

New Option in Photoprotection

Ines Sjerobabski Masnec, Lena Kotrulja, Mirna Šitum and Sanja Poduje

Clinical Department of Dermatovenereology, University Hospital »Sestre milosrdnice«, Zagreb, Croatia

ABSTRACT

All the people are exposed to solar ultraviolet radiation. Exposure to sun with living in an oxygen-rich atmosphere causes unwanted photodamage. Sunburned skin is a leading risk factor for melanoma and non-melanoma cancers. UV exposure causes immunosuppression via multiple mechanisms in the skin. In this review the main topic is to mention new or alternative ways of photoprotection. Sunscreens are commonly used as protection against sun damage. They reduce the penetration of damaging solar UV wavelengths in skin by reflecting or absorbing them. Sunscreens are very valuable, but they have limitations. They have to be used properly to gain the full effect (application a little while before UV exposure, at frequent time points and in adequate amounts). Also, they have the problem of photoinactivation, which is the degeneration of the UV-filter due to exposure to UV rays resulting in the loss of absorbing capacity. Products with immune protection factor contain DNA-repair enzymes and antioxidants that may reduce mutations and enable the immune system to combat photodamage. The use of antioxidants and polyphenols may exert an anti-aging effect by preventing and even reversing sun damage. Adequate photoprotection is essential to control photocarcinogenesis and photoaging.

Key words: photoprotection, UV radiation, DNA repair enzymes, polyphenols

The sun emits visible light, infrared radiation, which is not considered harmful to humans, and UV rays. Less than 5% of the sunlight that reaches the earth's surface is ultraviolet radiation. All the people are exposed to solar ultraviolet radiation. Exposure to sun with living in an oxygen-rich atmosphere causes unwanted photodamage. Exposure of the skin to ultraviolet radiation, mainly its UVB and UVA components, results in erythema, edema, hyperpigmentation, sunburn cells, immunosuppression, photoaging and skin cancer.

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. Sunburned skin is a leading risk factor for melanoma and non-melanoma cancers.

Four processes control the penetration of UV rays into the skin.

Reflexion is diffuse and occurs at the level of cornified layer.

Diffusion is important for the cornified layer and for melanin that mainly diffuses short wavelengths.

Absorption occurs in the cornified layer where 70% of UVB is absorbed through the polar amino acid of keratin and urocanic acid, while melanin and carotenoids absorb UV rays and visible light.

The fraction of the beam escaped the three latter processes corresponds to transmission and penetrate the skin¹.

When skin is exposed to sunlight, UV rays are absorbed by skin molecules that then can generate harmful compounds, called reactive oxygen species or ROS, which are highly reactive molecules that can cause oxidative damage. ROS can react with cellular components like cell walls, lipid membranes, mitochondria and DNA, leading to skin damage and increasing visible signs of aging².

Mechanisms of UV rays exposure induced skin damage

Sun-exposed skin areas are 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 photolesions 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^{2,3}.

When the skin is chronic exposure to UV rays, the epidermis responds with hypertrophy. The stratum corneum thickens, 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^{4,5}. 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 hydration⁶. Chronic sun-exposed skin display thickened basement membrane. Dermal changes 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 tortuous⁴. In addition, because of the diminution of the collagen framework, the blood vessels are poorly supported; they can easily 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 photochemical reactions. These chromophores are DNA, aromatic amino acids, 7-dehydrocholesterol, cytochromes, melanin and bilirubin^{7,8}. 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 changes⁹. DNA may absorb UVB, directly inducing changes between adjacent pyrimidine bases on one strand of DNA, although UVA can also generate thymine dimers^{10,11}. DNA changes are constantly being repaired by nucleotide excision repair. Whenever repair is incomplete and damage to the genome is great, photodamage may result⁵.

Reactive oxygen species are an inherent part of the anabolism and catabolism of skin. When oxidative stress is increased, including high metabolic demands and out-

side 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 – ROS⁵. Reactive oxygen species include superoxide anion, peroxide, and singlet oxygen. They can modify proteins in tissue to form carbonyl derivatives, which are, accumulate in the papillary dermis of photodamaged skin¹².

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 collagen¹³. 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- κ B (NF- κ B), is activated by UV radiation, which stimulates neutrophil attraction bringing neutrophil collagenase (MMP-8) into the irradiation site to further aggravate matrix degradation. Both AP-1 and NF- κ B 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 dermis¹⁴. UVA can induce lipid peroxidation in membranes that can lead to altered membrane fluidity. The DNA in mitochondria can also be altered by oxidative stress⁵.

