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Review

Photoaging

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A B S T R A C T

Among harmful environmental factors that contribute to extrinsic aging, long-term effects of repeated exposure to ultraviolet light are the most significant and are referred to as photoaging. Photoaging is a multisystem degenerative process that involves the skin and skin support system. It is a cumulative process and depends primarily on the degree of sun exposure and skin pigment. The epidermis and dermis are both affected by UVB, but the dermis is also affected to a significant extent by UVA. It has long been thought that the majority of human photo-lesions due to UVB rays, now it is believed that UVA play a substantial role in photoaging. Photoaging affects the sun-exposed areas and is characterized clinically by fine and coarse wrinkling, roughness, dryness, laxity, teleangiectasia, loss of tensile strength and pigmentary changes. There is also an increase in development of benign and malignant neoplasms on photoaged skin. During the years the progress has been made in understanding the photoaging in human skin. UV irradiation invokes a complex sequence of specific molecular responses that damage skin connective tissue. Restriction of UV irradiation and the use of high-protection, broad-spectrum sunscreens may slow progression of photoaging.

Key words: *photoaging, UV radiation, photoprotection*

Introduction

The sun is vital source of energy for all life on Earth, and as we know, the life is impossible without sun. The sun emits visible light, infrared radiation, which is not considered harmful to humans, and UV rays. Exposure to sun with living in an oxygen-rich atmosphere causes unwanted photo-damage. Less than 5% of the sunlight that reaches the earth's surface is ultraviolet radiation. All the people are exposed to solar ultraviolet radiation. Having a suntan has long been synonymous with beauty, good health and dynamism in our culture. Also, an increasing number of people are exposed to artificial source of ultraviolet radiation used in industries, commercial settings and leisure activities. Unlike chronological aging, which depends on the passage of time *per se*, photoaging depends primarily on the degree of sun exposure and skin pigment. Individuals who have outdoor lifestyles, live in sunny climates, and are lightly pigmented will experience the greatest degree of photoaging[1].

Among harmful environmental factors that contribute to extrinsic aging, long-term effects of repeated exposure to ultraviolet radiation are the most significant and are referred to as photoaging. Photoaging is directly correlated to the quantity of UV rays received during the course of a lifetime. The effects of photo-damage are often evident many years before intrinsic aging is apparent. Young people who are exposed to a great amount of UV rays appear prematurely aged.

Ultraviolet radiation is composed of UVA, UVB, and UVC wavelengths. UVA can be divided into UVA I, or far UVA (340–400 nm), and UVA II, or near UVA (320–340 nm). They are longest, lowest-energy wavelengths and comprise 98% of the UV radiation on the earth. UVA rays are always present, independent of cloud cover or glass. Large amount of the annual UVA dose (52%) is received outside summer time. The photobiological effects of UVA rays are cumulative and leads to both epidermal (stratum corneum thickening, lower immune potential) and dermal changes (inflammation, impairment of connective tissue, lysozyme deposit, elastosis, effect on collagen and glycoaminoglycans). That both participate in skin photoaging and these cellular changes have also been demonstrated in vitro reconstructed skin models[2]. UVA rays reach in 80% to the dermo-epidermal junction and penetrate deeper in the papillary dermis. Acute effects are erythema, photoallergic and phototoxic reactions.

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Chronic effects are photoaging, immunosuppression[3] and potential photocarcinogenesis[4,5].

UVB rays (290–320 nm) are lower-energy wavelengths, and the amount of rays reaching earth levels varies according to season, time and cloud cover but they are only 2% of the UV radiation on the earth's surface. UVB are mostly absorbed by the epidermis as 70% are blocked by the stratum corneum. Acute effects are sunburn and related inflammation. Chronic effects are photoaging, immunosuppression and photocarcinogenesis. UVB damage is mainly at the DNA level inducing photolesions which can lead to gene modification and cell transformation. UVB rays are the major cause of sunburns which is a leading risk factor for melanoma and non-melanoma skin cancers[3–5]. Also, they can cause delayed skin pigmentation.

