Effects of visible light in skin and hair and novel strategies of protection against photo damage

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Abstract

The interaction of light with skin is of fundamental importance for human health, however, excess of sun exposure causes damages to the skin and to the hair and consequently can accelerate skin aging, and cause skin cancer. It can also make your hair look ugly. The amount of light needed to maintain normal functioning of the epidermis and dermis, without harming them, certainly depends on the characteristics of each individual's skin. A biochemical route that critically depends on sun exposition is the activation of vitamin D. The continuous raise in the level of skin cancer, indicates that in addition to protecting the skin by blocking UVB and UVA, it is necessary to also consider the effect of other wavelength regions, such as the visible light. Since blocking visible light is a lot more challenging than blocking UV, it is important to consider other strategies of skin care, including maintenance of redox homeostasis. In this review, we will consider the basic photochemical reactions that are induced after light is absorbed by molecules present in the skin and in the hair, in each region of the sun spectrum, emphasizing the concepts of photosensitization and of oxidizing species, such singlet oxygen and free radicals. We will also look at the most important defenses against the redox misbalance and the biochemical routes important for the construction of the skin barrier function. We aim to discuss the opportunity for development of better strategies to prevent skin photoaging, as well as, hair damage.

Keywords: Skin, Hair, Sun protection, Free radicals, Singlet oxygen

Introduction

To understand the effects of light on the skin and hair, we need to understand how light interacts with these surfaces. Initially, we should understand that the earth stratosphere filters light emitted by the sun. The spectrum of light reaching the biosphere has different amounts of ultraviolet (UVC, UVB, UVA), visible (VIS) and infrared (IR). 100% of UVC ($220<\lambda<280$ nm), 95% of UVB ($280<\lambda<320$nm) and only 5% of UVA rays ($320<\lambda<400$ nm) are filtered before reaching the biosphere. Therefore, the spectral regions that are important to consider in relation to living organisms are the UVB/UVA regions and the visible and infrared regions. Note also that the percentage of light reaching our skin is 5% of UV, 45% of visible and 50% of infrared. Secondly, we need to resort to the first law of photochemistry that says: light must be absorbed by a compound for a photochemical reaction to occur. In other words, it is essential to know the molecules that absorb light at a given wavelength in order to understand the effect of light on the biological system. Thirdly, it is important to consider that when a molecule absorbs light it is taken to an excited state, which is intrinsically more reactive than the fundamental state, since the orbital configuration in the excited states favors many type of chemical reactions (Figure 1A). The subsequent production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) is also a very important factor of the effect of sun exposure. Interestingly, the wavelengths in the visible region are responsible for the majority of the radicals formed in human skin. Visible light is absorbed by flavins, and other vitamins, porphyrins and melanin, and the production of ROS/RNS occurs mainly by photosensitization.

Cutaneous and Capillary Photo Damage]

Effects in the skin

After the absorption of photons in the UVB, some nucleic acid bases have a tendency to react chemically. Adjacent thymines (nitrogenous pyrimidine bases) undergo photochemical processes forming mainly pyrimidine dimers (CPD) and 6-4 photoproducts (6-4 P), which lead to DNA damage and consequently to mutations, which are mainly transversion of cytisine to thymine (CT). Depending on the gene in which the mutations occur, it can lead to a lack of control of fundamental cell activities. In the case of UVA, proteins and nucleic acids do not directly absorb this radiation; however, endogenous chromophores such as riboflavin and melanin do, forming excited triplet species that can form singlet oxygen by photosensitization (Figure 1A). Singlet oxygen is an excited species of oxygen that is highly reactive against electron rich molecular sites damaging lipids, proteins and nucleic acids. In the reaction of singlet oxygen with DNA, the main target is guanine, forming usually 8-Oxo-2'-deoxyguanosine (8-oxo-dG) and leading to the thymine to guanine transversion (TG). In the case of proteins, the main targets are the aminoacids, histidine, cysteine, methionine, tryptophan and tyrosine. In the case of

lipids, oxidation of the double bond leads to the formation of a hydroperoxide and lipid truncated aldehydes, altering the properties of the membranes and causing its leakage respectively.

