Skin Care

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KEYWORDS

• Photoaging • Retinoid acid • Sunscreen • Cosmeceutical • Sun damage

KEY POINTS

- Photoaging can be minimized or delayed with proper use of sun protection.
- Retinoids can reverse some of the signs of aging when used consistently.
- Vitamins applied topically can minimize damage from sun exposure and also act as antioxidants to limit environmental damage.
- The SPF system may underestimate the deleterious effects on the skin by neglecting the contributions of ultraviolet A radiation.
- Human studies of topically applied antioxidants are limited, but early work suggests they may have a protective effect against cellular changes resulting from photodamage.

Aging skin is among the most common patient concerns in a facial plastic surgery practice. Ultraviolet (UV)-induced damage expedites the pace of intrinsic aging, resulting in many of the visible signs of aging, such as rough skin texture, pigmentation irregularities, fine and deep wrinkling, and inelasticity. Primary prevention of UV and environmental damage with proper skin care and the use of sunscreen are critical. There is great interest in topically applied products to reverse or delay the visible signs of photoaging. There is an extraordinarily diverse array of prescription and cosmeceutical products available for the consumer. This article discusses the most common topically applied agents for photoaging, reviewing their mechanisms and supporting evidence.

SUNSCREEN

UV irradiation leads to the production of reactive oxygen species and activation of intracellular signaling pathways that result in an increase in inflammatory mediators. These mediators interfere with synthesis of dermal collagens and trigger synthesis of enzymes that degrade the extracellular matrix. This effect is compounded by UV damage to the mitochondrial genome, resulting in double-stranded breaks that affect the mitochondrial ability to produce energy for the cell and lead to further accumulation of reactive oxygen species. Moreover, chronic UV irradiation modifies local immunoregulation and cell survival, leading to impairment of intrinsic cancer surveillance.[1] Both UVA and UVB spectrums are implicated in photodamage. UVB photons are on average 1000 times more energetic than UVA photons, making them a major contributor to photoaging and photocarcinogenesis. However, UVA is found in up to 10 times greater abundance in sunlight, and has greater depth of penetration into the dermis compared with UVB, giving it a possibly even greater role in photoaging.[2] The visible effects of chronic long-term UV exposure are well-understood. The facial skin of females living in regions exposed to higher UV had significantly more and longer wrinkles, more and larger hyperpigmented spots, rougher surface texture, and more yellow discoloration based on computer analysis than women living at lower UV levels.[3] Topical application of photoprotective agents significantly

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reduces the lifetime UV exposure compared with nonuse, with regular daily use beginning early in life being the most important factor.[4] A randomized, controlled Australian study of 903 adults investigated the effect of daily use of a broad-spectrum sunscreen versus discretionary use and found that daily sunscreen use reduces the signs of skin aging based on skin surface replicas (level I evidence).[5] Skin surface replicas were made from the back of the left hand by using a silicone-based impression material. These replicas were then graded by experienced evaluators who were blinded to their treatment group, and assessed based on severity of skin changes. This study found good intragrader and intergrader reliability.

There are 2 main categories of topically applied sunscreens: organic (previously called chemical) and inorganic (previously called physical). Organic sunscreens absorb UV irradiation, converting it to heat and thereby preventing its untoward effects in the skin. These compounds are typically not visible when applied and are therefore widely used. In contrast, inorganic sunscreens contain particles such as zinc oxide or titanium dioxide that reflect photons away from the skin. The earlier generations of inorganic sunscreens were less cosmetically desirable because their large particle size resulted in a visible and comedogenic coating on the skin. Newer iterations have micronized or nanosized the active particles, resulting in improved cosmetic appearance and alteration of the spectral absorption profile.[6] The protection provided by sunscreen agents is traditionally quantified by SPF, or sun protection factor. This measure is defined as the minimal erythemal dose in sunscreen-protected skin divided by the minimal erythemal dose in non–sunscreen-protected skin. As UVB is the overwhelmingly greater contributing factor to sunburn than UVA, the SPF system was created to indicate the level of UVB protection. Thus, there is concern that the SPF system may underestimate the deleterious effects on the skin by neglecting the contributions of UVA radiation. Furthermore, the use of skin erythema as a surrogate for the more important underlying cellular alterations and local immunomodulation is called into question.

New awareness of the effects of UVA and its underrepresentation in current labeling systems prompted the US Food and Drug Administration (FDA) in 2011 to publish new guidelines directing the labeling and effectiveness testing for sunscreens. This ruling outlined the testing standards for coverage against both UVA and UVB for sunscreen to carry a label of “broad spectrum.” Also, this ruling for the first time provided permission for broad-spectrum sunscreen with SPF of 15 or greater to carry the claim to decrease skin cancer and early skin aging.[7] One important issue addressed in the recent ruling is that there is no maximum allowable SPF label. Companies market sunscreens with SPF exceeding 100, which may provide consumers with a false sense of protection, resulting in prolonged UV exposure and failure to reapply sunscreen as directed. There is thus a recommendation by the FDA to cap the maximum SPF label at 50+, because there is not sufficient evidence to support increasing efficacy beyond SPF 50.[8] There is not currently a UVA rating system in the United States, although a 5-star UVA protection rating system has been recommended for industry use in the European Union.

RETINOIDS

Topical retinoids as a treatment for photoaging have the most extensive evidence-based support in the literature and, not surprisingly, has been widely adopted in clinical practice. Available topical retinoids include retinol, retinaldehyde, tretinoin (retinoic acid), isotretinoin, tazarotene, and adapalene. Tazarotene and adapalene are synthetic retinoinds.

Photodamage expedites the natural aging process resulting in skin discoloration, roughness, and wrinkles, the appearance of which can be improved with the use of retinoids. Retinoids act via a tretinoin-specific gene transcription factor, suggesting that retinoids leverage their effect on the skin through regulated gene expression.[9] On a molecular level, retinoids bind to specific retinoic acid receptors, which serve as ligand-dependent transcription factors that regulate a diverse array of mediators to increase epidermal integrity and modulate the production of procollagen.[10] As reviewed by Fisher and Voorhees in 1996,[11] retinoid acts in vivo by inducing keratinocyte proliferation and modulating their differentiation, thereby increasing epidermal thickness, even by as much as 40% in some studies (compared with 10% in controls).[12] This effect occurs regardless of topical or oral administration, which argues against a previously held belief that retinoid effect was mitigated solely through its irritant effect on the skin. Histologically, topical tretinoin has been shown to result in epidermal hyperplasia, compaction of the stratum corneum, and restoration of cell polarity.[13] In addition to modulating cellular proliferation, some of the effects of retinoids may be mediated through collagen synthesis. Dermal collagen is central for providing resiliency to the skin, and its regulation and synthesis is an important component in the mechanism of the effect of tretinoin on photoaging.