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The dark side of visible light

Visible light features the portion of electromagnetic radiation visible to the human eye (400 to 700 nm). The visible spectrum comprises 38.9% of sunlight when it reaches the surface of the earth, but visible light also encompasses artificial light used in daily life. Due to its wavelength, visible light is able to penetrate deep in dermis and even reach the hypodermis. For this reason, it displays various physiological effects on the skin, among them inducing skin pigmentation or promoting the production of reactive oxygen species (ROS) as much as ultraviolet radiations (UVR). It is also able to increase the production of pro-inflammatory cytokines and matrix metalloproteinases (MMP) which play a major role in skin aging.

Visible light also affects DNA through the formation of oxidized DNA bases, thus promoting skin aging and carcinogenesis.

For various decades, dermatologists are promoting the use of photo protectors, which in reality only protect against ultraviolet A (UVA) and ultraviolet B (UVB) radiation. It is time to consider that visible light is a threat for the skin and that an effective photo protection should also include protection against visible light.

Key words

Visible light, skin pigmentation, ROS, carcinogenicity, solar urticarial, chronic actinic dermatitis, polymorphous light eruption.

Electromagnetic radiation features a spectrum. It is classified based on its wavelength into radio waves, microwaves, infrared (IR), visible light, UV, X-rays, and γ radiation. Only fractions of these wavelengths are able to penetrate the ozone layer to reach the surface of the earth; these include ultraviolet radiation (UVR; 280—400 nm), visible light (VL; 400—760 nm), and infrared (IR; 760 nm^{-1} mm). The visible spectrum, used for general illumination, is defined as the portion of electromagnetic radiation visible to the human eye, which corresponds to wavelengths from 400 to 700 nm [1]. The visible spectrum comprises 38.9 % of sunlight when it reaches the surface of the earth [2]. It must be emphasized that visible light (VL) is not only emitted by the solar radiation, but also by artificial light where it represents almost 100 % of total radiation. This includes not only lamps, but also TV, PC, iPad and mobile phone screens, which indicates the importance of VL exposure in our life. The limited information on the incidence of VL on the skin is probably due to the lack of readily available broad spectrum light source thatemits only in the visible spectrum without UV or IR components [3]. In this review we are summarizing the current knowledge about the effects of VL on the skin, and current possibilities of protection against it.

Levels of penetration of visible light in the skin

The effects of light on skin are due to various degrees of absorption of electromagnetic radiation (EMR). The EMR represents the fundamental form of energy having wave and particle properties. According to Planck's law, long wavelength photons carry less energy than short wavelength photons [4]. The light-skin tissue interaction effects are due to absorptionand excitation of photons. Longer the wavelength and deeper its penetration. We know that ultraviolet B (UVB) can only reach the epidermis, whilst ultraviolet A (UVA) reaches the dermis and VL, obviously due to its higher wavelength will penetrate both epidermis, dermis and hypodermis. As seen in Fig. 1, wavelengths as short as 400 nm penetrate the whole epidermis and dermis, at 550 nm 5 % of the total VL radiation reaches the hypodermis and at 700 nm around 20 % will affect it.

Visible light and skin pigmentation

As far as 1962, Pathak et al. [6] presented during the twenty-third Annual Meeting of the Society for Investigative Dermatology the results of an investigation where the effect of long-wave ultraviolet and visible light on human skin had been studied. The forearm skin of 21 subjects (14 fair-skinned Caucasians and 7 individuals with pigmented skin,