UVC rays (200–290 nm) are high-energy, short wavelengths, and are almost completely absorbed by the stratospheric ozone layer.

Mechanisms of Photoaging

Photoaging is a multi-system degenerative process that involves the skin and skin support system. The skin support system includes the bone, cartilage, and subcutaneous compartments which provide the architectural support for dermis, epidermis, and stratum corneum[6]. Bone demineralization begins at around 25 and leads to dulling of the facial features. The architecture of the cartilage of the face with bones, defines the shape of the face. The change occurs during pregnancy due to the relaxins that are secreted at high levels during the final trimester. Subcutaneous facial fat is removed and loss probably to lower growth hormone levels[7]. The viable epidermis and dermis are the essence of the skin. The structural destruction and loss of dermal collagen fiber bundles leads to wrinkling; irregular melanization leads to lentigines, poikiloderma, and melasma. In skin with long-term sun exposure, the ratio of melanocyte density is approximately twice that of nonexposed skin[8]. Prominent teleangiectasias lead to erythema, and loss of hydratation in stratum corneum leads to fine wrinkling. Photoaging affects the sun-exposed areas and is characterized clinically by fine and coarse wrinkling, roughness, dryness, laxity, teleangiectasia, loss of tensile strength and pigmentary changes. There is also an increase in development of benign and malignant neoplasms on photoaged skin. It is a cumulative process and depends primarily on the degree of sun exposure and skin pigment. The epidermis and dermis are both affected by UVB, but the dermis is also affected to a significant extent by UVA. It has long been thought that the majority of human photo--lesions due to UVB rays, now it is believed that UVA play a substantial role in photoaging. Because UVB is essentially completely absorbed in the epidermis, it has been important to understand that photoaging changes can be produced by UVA alone. Indeed, these changes have been produced in photoprotected skin by a small number of low-dose exposures of UVA radiation[9].

When the skin is chronic exposure to UV rays, the epidermis responds with hypertrophy. The stratum corneum thickenes, epidermis becomes acanthotic, and there is progressive dysplasia with cellular atypia, and anaplasia. Keratinocytes are irregular with a loss of polarity. Melanocytes are irregular with pockets of increased and decreased numbers. The Langerhans cell population in the epidermis is reduced and that contributed to an impaired immune response to antigen and skin cancer cells[10,11]. The roughness of photoaged skin is result of combination of changes in stratum corneum and changes in the glycosaminoglycan content of the dermis. With age there is a decrease in glycosaminoglycans in the dermis. In photoaged skin there is paradoxical increase in glycosaminoglycans when compared with intrinsically aged skin. But, there are deposited on the abnormal elastotic material rather than in the papillary dermis and that location may make them unavailable as a source of hydratation[12]. Photoaged skin display thickened basement membrane. Dermal changes in photoaged skin are reduction in collagen and precursors of types I and III collagen, a degeneration of elastic fibres, which are replaced in time by an amorphous mass and chronic inflammation with an increase in degranulated mast cells, macrophages, and lymphocytes. Blood vessels are dilated and tortuous[10]. In addition, because of the diminution of the collagen framework, the blood vessels are poorly supported; they can easy rupture, resulting in solar purpura. For a photochemical reaction to occur in the skin, ultraviolet radiation from the sun must be absorbed by chromophore, beginning a series of photochemic reactions. These chromophores are DNA, aromatic amino acids, 7-dehydrocholesterol, cytochromes, melanin and bilirubin[13,14]. These reactions can result in changes DNA, including oxidation of nucleic acids and modify proteins and lipids, resulting in changes in function. Their accumulation may result in skin cancer or photoaging changes[15]. DNA may absorb UVB, directly inducing changes between adjacent pyrimidine bases on one strand of DNA, although UVA can also generate thymine dimers[16,17]. DNA changes are constantly being repaired by nucleotide excision repair. Whenever repair is incomplete and damage to the genome is great, photo-damage may result[11]. Reactive oxygen species are an inherent part of the anabolism and catabolism of skin. When oxidative stress is increased, including high metabolic demands and outside forces such as sunlight, smoking, and pollution, protective controls may not be adequate and oxidative damage may occur. The most damage occurs from free radicals which are molecules or atoms with an unpaired electron. These molecules are extremely chemically reactive and short-lived. They react at the place where they are created and called reactive oxygen species – ROS[11]. Reactive oxygen species include superoxide anion, peroxide, and singlet oxygen. They can modify proteins in tissue to form carbonyl derivates which are accumulate in the papillary dermis of photo-damaged skin[18]. Small amount of UV radiation result in the induction of series of matrix metalloproteinase (MMP) including MMP-1, MMP-2, MMP-3, and MMP-9. These proteases