Although it does not cause direct damage to DNA, in recent years' visible light has consolidated itself as an important agent that causes oxidative skin damage. Not only oxidative harms caused by the association of VIS radiation with UV and IV were identified, as well as the darkening of some skin types, showed that VIS is capable of inducing pigmentation.10 Zastrow and his group (2009) identified that VIS is responsible for 50% of the total skin oxidative load, which is a valuable information especially when it is taken into account that photosensitized melanin can generate singlet oxygen. Besides, visible light can form radicals derived from photosensitive compounds in this band and thus indirectly cause the DNA strand to split. This is the case of methyl cobalamin which, when irradiated with a green light (510-560 nm), produces a methyl radical capable of abstracting a hydrogen atom from the sugar-phosphate skeleton and after breaking the chain.

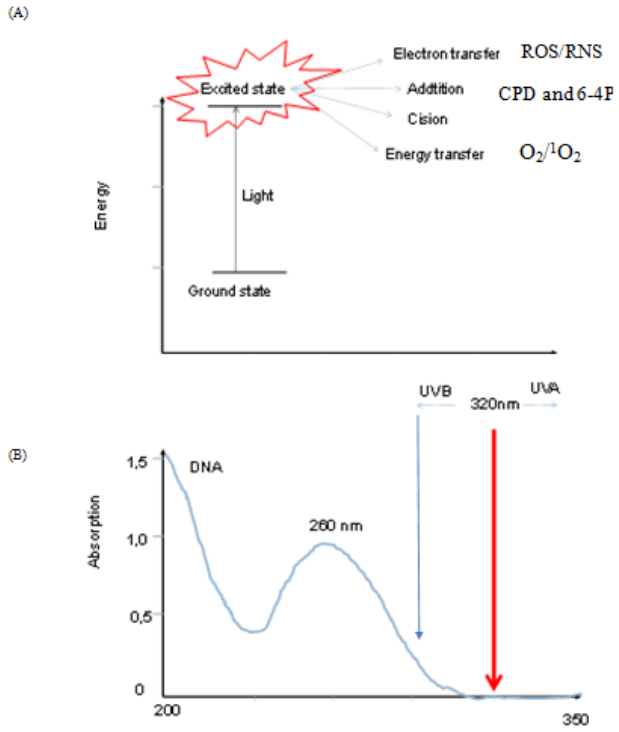

Figure1: *A) Ground and excited state energy diagram showing the increased reactivity of the excited states formed by light absorption, where ROS and RNS are reactive oxygen and nitrogen species, respectively, formed by many processes including, radical chain reactions. CPD and 6-4P are pyrimidine dimers and 6-4 that are formed by exciting the DNA pyrimide bases that engage in cyclo addition reactions and 1O2 is the excited state species of oxygen, formed by energy transfer reactions with the triplet excited state species. B) Absorption spectrum of a DNA molecule showing that it absorbs radiation in the UVB and does not absorb efficiently in the UVA and above. See reference 155 for the definition of type I and type II photosensitized oxidation reactions.*

Another fact to be considered is that the interaction of light with the skin is not always harmful. On the contrary, several fundamental processes for the health of the skin depend on the interaction with light. A very well-known example is the endogenous production of vitamin D3, which occurs in the skin irradiated with UVB. Therefore, one should avoid excesses of irradiation, but do not overdo the protection. In order to generate the active vitamin D (calcitriol), a multi-step mechanism is required (Figure 2). Initially, the precursor 7-dehydrocholesterol (7- DHC) absorbs photons coming from UVB radiation (300 nm) being converted to previtamin D3. By a non-enzymatic process, previtamin D3 is converted to calciferol molecule (Vitamin D3). This molecule reaches the blood circulation and, by the action of the enzyme 25-hydroxylase is metabolized to 25- hydroxy-vitamin D (25-OH or calcidiol) in liver, which is the major circulating form of vitamin D. At the kidneys, calcidiol is submitted to another hydroxylation, generating the biologically active form 1,25-dihydroxy-vitamin D (calcitriol). Vitamin D2 can also be hydroxylated in liver and generate calcidiol.

The formation of vitamin D3 is influenced by many factors such as skin pigmentation, sunscreen use, altitude, latitude, season, hour of the day, air pollution, and others. If correctly applied, sunscreen use reduces significantly vitamin D synthesis. Besides, the vitamin D intake involves the consumption of foods that provide the two forms of vitamin D (D2 and D3): Fruits and vegetables contain small amounts of vitamin D2, while fishes such as salmon, herring and mackerel, and fish oils contain vitamin D3. However, usually it is not easy to reach suitable vitamin D intake from these natural sources.15 These two forms of vitamin D can be also found as supplements; however, some clinical studies showed that vitamin D3 supplementation is more efficacious than vitamin D2 to raise calcidiol serum levels.