During the evolution the human organisms create different protective adaptation mechanisms to cope with the adverse effects on solar UV irradiation.

At tissue level, the tanning-response is probably the most beloved effect of UV rays. Augmented pigmentation, due to the increased melanin release and synthesis by melanocytes following UV irradiation, eventuate in increased protection by formation of an UV absorbing cap around the nucleus of keratinocytes^{15,16}. UVB and UVA exposure increases mainly epidermal but also dermal mitotic activity. Increased proliferation, and also differentiation, increases the thickness of the epidermis, resulting in an extension of the light path. Consequently, there is a decreased transmission of UV radiation to the vulnerable cells of the basal and suprabasal layers¹⁷.

The keratinocytes themselves have a few protection mechanisms at their disposal. UV-induced DNA-damage will specifically be removed by nucleotide excision repair. Here we can distinguish a very fast transcription coupled repair and a slower global genomic repair mechanism. Although the main protein players of these repair systems differ, the lesions will be removed via the same differential steps in both mechanisms. After recognition of the lesion, in global genomic nucleotide excision repair by the xeroderma pigmentosum group C protein and in transcription coupled nucleotide excision repair by blockage of RNA polymerase, nucleotide excision repair pro-

teins are recruited and a multiprotein complex will be formed^{15,18}. Then, the sequential action of helicases and endonucleases lead to the removal of the DNA segment containing the lesion and DNA polymerases fill the remaining gap. When damage is totally beyond repair, cells will go into apoptosis or programmed cell death to get rid of cells with unrepaired DNA-damage that may give rise to cancer^{19,20}.

Recent findings suggest a role for interleukin-12 (IL-12) in the response of skin cells to UV rays since addition of IL-12 to skin cells or skin results in a reduction of apoptosis or sunburn cell formation. IL-12 was also found to reduce cyclobutane pyrimidine dimers via induction of nucleotide excision repair²¹. Since UV-induced DNA-damage is an important trigger for UV-induced immunosuppression, the removal of DNA-damage via IL-12 may clarify its immunostimulatory effect^{21,22}.

The precise molecular mechanisms of a cell that regulates survival (via growth arrest and repair) and the switch to apoptosis, from a certain level of damage, is not unraveled yet.

Photoprotection

Sun avoidance is obviously the most efficient way of photoprotection, however not always practical and sometimes not possible.

Sunscreens

Sunscreens are commonly used as protection against sun damage, in the form of topical preparations that reduce the penetration of damaging solar UV wavelengths in skin by reflecting or absorbing them. When sunscreen is applied on the skin, special molecules called UV filters, cut down the amount of UV radiation that can penetrate the skin. These filters penetrate into the skin below the surface of the epidermis, the outermost layer of skin, leaving the body vulnerable to UV radiation.

Sunscreens are generally qualified with a sun protection factor (SPF), defined as the minimal erythema dose (MED) of protected skin divided by the MED of unprotected skin²³. Sunscreens reduced acute effects of UV rays like erythema solare or sunburn. Recent studies show that regular use of sunscreens can also protect against the chronic UV effects, as it reduces the carcinogenic risk²⁴, provides protection against immune suppression²⁵ and prevents photoaging²⁶.

Sunscreens are very valuable and should be part of the first line defense against UV, although they have their limitations. They have to be used properly to gain the full effect (application a little while before UV exposure, at frequent time points and in adequate amounts).

Sunscreens have to cope with the problem of photo-inactivation, which is the degeneration of the UV-filter due to exposure to UV rays resulting in the loss of absorbing capacity^{27–29}. Reactive intermediates of photostable filter substances may come into direct contact with the skin, where they may behave as photooxidants

or may also promote phototoxic or photoallergic contact dermatitis²⁹. Photostability of a sunscreen depends on the filter used, but also on the presence of other UV-filters or the solvent.

DNA repair enzymes

Importance of adequate DNA repair of especially cyclobutane pyrimidine dimers (CPD) lesions considering the cancer prone nature of cells with disrupted DNA³⁰.