are capable of degrading the collagen framework of skin. At the same time procollagen synthesis is inhibited, perhaps by a mechanism related to degraded collagen[19]. Series of mitogen-activated protein kinases activated induction of transcription factor activation protein (AP-1). Levels of procollagen I protein are decreased, whereas MMP-1 and MMP-2 activity are increased. In addition, the transcription factor, nuclear factor-kB (NF-kB), is activated by UV radiation, which stimulates neutrophile attraction bringing neutrophile collagenase (MMP-8) into the irradiation site to future aggravate matrix degradation. Both AP-1 and NF-kB are activated by ROS. Oxidative stress can also increase elastin messenger RNA levels in dermal fibroblasts providing a mechanism for the elastotic changes found in photoaged dermis[20]. UVA can induce lipid peroxidation in membranes that can lead to altered membrane fluidity. The DNA in mitochondria can also be altered by oxidative stress[11].

Prevention of Photoaging

The best prevention of photoaging is UV protection. Reasonable sun consumption and education about sun protection is the best way. The thickening of the horny layer end epidermal hyperplasia, recognized as a natural form of protection against UV exposure, is caused mainly by UVB. The protection provided by sunscreens depends greatly on how they are applied and the person's activities after application. Sunscreen application is not as frequent as it should be, and the amount applied does not meet the recommended quantity of 2 mg/cm^2 of skin, on average, most sunscreen users apply only one third of the recommended amount[21,22]. Most people identify sun exposure to sunbathing at the beach and forget that they can be sun exposed when walking or practicing outdoor sports. In addition, sun reflection can be important by snow (30 to 80%), sand (6 to 25%), sea (20%) or grass (0.5 to 4%). There has been an important changing in sun protection over the last 20 years. People have started to use sunscreens to protect their skin not only against sunburn, but also against photodermatoses, such as polymorphus light eruption, and against the long-therm adverse effects, such as skin aging and skin cancer[23].

During recent years a more study about behavior in the sun is done. Also, in many countries education campaigns are starts to change attitude and promote sun protections. The major goals of these campaigns are in positive changes in the knowledge about sun exposure and sun protection. Younger people, who have not yet noticed the consequences of exaggerated sun exposure on their skin, use lower Spfs, they interrupted sunscreen use the last days of vacationing to achieve a more intense tan, and they sunbath during hottest hours of the day[24]. Encouraging photoprotection and intensive education are the leading preventative health strategy used by physicians involved in skin care.