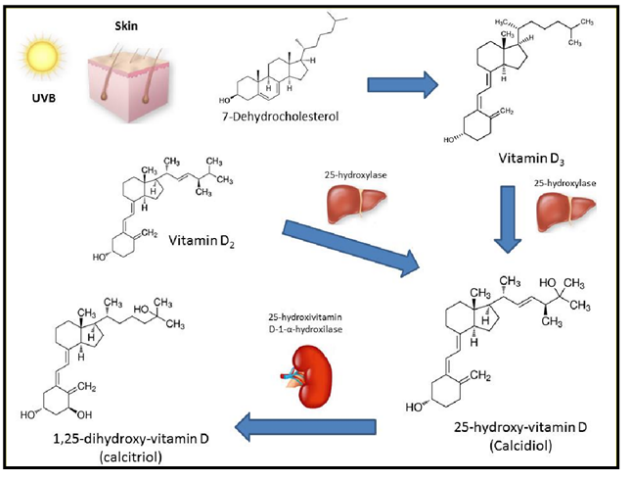

Figure2: *Endogenous synthesis of vitamin D3 and its metabolism, generating the active form, calcitriol.*

There is an extensive amount of literature related to vitamin D roles in the bone and in the mineral metabolism. More recently, non-classic actions of vitamin D have been proposed, associated to physiological and pathological functions, such as cardiovascular health, muscle health, cancer prevention,

obesity and skeletal muscle function, development of multiple sclerosis, diabetes and metabolic syndrome and immunity, anti-inflammatory effects, however some of them still require further studies. Some evidence indicates that vitamin D also presents an important role in epidermal differentiation and skin protection however, the mechanisms are not completely known. Due to its importance to the skin, epidermal keratinocytes present some enzymes which can convert calcidiol to calcitriol locally. During epidermal differentiation occurs the keratinocytes cornification process, which is responsible for the formation of the epidermis-stratified structure. In this peculiar process of programmed cell death, the basal layer keratinocytes undergo a differentiation process becoming corneocytes in the outer layers of the epidermis. In the spinous layer, cells reinforce their cytoskeletal keratin filaments network and interact through desmosomes to withstand the physical trauma. In the granular layer, keratinocytes become flatter and express proteins (such as profilaggrin and loricrin) which form the keratohyalin granules. In this layer, also occur lipids storage in lamellar bodies. Keratinocytes lose their organelles, including the nucleus, becoming corneocytes in stratum corneum. Recently it was reported that the cell death mechanism involved in cornification can not be considered the classical apoptotic one, and caspase-14 is the main effector molecule.

Caspases are enzymes from the cysteine-aspartic proteases family. They present a single catalytic mechanism that involves the cysteine's nucleophilic thiol group and they recognize a sequence of four aminoacids P4 -P3 -P2- P1, while the cleavage usually occurs at P1, which usually is an aspartate residue. These proteases are expressed as proenzymes with sizes varying from 30 to 50 kDa, and presents three domains (the NH2-terminal, the large subunit and the small subunit, presenting about 20 and 10 kDa, respectively). These proenzymes exhibit small or absence of catalytic activity. Their activation process involves domains processing and association between large and small subunits, forming a heterodimer. This activation occurs through the action of another protease or autocatalysis. Caspases are known for their role in the apoptotic process, acting as initiator caspases (caspases -2, -8, -9 and -10) or effector caspases (caspases -3 and -7); in inflammation (caspases -1, -4 and -5); and in the proliferation processes and cellular differentiation (caspase-14). The effector caspases are known by their role in the proteolytic activity in apoptosis (Caspases -6 and -7, which are activated after caspase-3). They have a short prodomain (20-30 amino acids), they are dimeric, so that the proteolytic cleavage is essential for their activation. Activation occurs through limited proteolysis by the initiator caspases. The initiator caspases are the arms of the apoptotic machinery, i.e., they convert the apoptotic signaling in proteolytic activity. The initiator caspases are characterized by a long prodomain (>90 aminoacids), they are monomeric and the dimerization is essential for their activity. The initiator caspases contain two regions of protein-protein interactions: the CARD (caspase recruiting domain) and DED (death effector domain). These regions interact with similar regions present in oligomerized adapter proteins, making several initiator caspases to be close and facilitating self-activation. They integrate the signal in response to binding to death receptor (apoptosis through the extrinsic pathway with the participation of caspases- 8 and -10 and probably caspase-2) and in response to cellular stress (intrinsic pathway by caspase-9 action). There are also caspases are involved in inflammation: Caspase-1 is a cytokine activator since it is involved in the pro-IL maturation. Caspases 4 and 5 are poorly studied but are considered cytokine activators since they present high protein identity to caspase-1, domain organizational similarity and comparable substrate specificity.