T4 endonuclease V is a bacterial DNA repair enzyme which specifically recognizes CPD lesions in DNA and initiates excision repair by cleaving CPD. Subsequently, host cell enzymes remove the hanging lesion by exonuclease activity and refill the remaining gap using the opposite strand as a template. T4 endonuclease V, use as topical treatment in a liposome crème, increases the rate of repair of sunlight induced DNA-damage and reduced the incidence of pre-malignant actinic keratosis and basal cell carcinoma Treatment of patients with xeroderma pigmentosum with T4 endonuclease V lowered the rate of development of skin cancer^{31,32}.

T4 endonuclease V is a bacterial DNA excision repair enzyme that repairs cyclobutane pyrimidine dimers in DNA. As a topical treatment, it removes cyclobutane pyrimidine dimers in DNA in the epidermis of animals and human beings³³. Topical application of T4 endonuclease V for 1 year decreased the rate of development of actinic keratoses and basal cell carcinomas^{15,33}.

Photolyase, a DNA repair enzyme, decreased UVB-induced DNA damage cyclobutane pyrimidine dimers formation by 40% to 45% in human skin when applied immediately after UVB exposure³³, resulting in prevention of immunosuppression, erythema and sunburn formation¹⁵.

DNA repair performed by photolyases occurs via a mechanism known as 'photoreactivation' pointing to activation of the enzyme after capture of blue light (300–500 nm) photons. Photoreactivation results in partial repair of cyclobutane pyrimidine dimers and an increased resistance to UV irradiation. Photoreactivation might be expected to prevent sunburn and the appearance of skin cancer. Topical application of photolyase encapsulated into liposomes decreased the number of UV-induced thymine dimers¹⁵.

Antioxidants

Antioxidants are molecules that reduce ROS in the skin, which are generated by UV damage and lead to breakdown of collagen. The predominant antioxidants in skin are vitamin C and E. They neutralize reactive oxygen species before these can produce oxidative stress. Although the amount of vitamins, originating from nutrition, delivered to skin is limited, it appears to be possible to achieve a higher level of photoprotection by using topical vitamins³⁴. Antioxidants have been administered orally and topically for photoprotection in combination with sunscreens to enhance efficacy. Tocopherol (vitamin E) and ascorbate (vitamin C) reduce erythema, and sun-

burn cell formation. Vitamin E also reduces chronic UVB-induced photodamage and photocarcinogenesis³⁵. Their no correlation was found between epidermal content of vitamin E and MED, repeated ingestion of a combination of tocopherol and ascorbate increased the threshold for erythema induction significantly³⁶. Topical vitamin C has shown to protect the skin from UV damage caused by prolonged sun exposure by reducing the amount of free radical formation and/or sunburn cells. Exposure to UV light has also shown to decrease the naturally occurring vitamin C levels in the skin, thus topical application of vitamin C restores these photoprotectant levels. Other studies also suggest that vitamin C may play a part in the collagen biosynthetic pathway by activating collagen metabolism and dermal synthesis of elastic fibres³⁵. Topical vitamin C 5% cream applied for six months led to clinical improvement in the appearance of photoaged skin with regard to firmness, smoothness, and dryness compared to vehicle³⁷. Topical vitamin C stimulates the collagen-producing activity of the dermis³⁸.

Polyphenols

Polyphenols, such as flavonoids, are efficient antioxidants due to the presence of hydroxyl groups. However, those hydroxyl groups bring about a pronounced first pass effect with a low bioavailability of free flavonoids as a consequence³⁹. According to recent data, the absorption of flavonoids might be better than originally believed^{36,40}.

Green tea polyphenols exhibit anti-inflammatory activity, causing inhibition of UV-induced skin erythema, edema, and a decrease in the number of sunburn cells⁴¹. Epigallocatechin-3-gallate (EGCG) is the main polyphenol in green tea and was found to induce differential effects between tumor cells and normal cells. EGCG was reported to induce differentiation and proliferation in epidermal keratinocytes⁴². Topical application and oral administration resulted in a strong inhibition of the sunburn response and a reduction in sunburn cell formation⁴³. EGCG inhibits UVB induced erythema, edema, depletion of the antioxidant enzyme system, IL-10 and IL-12 production and skin carcinogenesis⁴¹. Effects on photocarcinogenesis include a decrease in tumor burden, inhibition on the formation and size of malignant and non-malignant tumors and tumor regression in mice with established tumors. Effects of green tea polyphenols in photoaging include the inhibition of UVB induced expression of matrix metalloproteinases and reduction of UVB-induced collagen cross-linking³³.