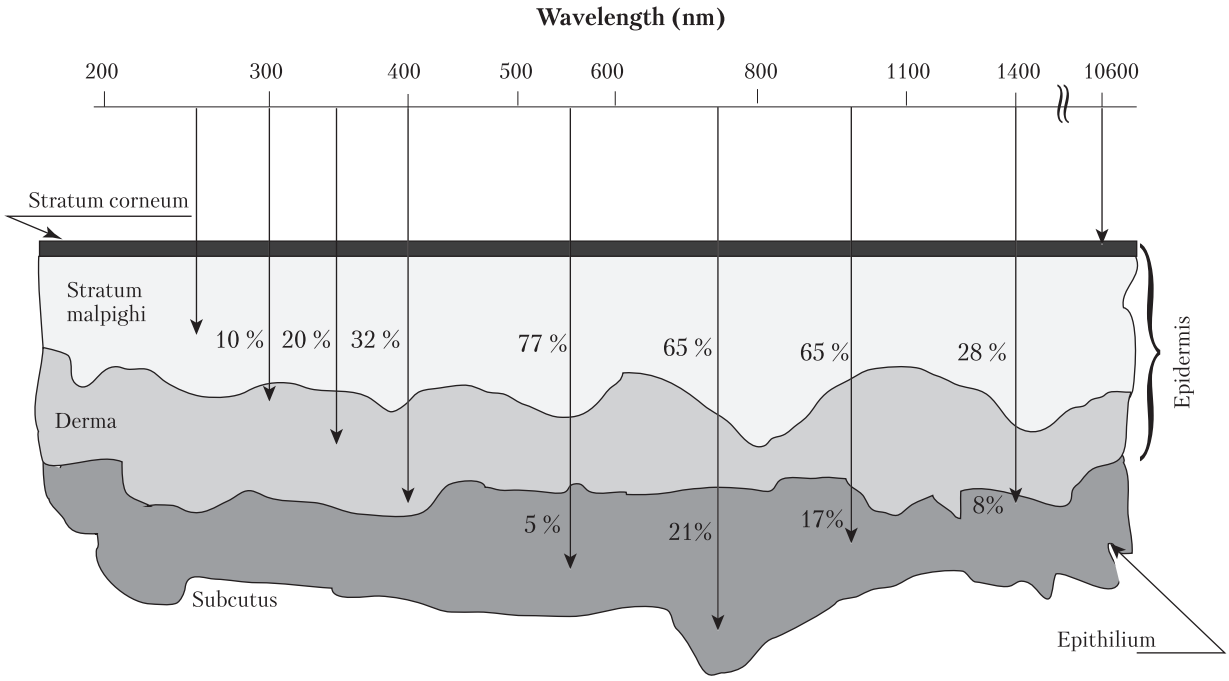

Fig. 1. **Depth of penetration of radiations into the skin according to wavelengths**

being Orientals, East Indians, and lightly pigmented Blacks) was irradiated with a light source delivering radiations between 320 nm and 700 nm (near-UVA and VL). In this study, normally pigmented skin (Oriental, East Indian, Black) has consistently responded to irradiation with immediate pigment darkening (IPD). The response reached its maximal intensity immediately after the end of irradiation and thereafter gradually to diminish (10 to 30 minutes). Fair skin, by contrast, has not shown the IPD response to irradiation with this degree of consistency, but however this response was present. This increase in pigmentation seemed to be caused by newly formed melanin, because it has persisted for more than 3 months in the subjects studied. The action spectrum for IPD was maximal at 400 nm and gradually decreasing till 700 nm. IPD appears as the result of oxidation and darkening of preformed, light melanin. These authors also showed that new melanin formation can be induced by long-wave UV and even by visible light if a proper amount of energy is available.

In a further study following a similar protocol [7], the skin of the lower inner arm of eight volunteers was irradiated with a 390—1700 nm light source. In order to study the colour changes induced with continuous irradiation, remittance spectra were obtained at 15 min intervals. The results showed that pigmentation changes in human skin, in vivo, could be brought about with irradiation using visible and near IR light only. In the beginning of the irradiation it is definitely an IPD reaction with minimal delayed pigment darkening (DPD) reaction. it was also observed that for irradiation times greater than 1 h (or for total light energy delivered greater than 720 J/cm^2) a pigment is generated that has been observed to last up to 10 weeks. In terms of DPD reaction this would imply that DPD appears with doses greater than 720 J/cm^2, corresponding roughly to a 2 h exposure to midday sun.

In an attempt to quantify visible light-induced melanogenesis in human skin [8], exposure of normal skin to visible light (400—700 nm) resulted in the induction of IPD, immediate erythema and a DPD reaction. Both IPD and immediate erythema faded over a 24-h period but the pigmentation did not totally disappear and the residual tanning response remained unchanged for the rest of the 10-day observation period. Of interest, the threshold dose for IPD with visible light was between 40 and 80 J/cm^2, while the threshold dose for «persistent» pigmentation was greater than or equal to 80 J/cm^2.

