator protein 1 (AP-1) and NF-κB.

Fig. 1 demonstrates the pathogenesis of photoaging. UV radiation activates growth factor and

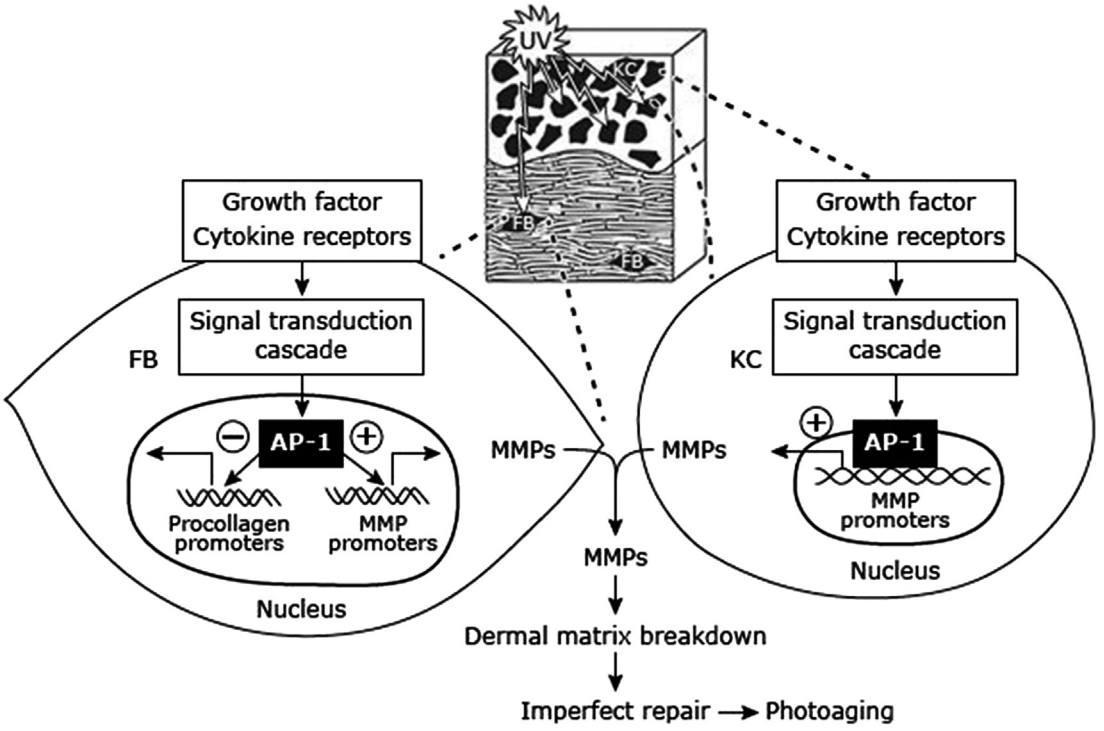

Fig. 1. Pathogenesis of photoaging. FB, fibroblast; KC, keratinocyte. (*From* Fisher GJ, Kang S, Varani J, et al. Mechanisms of photoaging and chronologic skin aging. Arch Dermatol 2002;138:1462; with permission.)