Genistein is a soybean isoflavone with a wide range of antioxidant and anticarcinogenic effects in skin. It is a potent antioxidant and phytoestrogen presented also in ginkgo biloba extract, oregano, and sage⁴¹. Clinical studies indicate that genistein has antiphotocarcinogenic and antiphotphotoaging effects for both UVA and UVB. Genistein blocks UVB-induced sunburns in humans as well as psoralen-UVA (PUVA) induced molecular damage in mice. In addition, when applied after UVB exposure,

genistein provides significant comfort and slightly reduces erythema or sunburn⁴⁴. Genistein also had a powerful potential to reduce the inflammatory edema reaction and suppressed contact hypersensitivity induced by moderate doses of solar stimulated UV radiation⁴⁵. Recent study has demonstrated that genistein potentially minimizes the detrimental effects of UVB irradiation in human skin by preserving cutaneous proliferation and repair mechanics, inhibition of oxidative and photodynamic damage to DNA⁴¹.

Results indicate that flavonoids are promising candidates for cancer prevention, and in that matter they might be successful photoprotectants³⁹.

The plant extract *Polypodium leucotomos* (PL) increases the UV dose required for immediate pigment darkening, minimal erythema dose, minimal melanogenic dose and minimal phototoxic dose and down-regulates psoralen-UVA-induced phototoxicity and pigmentary and histological changes³³. In a study involving 26 patients with polymorphous light eruption and two patients with solar urticaria, oral PL has been reported to improve photosensitivity⁴⁶.

The mechanism of action of extract *Polypodium leucotomos* (PLE) is fairly complex. PLE acts as a scavenger to mop up free radicals and reactive oxygen species (ROS), particularly superoxide anions. PLE inhibits the depletion of Langerhans cells and reduces the number of sunburn cells. PLE protects DNA by inhibiting the formation of cyclobutane pyrimidine dimers induced by UVB radiation. PLE preserves skin tissue structure by inhibiting the infiltration of mast cells into skin which leads to inhibition redness, inflammation and itching.

It should be considered as another layer of protection and used in conjunction with a good sunscreen and protective clothing.

PLE can also be used as a chemophotoprotector against PUVA-induced skin phototoxicity. Extensive PUVA treatment results in premature aging changes in the skin and can increase the chance of skin cancer. Fair skinned individuals or those with previous sun or radiation damage are most at risk. In clinical trials, PLE has proven to be the first oral agent effective in reducing the harmful side effects of PUVA treatment⁴⁷.

Polypodium leucotomos blocked the deleterious effect of UV irradiation both in vivo and in vitro. The molecular basis of photoprotection relies on its ability to inhibit free radical generation, prevent photodecomposition of both endogenous photoprotective molecules and DNA, and prevent UV-induced cell death. Its complete loss of toxicity combined with its multifactor protection makes it a valuable tool not only for direct photoprotection, but also as an efficacious adjuvant to phototherapy of various skin diseases⁴⁸.

Conclusion

In the past, carcinogenesis of the skin was mainly attributed to UVB radiation and the most sun-protection measures were geared toward blocking UVB rays. The

recently research has revealed the importance of UVA exposure in skin cancer and premature photoaging². UVA is just as dangerous as UVB, and new developments are focused on UVA protection, immune protection, and public awareness⁴⁹. The main topic of this review is to mention new or alternative ways of photoprotection.

UV exposure causes immunosuppression via multiple mechanisms in the skin including via urocanic acid. Any exposure to the sun disables the immune system's ability to fight skin cancer. The DNA mutations formed during childhood tanning or burning may cause skin cancers in adulthood, especially with more and more sun exposure. Products with immune protection factor contain DNA-repair enzymes and antioxidants that may reduce muta-

tions and enable the immune system to combat photodamage. Topical treatment with repair enzymes may be a promising avenue for after sun protection of UV-irradiated skin. If topical immune protection factors prove to be safe but only moderately effective, systemic medications targeted at immunomodulation of the skin and immunity-driven technology will be a future direction in skin cancer prevention and treatment.

The use of antioxidants both orally and topically, as preliminary studies demonstrate may exert an anti-aging effect by preventing and even reversing sun damage.

Adequate photoprotection is essential to control photocarcinogenesis and photoaging.