REFERENCES

1. FISHER GJ, KANG S, VARANI J, BATA-CSORGO Z, WAN Y, DATTA S, VOORHEES J, Arch. Dermatol, 138 (2002) 1462. — 2. BERNERD F, ASSELINEAU D, Cell Dath Diff, 5 (1998) 792. — 3. MOYAL D, FOURTANIER A, KOLLIAS N, Exp Dermatol, 11 (2002) 28. — 4. STREILEIN JW, N Engl J Med, 325 (1991) 884. — 5. GILCHREST BA, Br J Dermatol, 127 (1992) 14. — 6. DRAELOS ZD, S Th Lett, 11 (2006). — 7. JOHANNSSON G, BENGTSSON BA, J Endocrinol Invest, 22 (1999) 41. — 8. CASTANET J, LANGTRY J, BURNS R, Arch. Dermatol, 133 (1997) 1296. — 9. LOWE NJ, MEYERS DP, WIEDER JM, LUFTMAN D, BORGET T, LEHMAN MD, J Invest Dermatol, 105 (1995) 739. — 10. LAERENCE, N, Dermatol Clin, 18 (2000) 99. — 11. PINNELL SR, J Am Acad Dermatol, 48 (2003) 1. — 12. BERNSTEIN EF, UNDERHILL CB, HAHN PJ, Br J Dermatol, 135 (1996) 255. — 13. KRUTMANN J, ELMETS CA, Photoimmunology (Blackwell Science, Oxford London, Edimburgh, Cambrige, 1995). — 14. NOLA I, KOSTOVIĆ K, KOTRULJA L, LUGOVIĆ L, MEŠTROVIĆ ŠTEFEKOV J, SJEROBABKI MASNEC I, Acta Clin Croat, 42 (2003) 119. — 15. TRAUTINGER F, Clin Exp Dermatol, 26 (2001) 573. — 16. YUNG AR, POTTER CS, NIKAIDO O, PARSONS PG, BOENDERS J, RAMSDEN JM, J Invest Dermatol, 111 (1998) 936. — 17. KIELBASSA C, EPE B, Methods Enzymol, 319 (2000) 436. — 18. SANDER CSC, J Invest Dermatol, 118 (2002) 618. — 19. VARANI J, SPEARMAN D, PERONE P, FLIGIEL SEG, DATTA SC, WANG ZQ, Am J Pathol, 158 (2001) 931. — 20. KAWAGUCHI Y, TANAKA H, ORADA T, KONISHI H, TAKAHASHI M, ITO M, Free Radic Bio Med, 23 (1997) 162. — 21. DIFFEY BL, GRICE J, Br. J. Dermatol, 137 (1997) 103. — 22. MARROT L, Br J Dermatol, 151 (2004) 1234. — 23. ROELANDTS R, Int J Dermatol, 37 (1998) 729. — 24. SJEROBABSKI MASNEC I, VODA K, M. ŠITUM, Coll Antopol, 31 (2007) 97.

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FOTOSTARENJE KOŽE

SAŽETAK

Između brojnih štetnih utjecaja okoline koji sudjeluju u starenju, dugotrajno i opetovano izlaganje ultravioletnim zrakama je najznačajnije te uzrokuje foto-starenje kože. Foto-starenje kože je degenerativni proces koji osim kože zahvaća i potporne strukture kože. To je kumulativni proces koji prvenstveno ovisi o stupnju izloženosti sunčevim zra-

kama i pigmentu kože. Promjenama koje nastaju pod utjecajem UVB zraka zahvaćeni su epidermis i dermis, a u dermis prodiru i UVA zrake. Dugo se smatralo da su za većinu lezija kože koje uzrokuje sunčevo zračenje odgovorne UVB zrake, danas se smatra sa UVA zračenje ima važnu ulogu u foto-starenju kože. Foto-starenje zahvaća kožu izloženu sunčevom zračenju te se manifestira pojavom naboranosti, crvenilom kože, suhoćom, gubitkom elasticiteta, pojavom teleangiectasia te pigmentnim promjenama. Izloženost UV zračenju povećava pojavu benignih i malignih novotvorevina kože na foto-eksponiranim dijelovima. Tijekom godina istraživanja postignut je napredak u razumijevanju procesa foto-starenja kože. UV zračenje uzrokuje kompleksne procese na specifičnim molekulama čiji odgovor uzrokuje oštećenja vezivnog tkiva kože. Smanjeno izlaganje UV zračenju i upotreba sredstava sa visokim UV filtrima može usporiti foto-starenje kože.