Caspase-14 presents a catalytic site similar to the cytokine activator caspases (1, 4 and 5), and a short prodomain which is characteristic of effector caspases. Thus, caspase-14 exhibit substrate specificity very much alike the cytokine activator caspases however, its activation mechanism is similar to the apoptosis effector caspases, requiring proteolytic cleavage. Caspase- 14 cleavage during keratinocyte differentiation has been observed, generating the 11 and 17 kDa fragments, and this cleavage occurs between isoleucine and lysine residues (Ile152/Lys153), differently from other caspases that are cleaved at the aspartate residue. As a result, it is concluded that caspase-14 is not cleaved by another caspase *in vivo*, even though other caspases are not activated during the epidermal differentiation process. It is known that this cleavage site is common to calpain substrates and that cleaved caspase-14 is active, *in vitro*, in the presence of cosmotropic salts, such as sodium citrate. However, there is no information yet about the physiological protease which cleaves caspase-14 and how its dimerization occurs *in vivo*.

Unlike apoptotic caspases that are expressed in several tissues, caspase-14 is expressed only in differentiated and cornified epidermal layers and hair follicle. In stratum granulosum, caspase-14 is associated to the keratohyalin granules and to corneocytes, being found in the cytoplasm with association to corneodesmosomes and nuclear remainders. Inside cells, caspases localize in different compartments, including the nucleus. It has been suggested that nuclei caspase-14 accumulation is part of the differentiation process, as well as, caspase-14 role in the nuclear degradation process during cornification. It is believed that caspase-14 activation occurs at the interface between the granular and cornified epidermal layers or in the early cornification steps.

The active caspase-14 is involved in profilaggrin cleavage to filaggrin. Filaggrin acts in the maintenance of cornified cells structure in external skin layers since this molecule adds intermediate keratin filaments to other intermediate filaments in tight bundles that, after collapse, promote significant changes in the morphology of the epidermal cells, from elliptical to flat. Then, filaggrin monomers are degraded releasing free amino acids that act as natural moisturizing agents of the stratum corneum, since they are essential for the water retention, osmolarity and cornified layer flexibility. As a result, caspase-14 deficiency/low activation is related to low filaggrin concentration in stratum corneum, as well as, their degradation products such as urocanic acid and pyroglutamic acid, and consequently low moisturizing levels. Besides, this

modified skin homeostasis causes a misbalance in bacterial communities, been frequently associated to inflammatory diseases including psoriasis, atopic dermatitis, and acne and chronicle ulcerations. Interestingly there is a cross talk between caspase-14 action and Vitamin D, since vitamin D3 induces Caspase-14 expression and enhances Caspase-14 Processing Cultures. Therefore, it seems that skin barrier also depends of a bit of sun exposition.

Effects in the hair

Regarding hair, although hair always grow and time allows visual recovery in people with active hair follicles, it is important to understand hair damage, especially for the market interested in people's beauty. Many events occur after sun exposure on the hair fibers. In fact, both UV and in the VIS photons interact with hair and can cause damage. Our research group recently demonstrated that visible light generates singlet oxygen in hair causing damage to melanin and keratin. The decrease in the mechanical resistance of the hair fibers is one of the most notorious effects after exposure to solar radiation, this possibly reflects the breaking of disulfide, R-S or S- S bonds in addition to the formation of carbonyl and amine groups by protein degradation. Importantly, since the mechanical resistance of the hair is largely associated with the cortex (the layer corresponding to 90% of the capillary weight), this is possibly the most affected layer. Studies with the application of VIS, UV, IR, and total radiation have shown different results for the damage observed. Importantly, for each study, the hair colors reflect different types of melanin and, consequently, different responses to irradiation. Richena and his group observed that the combined effects of VIS radiation with IR, compared to total radiation in hair without pigment were more responsive to discoloration. Hoting and co-authors reported that strands with more pheomelanin are responsive to all types of radiation except IR, while black hair is more affected when exposed to VIS or total radiation. Another study, the causal effect of depigmentation of natural threads was mostly thanks to VIS radiation, while UV radiation was responsible for mechanical damage.