REFERENCES

- BEDANE C, Optical properties of skin, accessed 22.10.2009. Available from: <http://www.clubdermaweb.com>.
- SJEROBABSKI MASNEC I, PODUJE S, Coll Antropol, 32 (2008) 177.
- LOWE NJ, MEYERS DP, WIEDER MJ, LUFTMAN D, BORGET T, LEHMAN MD, J Invest Dermatol, 105 (1995) 739.
- LAERENCE N, Dermatol Clin, 18 (2000) 99.
- PINNELL SR, J Am Acad Dermatol, 48 (2003) 1.
- BERNSTEIN EF, UNDERHILL CB, HAHN PJ, Br J Dermatol, 135 (1996) 255.
- KRUTMANN J, ELMETS CA, Photoimmunology (Blackwell Science, Oxford, London, Edinburgh, Cambridge, 1995).
- NOLA I, KOSTOVIĆ K, KOTRULJA L, LUGOVIĆ L, MEŠTROVIĆ ŠTEFEKOV J, SJEROBABSKI MASNEC, Acta Clin Croat, 42 (2003) 119.
- TRAUTINGER F, Clin Exp Dermatol, 26 (2001) 573.
- YUNG AR, POTTER CS, NIKAIKO O, PARSONS PG, BOENDERS J, RAMSDEN JM, J Invest Dermatol, 111 (1998) 936.
- KIELBASSA C, EPE B, Methods Enzymol, 319 (2000) 436.
- SANDER CSC, J Invest Dermatol, 118 (2002) 618.
- VARANI J, SPEARMAN D, PERONE P, FLIGIEL SEG, DATTA SC, WANG ZQ, Am J Pathol, 158 (2001) 931.
- KAWAGUCHI Y, TANAKA H, ORADA T, KONISHI H, TAKAHASHI M, ITO M, Free Radic Bio Med, 23 (1997) 162.
- VERSCHOOTEN L, CLAERHOUT S, VAN LAETHEM A, AGOSTINIS P, GARMYN M, J Photochem Photobiol, 82 (2006) 1016.
- AGAR N, YOUNG AR, Mutat Res, 57 (2005) 121.
- LEE JH, AN HT, CHUNG JH, KIM KH, EUN HC, CHO KH, Photodermatol Photoimmunol Photomed, 18 (2002) 253.
- COSTA RM, CHIGANCAS V, GALHARDO RS, CARVALHO H, MENCK CF, Biochimie, 85 (2003) 1083.
- VAN LAETHEM A, CLAERHOUT S, GARMYN M, AGOSTINIS P, Int J Biochem Cell Biol, 37 (2005) 1547.
- COTTON J, SPANDAU DF, Radiat Res, 147 (1997) 148.
- SCHWARZ A, SLANDER S, BERNEHURG M, BOHM M, KULMS D, VAN STEEG H, GROSSE-HEITMEYER K, KRUTMANN J, SCHWARZ T, Nat Cell Biol, 4 (2002) 26.
- SCHWARZ A, MAEDA A, KERNEBECK K, VAN STEEG H, BEISSERT S, SCHWARZ T, J Exp Med, 201 (2005) 173.
- LOWE NJ, Dermatol Clin, 24 (2006) 9.
- FOURTANIER A, Photochem Photobiol, 64 (1996) 688.
- SERRE IJ, CANO P, PICOT MC, MEYNADIER J, MEUNIER L, J Am Acad Dermatol, 37(1997) 187.
- TSUKAHARA K, MORIWAKI S, HOTTA M, FUJIMURA T, SUGIYAMA-NAKAGIRI Y, SUGAWARA S, KITAHARA T, TAKEMA Y, Biol Pharm Bull, 28 (2005) 2302.
- MAIER H, SCHAUBERGER G, BRUNNHOFER K, HONIGRMANN H, J Invest Dermatol, 117 (2001) 256.
- DAMIANI E, ROSATI L, CASTAGNA R, CARLONI P, GRECI L, J Photochem Photobiol B, 82 (2006) 204.
- GASPAR LR, MAIA CAMPOS PM, Int J Pharm, 13 (2006) 123.
- YOU YH, LEE DH, YOON JH, NAKAJIMA S, YASUI A, PFEIFER GP, J Biol Chem, 276 (2001) 44688.
- YAROSH DB, CANNING MT, TEICHER D, BROWN DA, Mutat Res, 571 (2005) 57.
- YAROSH D, KLEIN J, O'CONNOR A, HAWK J, RAFAL E, WOLF P, Lancet, 357 (2001) 926.
- KULLAVANIJAYA P, LIM HW, J Am Acad Dermatol, 52 (2005) 937.
- LIN JY, SELIM MA, SHEA CR, GRICHNIK JM, OMAR MM, MONTEIRO-RIVIERE NA, PINNELL SR, J Am Acad Dermatol, 48 (2003) 866.
- EBERLEIN-KONIG B, RING J, J Cosmet Dermatol, 4 (2005) 4.
- SIES H, STAHL W, Ann Rev Nutr, 24 (2004) 173.
- HUMBERT PG, HAFTEK M, CREIDI H, LAPIERE C, NUSGENS B, RICHARD A, Exp Dermatol, 12 (2003) 237.
- NUSGENS BV, HUMBERT P, ROUGIER A, RICHARD A, LAPIERE CM, Eur J Dermatol, 4 (2002) 39.
- BIRT DF, HENDRICH S, WANG W, Pharmacol Ther, 90 (2001) 157.
- ROSS JA, KASUM CM, Ann Rev Nutr, 22 (2002) 19.
- AFAQ F, MUKHTAR H, Exp Dermatol, 15 (2006) 678.
- HSU S, BOLLAG WB, LEWIS J, HUANG Q, SINPH B, SHARAWY M, YAMAMOTO T, SCHUSTER G, J Pharmacol Exp, 306 (2003) 29.
- ELMETS CA, SINGH D, TUBESING K, MATSUI M, KATIYAR S, MUKHTAR H, J Am Acad Dermatol, 44 (2001) 425.
- WEI H, SALADI R, LU Y, J Nutr, 133 (2003) 3811.
- WIDYARINI S, SPINKS N, HUSBAND AJ, REEVE VE, Photochem Photobiol, 74 (2001) 465.
- CACCIALANZA M, PERCIVALLE S, PICCINNO R, BRAMBILLA R, Photodermatol Photoimmunol Photomed, 23 (2007) 46.
- MIDDELKAMP-HUP MA, PATHAK MA, PARRADO C, J Am Acad Dermatol, 51 (2004) 910.
- GONZALEZ S, ALONSO-LEBRERO JL, DEL RIO R, JEAN P, Drugs Today (Barc), 43 (2007) 475.
- SJEROBABSKI MASNEC I, VODA K, ŠITUM M, Coll Antropol, 31 (2007) 97.