In another study [3] whose purpose was to determine the effect of visible light on IPD and DPD of melanocompetent skin, the results were compared with those induced by long-wavelength UVA (UVA1). Pigmentation was assessed by visual examination, digital photography with a cross-polarized filter, and diffused reflectance spectroscopy at 7 time points over a 2-week period. Results showed that although both UVA1 and visible light can induce pigmentation in skin types IV—VI, pigmentation induced by visible light was darker and

more sustained. No pigmentation was observed in skin type II. When comparing the quality of pigmentation observed following UVA1 and visible light irradiation in skin types IV—VI, it was noted that pigmentation induced by UVA1 was initially grey in colour and then turned brown after 24 hours, whereas pigmentation induced by visible light was dark brown from the start, showing a differential type of pigmentation between these two light sources.

It was further shown that there were differences in visible light-induced pigmentation according to wavelengths [9]. The potential pro-pigmenting effects of two single wavelengths located at both extremities of the visible spectrum: the blue/violet line (λ = 415 nm) and the red line (λ = 630 nm) were assessed in this study. Colorimetric and clinical assessments showed a clear dose effect with the 415-nm irradiation, in both skin type III and IV subjects, whereas the 630 nm did not induce hyperpigmentation. When compared to UVB irradiation, the blue-violet light induced a significantly more pronounced hyperpigmentation that lasted up to 3 months. The purpose of another recent study [10] was to determine the effect of visible light on the pro-pigmentation pathways and melanin formation in skin. Results showed that a single exposure to visible light induced very little pigmentation whereas multiple exposures with visible light resulted in darker and sustained pigmentation. A significant increase in tyrosinase gene expression by almost 3 fold was registered on day 7, and at day 3 and 7 respectively higher tyrosinase enzyme activity was found when exposed to VL on both days. Spectral characteristic of VL induced pigment was different from that of native epidermal melanin, suggesting that the pigment formed at earlier time pointsis a mixture of products of photo-oxidation and/or various precursors of melanin and various metabolites altogether whereas the pigment formed at later time points appears to be native pigment.

In summary, we know for a long time that VL may induce skin pigmentation by two different mechanisms: IPD which is due to a phenomenon of photo oxidation combined with an activation of precursors of melanin present at that time in the epidermis, and when the dose of irradiation is higher and/or there are repetitive irradiations, DPD appears, similar to the tanning provoked by UV radiation and due to a physiological process of melanogenesis.

Obviously, this must be taken into account in diseases where melanin formation takes an important place. For instance, in melasma, even when we bring protection to the skin against UVR, there will be a deleterious effect of VL in the occurrence and maintenance of the symptoms. Melasma worsens with sun exposure, but a study [11] has shown that even the low energy of artificial indoor VL is enough to react with photocontactants followed by a pigmentation response that may account for its clinical appearance as a mostly non-inflammatory, slowly evolving facial pigmentation. In another trial [12] normally pigmented skin and vitiligo-involved skin of 23 patients with vitiligo (within a distance of 20 cm from each other) were exposed to VL and near infrared radiation (NIR). Normal skin exhibited an increment of absorbance at a broad spectral range from 450 to 720 nm and minimal change in absorption in the 400—420 nm region, which is similar but not exactly the same as UVA-induced IPD spectra and an indication of pigment formation by VL-NIR radiation. By comparison, the vitiligo-involved skin exhibited no distinctive change in absorbance induced by the pigment formation. It was found that VL-NIR radiation produced IPD only in normally pigmented skin and that the presence of constitutive pigment was required to induce IPD response. It was concluded that the degree of formation of IPD from VL-NIR radiation is related to the content of constitutive pigment expressed at short wavelengths (390—450 nm). In a collection of 110 patients with polymorphous light eruption, abnormal reactions to visible light were evident, but were almost exclusively observed in those patients who reacted pathologically to both UVB and UVA (43 % of the male patients, 11 % of the female patients) [13].