Mechanically, the loss of pigments from hair exposed to sunlight is possibly the result of the conversion of benzothiazine to benzothiazole in wicks with pheomelanin and oxidation of the indolquinone group in hair with eumelanin. The duality of function is inherent to different types of melanin since the photoreactive characteristic of these molecules makes it capable of originating hydrogen peroxide and superoxides, while the photoprotective capacity can decrease the amount of reactive free radicals. The protective role of melanin becomes evident mainly in more accentuated color damage in chemically dyed hair, in which radiation doses to exert the photobleaching effect on these hairs are twenty times lower compared to the same effect on natural hair. The difficulty in standardizing study parameters such as dose, irradiance, temperature, and relative humidity, results in contradictions in the literature about the specificity of effect on the capillary fiber at the UV, VIS, and IR ranges. However, harmful changes in colour, protein content, decreased strength and elasticity are frequent in hair subjected to solar radiation.

Relevance of Antioxidants to Light-Related Damage

Cellular homeostasis is maintained due to the balance between the rate of formation of ROS and RNS and the rate of suppression of these species. Excessive exposure to light is one of the conditions that can cause a large redox imbalance, while antioxidants react with ERO and ERN and singlet oxygen. If there is DNA damage, the imbalance can lead to cancer. Inflammation is another cellular defense mechanism, but it can worsen the scenario, increasing oxidative imbalance. Antioxidant is a substance that when present in low concentrations in relation to the oxidizable substrate significantly or totally decreases the oxidation reaction, being able to act by several mechanisms: (a) Preventing the generation of oxidants, which includes preventing light absorption or anti-inflammatory activity; (b) suppression, chelation and removal of oxidants; (c) repair of damage and excretion of toxic products and, (d) adaptive responses. Since the 1970s, the concept of endogenous photoprotection has been discussed and the term “systemic photoprotection” or “nutritional” is increasingly explored in the context of food-borne photochemical chemopreventives. In addition to the antioxidant effects mentioned before, compounds with the ability to suppress cell damage and responses (such as inflammation) or even modulate stress-dependent cell signaling pathways are also included in this class. Applicability in the context of endogenous photoprotection ranges from micronutrients with direct activity against prooxidants, antioxidant enzyme constituents, to the action of carotenoids, polyphenols, vitamins E and C as endogenous photoprotectors. It is worth mentioning that pharmacokinetic parameters such as absorption, metabolism, and bioavailability must be considered with caution, considering that these compounds have minimal concentration so that the protective effect is possible.

Clinical studies have already proven skin photoprotection based on the consumption of lycopene derived from tomatoes and cocoa-rich in flavonoids. Another product with potential use in systemic photoprotection is the dye of American origin and approved by the FDA bixin apocarotenoid, a derivative of lycopene by oxidative cleavage and widely used in the world as a seasoning, food coloring and even in cosmetic and pharmaceutical inputs. In addition to food use, apocarotenoid bixin is used in topical preparations for therapeutic healing of wounds, mouth ulcers, and other skin injuries that have direct damage to the skin barrier. They also provide systemic protection against xenobiotics, including methylmercury and carbon tetrachloride. The systemic use of this molecule contained in the annatto extract activates NRF2 by exercising cytoprotective and suppressing skin photodamage. Other carotenoids in the diet, as well as biosynthetic precursors, were also tested for chemo and photoprevention of the skin. The cutaneous accumulation of these molecules involves the absorption of photons as sacrificing antioxidants and singlet oxygen inhibitors. Solar radiation initiates harmful

photooxidative reactions and studies have already shown the photoprotective effects that a diet rich in antioxidants can have. Lipid peroxidation, erythema, apoptosis, and DNA damage can be avoided by supplementing micronutrients in minimal amounts of action. Although many of the protection mechanisms are not yet fully understood, nutritional photoprotection is already considered advantageous and complementary to sunscreen of topical use in the prevention of different skin disorders of solar origin.