I. Sjerobabski Masnec

Department of Dermatovenerology, University Hospital »Sestre milosrdnice«, Vinogradska 29, Zagreb, Croatia
e-mail: ines@kbsm.hr

NOVE MOGUĆNOSTI FOTOPROTEKCIJE

SAŽETAK

Svi ljudi su izloženi sunčevom ultravioletnom zračenju. Izlaganje suncu, uz život u atmosferi bogatom kisikom, dovodi do neželjenih oštećenja kože. Izrazito osunčana koža je vodeći faktor rizika za nastanak maliginog melanoma i ostalih tumora kože. Ultravioletno zračenje, različitim mehanizmima, uzrokuje imunosupresiju u koži. U ovom preglednom radu željeli smo upozoriti na nove i alternativne mogućnosti zaštite od sunca. Sredstva za zaštitu od sunca koriste se za prevenciju i smanjenje oštećenja uslijed izlaganja ultravioletnim zrakama. Ona smanjuju prodiranje ultravioletnih zraka u kožu tako da ih reflektiraju i apsorbiraju. Korisna su, ali imaju ograničenja. Moraju se koristiti na pravilan način da bi polučili efekt (aplikacija neposredno pred izlaganje suncu, učestala aplikacija u dovoljnoj količini). Također, dolazi do inaktivacije ultravioletnih filtera tijekom izlaganja ultravioletnim zrakama i gubitka sposobnosti apsorpcije. Proizvodi koji sadrže imunološki zaštitni faktor sadrže DNK reparirajuće enzime i antioksidanse. Sposobni su smanjiti mutacije te povećavaju sposobnosti imunološkog sistema u obrani od foto-oštećenja kože. Upotreba antioksidansa i polifenola može prevenirati i smanjivati oštećenja uslijed izlaganja ultravioletnim zrakama te unaprijediti zaštitu od starenje pod utjecajem sunca. Pravilna zaštita od sunca neophodna je u kontroli foto-karcinogeneze i foto-starenja.