Visible light and erythema

In the first previously reported study [6], a mild erythemal response could be detected visually in all 14 fair-skinned individuals immediately after irradiation. In all of them, the immediate erythemal response disappeared within 1—2 hours and reappeared after an interval of 10—18 hours.In all dark-skinned subjects, there was immediate vasodilatation (erythemal response). In these individuals, erythema was not grossly visible 12—18 hours after irradiation, but it could be detected after this interval on the reflectance spectrophotometer. Contrarily, Kollias et al did not reported the occurrence of erythema in their experiment [7]. This is maybe due to the fact that subjects included were all dark-skinned and erythema is more difficult to detect in such a population. However, Porges et al. [8] reported occurrence of an immediate erythema along with immediate IPD when the skin was irradiated with VL. For Mahmoud et al. [3], the immediate pigmentation caused by UVA-1 irradiation was characterized by being dark brown from the start and surrounded by ill-defined erythema,

which disappeared in less than 2 hours. Following exposure to visible light, erythema appeared immediately after irradiation surrounding the pigmentation. It started to fade after half an hour and completely disappeared 2 hours after irradiation. It was proposed that perhaps VL induces a reaction within the chromophores that generates heat, which could be responsible for the erythema.

Visible light and oxidative stress

To assess the role of visible light on skin, human epidermal equivalents were exposed to a dose–response of visible light, and the production of ROS, inflammatory cytokines, and MMPs were determined [14]. Visible light induced a dose dependent increase in intracellular hydrogen peroxide formation (up to 18-fold). Visible light was also found to increase the release of proinflammatory cytokinesfrom epidermal equivalents. IL-1α release was increased up to 2.5-fold. A similar effect was seen with release of IL-1 receptor antagonist, IL-6, GM—CSF, and IL-8. In contrast, visible light, even at doses that induced other proinflammatory mediators, did not increase TNFα release. MMP release was also increased after exposure to visible light. MMP-1 release was increased by 2-fold and MMP-9 release was similarly increased by approximately 2-fold from visible light doses.

The same study demonstrated that visible light can induce activation of the EGFR pathway in keratinocytes in a manner similar to UV. Aberrant EGFR signalling has been implicated in psoriasis and eczema [15]. In an attempt to confirm the results found in vitro, free-radical production was studied on the skin of human subjects [14]. Areas of the skin high in porphyrin content such as the forehead responded to low levels of visible light to induce free-radical production, which could be measured by photon emission or chemiluminescence. A 50 Jcm^{-2} dose at 150 $mWcm^{-2}$ of visible light was able to significantly increase the amount of free radicals by 85.8 % over baseline measurements, consistent with the in vitro ROS results. In another work [16] quantitative ESR-X band spectroscopy was performed to directly detect and quantify the excess free radicals produced in an ex vivo skin model. The result showed a dramatic shift to values of 50 % of the total oxidative burden, dedicated to the visible part. Compared to initial action spectrum values, a reduction to 4 % for UVB and a nearly constant part of 46 % in UVA resulted from the calculation. This means that from the total production of ROS by sun exposure, 50 % is attributable to VL.

The calculated 50 % of free radicals created in the visible part could be experimentally confirmed by carrying out measurements on a clear summer day in Berlin, Germany.

Skin biopsies were exposed to outdoor sun, first directly and then after having interposed a 430-nm cut-off filter, in order to eliminate UV light from the sun spectrum. Comparison of both configurations allowed attribution of approximately 49 % of the total free radical production to the visible part of the sunlight, which was nearly identical with the value previously calculated [16]. To mimic a varying daylight situation, a commercial spotlight which emits visible light (400—700 nm) at a high intensity was used. Already very low levels of visible light create measurable amounts of free radicals. With values of 10,000—20,000 lx, typical of a clear day (not direct sunlight), an RG value of $2—3 \cdot 10^{12}$ radicals/mg was reached in about half an hour — this amount is only slightly below the amount of free radicals produced by UVB/UVA necessary for sunburn erythema. Illuminance of 76,000 lx leads to 4 times this value. A clear sunny sky at 50° latitude and a common surgery room can reach 100,000 lx, indicating that it is important to take the potential damaging powers of intense visible light into account [16].