NRF2 as a target in photo protection

Nuclear transcription factor 2 (NRF2) is ubiquitously expressed in the skin, strongly regulated, has redox sensitivity, participates in inflammatory signaling, DNA repair, and in the antioxidant response. Taken together, these data suggest not only the participation of this protein in the skin barrier but also a possible molecular target in the prevention of diseases related to damage solar UV-induced by and carcinogenesis. Under normal conditions, NRF2 remains in the cytosol linked to its negative regulator and other components liable to ubiquitylation and, therefore, degradation via the 26S proteasome. When reactive species come into contact with this complex, the KEAP1 protein linked to the negative regulator changes its conformation, preventing the degradation of NRF2, which accumulates in the cytosol and translocate to the nucleus, where it undergoes heterodimerization and binding in regulatory regions of antioxidant response genes. Briefly, NRF2 can act as a therapeutic target in different pathologies associated with the skin, including (i) healing of diabetic wounds; (ii) psoriasis; (iii) allergic and atopic dermatitis; (iv) melanocytic dysfunction; (vi) malignant melanoma; (vii) chronological aging and proger. Despite proof of the photoprotection exercised by inducing the increase of NFR2 in cells in vitro, there are still few studies involving systemic administration of inducers of this factor for this purpose.

In addition to NRF2, other molecular targets influence the skin. Peroxisome proliferator- activated receptors (PPARs) are an example. More specifically, PPARα, after its activation by bixin, plays anti-apoptotic, anti-oxidant, and anti-inflammatory functions in the skin.111 Another example is that the similarity between the reactivities of thioredoxin reductase and KEAP1, electrophilic compounds that activate NRF2 by inhibiting KEAP1, they are also capable of inactivating thioredoxin reductase. Knowing the relevance that the Toll-like 4 receptor/ nuclear factor kappa B (TLR4/NF-KB) presents in UV-induced photodamage and skin inflammation and that bixin acts as an antagonist of this receptor, there is a possibility that this way, bixin consumption may influence skin homeostasis. Another molecule that also influences skin disorders is pioglitazone. Mastrofrancesco and collaborators (2014) showed that the topical administration of this compound was effective in reducing inflammation-mediated by T lymphocytes and epidermal hyperplasia. Silva-Abreu and collaborators (2017), who in turn studied pioglitazone-limonene, report improvements in inflammatory processes related to rosacea (a disease previously considered limited in terms of cure). It is also worth mentioning, concerning inflammatory regulation and immunological in dentritic cells, the agonism of pioglitazone in PPAR ɣ, which inhibits MAPK and NF-κB signaling pathways, also suggesting that other agonists of the same receptor, such as thiazolidinediones, may have beneficial effects in inflammatory diseases.

Caspase 14 as a target for photo protection

It is known that the caspase-14 expression is essential to the biochemistry of the cornification process and to the maintenance of the epithelial barrier function, presenting a fundamental role in the prevention of trans epidermal water loss and skin protection against radiation UVB. The caspase-14 expression/activity results in improved UVB filtering capacity of stratum corneum, promoting skin protection. Thus, modified caspase-14 expression may be related to skin photoaging and development of skin cancers. Many studies have shown that treatment with 1, 25-dihydroxyvitamin D3 induces caspase-14 expression *in vitro*, normal skin and in psoriatic lesions of patients, and consequently keratinocytes differentiation. Thus, it reinforces differentiation program, speeds up the formation of cornified envelopes and acts in cornified layer development. Vitamin D3 action is related to its binding to the receptor as well as to the action as transcription factors. Increased susceptibility to photocarcinogenesis was observed in vitamin D receptor knock- out mice. In caspase-14 knockout mouse, defects in skin barrier were observed, specially related to high transepidermal water loss (TEWL) and increased sensibility to UVB photodamage. Once vitamin D induces caspase-14 expression, which influences fillagrin processing and the generation of urocanic acid and other factors, we believe that it is important in skin protection. Self-administration of vitamin D supplements, especially without medical supervision can result in vitamin D intoxication and hyper calcemia. Vitamin D associated to calcium supplementation can increase the risk of generating kidney stones. On the other hand, during the conversion of 7-dehydrocholesterol to vitamin D3, excessive exposure to UVB can generate inactive products (lumisterol and tachysterol), in a way to regulate vitamin D production an avoid intoxication.