Singlet oxygen, $^1O_2$ is one of the most potent and harmful free radicals. $^1O_2$ with activation by light can produce severe photosensitivity and/or phototoxicity. The excitation of melanin using visible light generates $^1O_2$ [17] and, consequently, the triplet species derived from melanin. Therefore, cellular damage can occur by both a type I mechanism (directreaction between the triplet photosensitizer and biological targets, typically through an electron transfer reaction) and a type II mechanism (energy transfer reaction between the triplet photosensitizer and oxygen-forming $^1O_2$) [18]. Depending on the severity of the damage, cell death will be the main outcome from visible light exposure.

MMP-9 induction at transcriptional and protein levels in different structures of the rat eye was demonstrated following over-stimulation with white light [19]. Irradiating the buttocks of 16 healthy volunteers with VL, it was reported that visible light spectrum of sunlight significantly increased MMP-1 and MMP-9 expression and decreased type I procollagen expression in the skin, but also contributed to macrophage infiltration [20].

Due to these properties of VL of stimulating the production of ROS and proinflammatory mediators, but also increasing the levels of various MMPs, VL irradiation will represent a key factor in promoting skin inflammation and premature skin aging.

Visible light and carcinogenecity

Another potential outcome from the oxidative stress provoked by VL, which is more dangerous, is

the generation of oxidative DNA products, which could lead to mutagenic compound accumulation, genomic instability and cancer. A comet assay performed under low-dose irradiation with VL showed a considerable increase in the number of strand breaks, which were absent in the controls [17]. This presence of strand breaks demonstrates that melanin photosensitization by visible irradiation induces direct oxidative damage to nuclear DNA. Under visible light irradiation (but also UVA) singlet oxygen is likely to be mostly involved in the formation of 8-oxo-7,8-dihydroguanine that was observed within both isolated and cellular DNA. However, it may be expected that the latter oxidized purine lesion together with DNA strand breaks and pyrimidine base oxidation products are also generated with a lower efficiency through Fenton type reactions [21]. In AS 52 Chinese hamster cellsexposed to extensively filtered monochrome or broad-band radiation [22], between 290 and 315 nm (UVB) the ratio of base modifications sensitive to Fpg protein(i. e. 8-hydroxyguanine and formamidopyrimidines) and T4 endonuclease V (i. e. cyclobutane pyrimidine dimers) was constant, indicating that the direct excitation ofDNA is responsible for both types of damage in UVB range. While the yield of pyrimidine dimers per unit dose continued to decrease exponentially beyond 315 nm (i. e. in UVA range) the yield of Fpg-sensitive modifications increased to asecond maximum between 400 and 450 nm (visible/blue light). The damage spectrum in this wavelength range consisted of only afew other modifications (strand breaks, abasic sites andpyrimidine modifications sensitive to endonuclease III) and was attributed to endogenous photosensitizers that giverise to oxidative DNA damage via singlet oxygen and/or type I reactions [22]. In another similarly designed study [23], for the three cell lines tested, viz. HaCaT cells, L1210 mouse leukaemia cells and AS 52 Chinese hamster cells, the yield of oxidative base modifications generated by a low dose of visible light appeared to be correlated with the basal concentrations of porphyrins in the cells.The damage was inhibited by more than 50 % in the presence of ascorbic acid (100 microM), while alpha-tocopherol and the iron chelator alpha-phenanthroline had no effect and beta-carotene even increased the damage. Even high doses of visible light did not significantly increase the numbers of micronuclei in L1210 cells or of gpt mutations in AS 52 cells. The negative outcome can be fully explained by the photobleaching of the endogenous photosensitizers, which prevents the generation of sufficiently high levels of oxidative DNA damage. Therefore, the mutagenic risk arising from the indirectlygenerated oxidative DNA modifications induced by sunlight may be underestimated when resultsobtained at high doses are extrapolated to low doses or low dose rates [23]. This mechanism of DNA damage provoked indirectly by the excitation of endogenous photosensitizers, which causes oxidative DNA modifications is the only one proceeding in the visible range of the spectrum. The generation of micronuclei associated with the induction of oxidative DNA damage by visible light was analysed in melanoma cells and primary human skin fibroblasts [24]. Similar yields of light-induced oxidative DNA base modifications sensitive to the repair glycosylase Fpg (7,8-dihydro-8-oxoguanine and other oxidative purine modifications) were observed in the normal fibroblasts and the malignant melanoma cells of the same donor. When irradiations were carried out at intervals to compensate for a photodecomposition of the endogenous chromophore, a significant generation of micronuclei was observed in both cell types. Cyclobutane pyrimidine dimers could be excluded to be responsible for the micronuclei induction at wavelengths > 395 nm. Where skin equivalents were treated with visible light or the positive control (UV) and stained for thymine (T–T) dimer formation, UV led to strong induction of T–T dimers, but visible light did not result in T–T dimer formation even at higher doses of visible light [14]. Then it appears clearly that VL affects DNA through the formation of oxidized DNA bases as seen with UVA, but not through dimer formation as observed with UVB.

Visible light and skin barrier function

It was shown that VL can affect epidermal permeability barrier recovery [25]. VL in different wavelength ranges has different effects on the skin barrier recovery rate of hairless mice after barrier disruption by tape stripping. Blue light (430—510 nm) delayed the barrier recovery, whereas red light (550—670 nm) accelerated it, compared with the control kept in the dark. Green light (490—560 nm) and white light (400—670 nm) did not affect the barrier recovery rate. To confirm that these results reflected a biological phenomenon in the skin, that is, that they were independent of the nervous system or circulatory system, the effect of light was evaluated on cultured sections of hairless mouse skin. In this organ culture system, blue light again delayed the barrier recovery and red light accelerated it.

Visible light and skin diseases

Solar Urticaria (SU)

SU is a rare urticarial reaction triggered by electromagnetic radiation of the optical radiation spect-

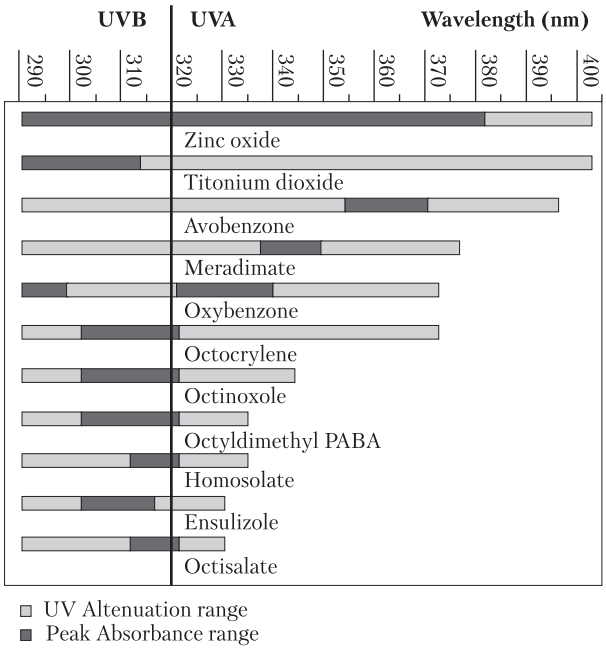

Fig. 2. **Range of absorption of sunscreens**

rum, which usually occurs a few minutes after the start of sun exposure or irradiation with artificial light [26]. Visible light appears to be a frequent trigger. In a pooled review [27], VL appears to be the unique causing factor of SU in 43.4 % of patients, and in 19.4 % of patients when associated with UVA.

Chronic actinic dermatitis (CAD)

Chronic actinic dermatitis (CAD) is an immunologically mediated photodermatosis characterized by pruritic eczematous and lichenified plaques located predominantly on sun-exposed areas with notable sparing of eyelids, skin folds, and postauricular skin [28]. CAD is thought to be due to secondary photosensitization of an endogenous antigen in the skin. The most common action spectrum for CAD is UVB plus UVA, resulting in a decreased minimal erythema dose (MED) for both UVB and UVA in most patients, however, CAD may be seen with decreased MED-B or MED-A alone (12 %–25 %), or with a combination of sensitivity to UVB, UVA, and visible light [28]. Meanwhile there is a scarcity of available data regarding the role of VL in this disease. In a small series of six Japanese patients with CAD, provocative phototests revealed that two of them (33.3 %) had hypersensitivity to VL, with corresponding decrease in MED [29]. Recently, an unusual case of chronic actinic dermatitis was described, which was exacerbated by a tungsten lamp, which emits light in the visible spectrum [30].