Due to the risk of sunburn and skin carcinogenesis, the unprotected sun exposure should not be recommended as a way to obtain vitamin D. Since the patients usually does not apply the amount of sunscreen recommended (the one tested in laboratory to promote efficient protection is 2mg/cm2), studies has shown that its use does not result in vitamin D insufficiency. Less than one minimal erythema dose, i.e., the sun exposure need to reach redness of the skin 24 h after the exposure, is necessary to reach the maximum levels of previtamin D3. In Brazil, in the city of São Paulo, 10 minutes of sun exposure daily of face and hands, even in cloudy and rainy days, is the exposure enough to suitable vitamin D production in a person with photoype II skin. Ichthyosis vulgaris, topic dermatitis and asthma are skin disorders related to ineffective profillagrin processing and besides further clinical studies are needed, the treatment with vitamin D has shown interesting results. Due to its action in caspase-14 expression and keratinocytes differentiation, vitamin D3 has

been used in treatments of skin diseases, such as psoriasis. Vitamin D treatment can improve mild psoriasis symptoms, since it can increase caspase-14 expression. On the other hand, severe symptoms are usually treated with phototherapy. It has been shown that daily low-UV light treatment can increase vitamin D blood levels and improve psoriasis severity.

Not only treatments involving 1, 25-dihydroxyvitamin D3 or low-frequency daily radiation to increase vitamin D can influence caspase-14. George and colleagues identified that luteolin, a natural flavonoid, is promising in inducing differentiation of keratinocytes immortalized by a mechanism dependent on caspase 14 activation.

Photo Protection of Hair

In the case of hair care, not only in dermo-cosmetic products, UVA and UVB filters are incorporated as photo protective assets. Also in shampoos and conditioners, there is an attempt to achieve photo protection with non-ionic chemical filters, however, the inability to form an adherent film on the hair facilitates the removal and reduces the effectiveness of the intended capillary protection. To circumvent the issue that prevents the formation of the capillary film by the non-ionic filter, it is necessary to consider the isoelectric point of the keratin and, consequently, the negative charges that predominate on the surfaces of the wires in addition to other factors inherent to consumption, such as the environment and capillary individuality. Exploring these issues, quaternary molecules such as cinnamidopropyltrimonium chloride, dimethylpabamidopropyl laurdimonium tosylate, propanediol torsilate, dodecyl dimethylaminobenzamidopropyl dimethyl ammonium tosylate, as well as cationic derivatives of chromone with high affinity for hair, as well as cationic derivatives of chromone with high affinity for hair, were developed and disseminated.

Another relevant class for hair protection is that involving silicones. These polymers of silicon, carbon, hydrogen, oxygen, nitrogen or sulphur have wide application in the hair market due to the ease in providing shine and comb ability to the hair, with the adherence of this component to the hair, dependent on the chemical structure of the polymer as well as the intrinsic properties of the wires. It is not new that sunlight causes photobleaching in hair fibers. Studies that investigated the protective effects of polymeric organosiloxane, trimethylsiloxysilicate and propyl-phenyl-silsesquioxane as sunscreens have shown beneficial results in protecting color in chemically dyed hair. Finally, as the main agents against oxidative stress, antioxidants are also present in the study in search of capillary photo protection. The use of this class of molecules has already proved effective in protecting the color of blond hair after UV/VIS exposure. The pre-exposure protective applicability is also explored. In this case, natural products in cosmetics, which already have a consolidated use in trade, continue to be studied regarding their photo protective functions. Plant extracts with antioxidant functions have shown interesting results, ranging from the prevention of lipid peroxidation and protein degradation by extracts rich in hydroxycinnamic derivatives to the preservation of strength, shine and hair color by extracts rich in tannins or flavonoids. It is worth mentioning that, for the antioxidant activity to be satisfactory, it is important that the activity in question penetrates deeply into the thread so that its interaction with the melanin present in the cortex is possible.

Conclusion

Many cell signaling pathways can positively influence skin disorders or even skin photo protection. This makes the investment of forces in the search for new molecular targets in relevant signaling cascades, especially about antioxidant responses. However, not only does the skin suffer from photo damage. Hair is an important object of study in aesthetic health, being explored in different chemical contexts. In capillary photo protection, it is difficult to adapted photo protection actives in hair cosmetics efficiently and to explore photo protection alternatives in addition to those that already exist. In order for these points to be resolved and the demand for products of the type to be met, it is important that experimental standards be explored. Importantly, options for photo protection against visible radiation are still scarce, a sensitive and important issue since exposure to this radiation is inevitable and at the same time can generate relevant oxidative damage to the skin and hair. Therefore, other sun care strategies such as anti- oxidant, anti-inflammatory, and activation of antioxidant defenses (locally and systemically) is possibly a promising path in the search for comprehensive protection from solar damage.

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