Polymorphous Light Eruption

Polymorphous light eruption (PMLE) is the most common photodermatosis, with a prevalence of up to approximately 20 %, particularly among young women in temperate climates. Several hours to days after the first exposure to an intense dose of sunlight in spring or early summer, pruritic, non-scarring lesions of distinct morphology appear on sun-exposed skin. These usually subside in afew days if further exposure is avoided. As summer progresses and after repetitive exposures to sunlight, many individuals experience a hardening effect. This means that skin lesions are less likely to occur, or may be less severe than they were in early spring, which permits patients with PMLE to tolerate prolonged sun exposure [31]. In spite of a thorough bibliographical research, except the paper previously cited [13] we could only identify one paper about action spectrum in polymorphic light eruption including VL [32]. In fourteen patients (25 %) abnormal responses occurred with irradiation from within the long UV and/or visible wavebands. Due to the prevalence of this disease, further investigations would be warranted in order to analyse the real incidence of VL in the occurrence of PMLE.

Pigmentary disorders

As it is currently widely demonstrated that VL impacts the production of melanin, all pigmentary disorders resulting from hypermelanogenesis, for instance melasma, chloasma, post-inflammatory hyperpigmentation, solar lentigo, periorbital pigmentation, acanthosis nigricans... will be obviously worsened by VL irradiation.

Possible protection against visible light

Currently, when we think photoprotection, we are exclusively considering protection against UVA and UVB. This protection can be brought by «chemical» sunscreens which feature organic molecules absorbing UVA and/or UVB radiation in a determinate spectrum (Fig. 2). Of interest, none of these organic sunscreens can protect against the whole UVA/UVB spectrum, which explains that all photoprotectors launched on the market are including a combination of various organic sunscreens in order to bring an adequate protection. Besides are the inorganic particulate UV filters («physical» filters). They feature inorganic materials that absorb in the UV range. They comprise talc (magnesium silicate), titanium dioxide (TiO_2), zinc oxide (ZnO), and various iron oxides. TiO_2 and ZnO show good absorption in the UV range and none in the visible range, which qualifies them to be used in sunscreens (colourless). The iron oxides are coloured materials absorbing in the visible and

some UV wavelengths. Due to their colour, the iron oxides are not suited for use in photoprotectors, unless it being a make-up.

Applying current UVA/UVB photoprotectors protects the skin from the radiations in this range of wavelengths, but not from visible light. Nowadays there exist very few sunscreens absorbing VL and cosmetically acceptable. Obviously, the best protector against VL would be melanin, which constitutes our physiological shield against external radiations. Using animal melanin would be risky because of its potential allergenicity. It should be synthetic melanin, but the inconvenience is its dark-brown colour which makes it cosmetically unacceptable. By chance, now it exists a synthetic, fragmented melanin which is available for cosmetic formulations. The appearance of the creams is perfect, as there is no dark colouration. With concentrations as low as 0.5 % it is possible to obtain a very good protection against VL. We have experimentally formulated such a cream and tested its absorbance in the visible spectrum. As it can be seen from Fig. 3 we have reached an absorbance of 100 % at 400 nm (wavelength of harmful blue light), gradually decreasing along the visible spectrum to reach 70 % at 700 nm.

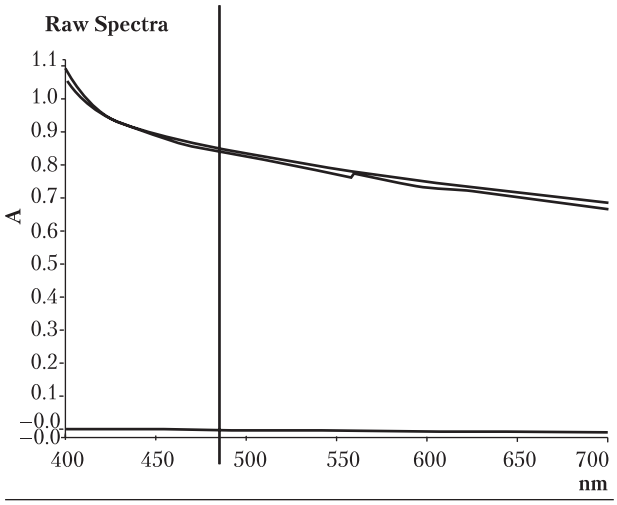

Fig. 3. **Spectrum of absorption in visible light of a cream with 0.5% synthetic fragmented melanin**

Conclusions

There is no question of discussing the harmfulness of UVA and UVB radiation and to question the applications of currently available sunscreens. However, we must be aware that VL is responsible for the same deleterious effects on the skin as those caused by UVA/UVB. Further, the penetration of VL is deeper in dermis than that of UVA/UVB. On the other side, VL represents 50 % of the energy emitted by sun radiation, and almost 100 % of the energy transmitted to the skin by artificial light, it being from lamps, TV monitors, PC or cell phone screens. For this reason, it is compulsory bringing to the skin not only UVA/UVB protection but also protection from VL. The first cosmetics permitting this wide spectrum protection are appearing on the market and we have to change our prescribing habits and offer to our patients more complete protection, which will undoubtedly represent a revolution in photoprotection.

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Темний бік видимого світла

Видиме світло становить порцію електромагнітного випромінення, видимого для людського ока (від 400 до 700 нм). Видимий спектр охоплює 38,9 % сонячного світла, коли воно досягає поверхні землі, але видиме світло також містить у собі штучне світло, яке використовують в щоденному житті. Через довжину його хвилі видиме світло може легко проникати глибоко в шкіру і навіть досягати гіподерми. Тому воно може справляти на шкіру різні фізіологічні ефекти, як і УФ-радіація. Серед них — індукція шкірної пігментації або стимулювання продукції реактивних форм кисню. Воно також може збільшити виробництво запальних цитокінів і матричної металопротеїнази, які грають головну роль у старіння шкіри.

Видиме світло також впливає на ДНК через утворення окислених основ ДНК, тому стимулює старіння шкіри і онкогенез.

Протягом кількох десятиліть дерматологи рекомендують використовувати фотозахист, який фактично захищає лише від ультрафіолетової радіації ІФЛА і УФЛВ. Настав час вважати, що видиме світло становить загрозу для шкіри, і фотозахист повинен також передбачати захист від видимого світла.

Ключові слова: видиме світло, пігментація шкіри, реактивніф форми кисню, канцерогенність, сонячна уртикарія, хронічний актинічний дерматит, поліморфний фотодерматоз.

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Темная сторона видимого света

Видимый свет представляет собой порцию электромагнитной радиации, видимой человеческому глазу (от 400 до 700 нм). Видимый спектр охватывает 38,9 % солнечного света, когда он достигает поверхности земли, но видимый свет также содержит в себе искусственный свет, который используется в каждодневной жизни. Благодаря его длине волны видимый свет может легко проникать глубоко в кожу и даже достигать гиподермы. Поэтому он может оказывать на кожу различные физиологические эффекты, как и УФ-излучение, среди которых индукция кожной пигментации или стимуляция продукции реактивных форм кислорода. Он также может увеличить производство провоспалительных цитокинов и матричной металлопротеиназы, которые играют главную роль в старении кожи.

Видимый свет воздействует на ДНК через образование окисленных основ ДНК, таким образом стимулируя старение кожи и онкогенез.

В течение нескольких десятилетий, дерматологи рекомендуют использование фотозащиты, которая фактически только защищает от ультрафиолетовой радиации УФЛА и УФЛВ. Пришло время понять, что видимый свет также представляет угрозу коже, и что фотозащита должна включать защиту и от видимого света.

Ключевые слова: видимый свет, пигментация кожи, реактивные формы кислорода, канцерогенность, солнечная уртикария, хронический актинический дерматит, полиморфный фотодерматоз.

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