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HYPERPIGMENTATION

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EDITO

Stéphane FAUVERGHE
NAOS International Medical Director

Dear All,

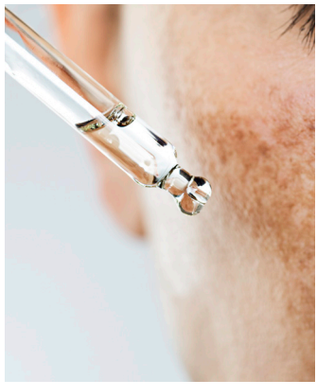
I am very pleased to present you the third edition of BIODERMA Updates Series dedicated to updates in Dermatology.

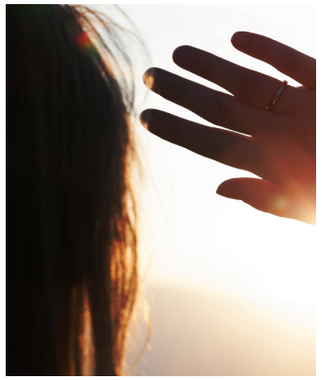
For 3 years now, BIODERMA has been regularly organizing international events dedicated to Dermatology, for dermatologists and all healthcare professionals interested in Dermatology, always presented by renowned experts in their field. In our approach to promote the development of knowledge in Dermatology, we have the pleasure to propose you this new publication, that is the summary of the BIODERMA Symposium held during the EADV meeting in Milan in September 2022: **Hyperpigmentation and visible light : new challenges** *with Prof. Henry LIM from the United States, Prof. Thierry PASSERON from France, and myself as speakers.*

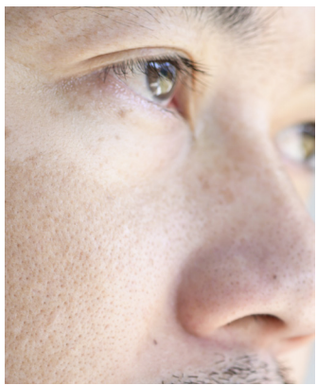
During this symposium, Henry LIM presented the actual knowledge and new data on hyperpigmentation and visible light, Thierry PASSERON delivered a lecture on an original scale and method to assess the protection against visible light and finally I presented innovative solutions against hyperpigmentation.

I wish you all an enjoyable, enriching, and interesting reading.

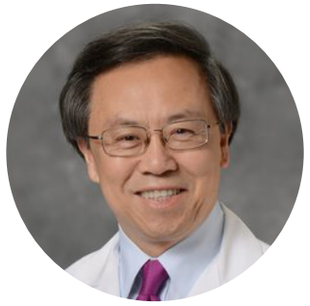
SUMMARY







SPEAKERS'S SHORT BIOGRAPHIES



Henry LIM
USA

Henry W. LIM is the Chair Emeritus of the Department of Dermatology, Henry FORD Hospital, and Senior Vice President for Academic Affairs, Henry FORD Health System, Detroit, Michigan, USA.

He has published more than 400 articles. He has edited 8 textbooks (on vitiligo, photodermatology, photoprotection, and skin of color). He is a recognized world authority on Photodermatology.

Dr. LIM has served as president of Michigan Dermatological Society, vice president of the American Academy of Dermatology, president of the American Board of Dermatology, and president of the American Dermatological Association. From March, 2017 to Feb 2018, he was the president of the American Academy of Dermatology, the world's largest dermatological society.

He was an Associate Editor of the Journal of Investigative Dermatology, and was Editor-in-Chief of Photodermatology, Photoimmunology & Photomedicine. He is currently a Senior Editor of Journal of Drugs in Dermatology, and a member of the editorial board of Photodermatology, Photoimmunology, and Photomedicine. He was a former editorial board member of Journal of Investigative Dermatology, Journal of the American Academy of Dermatology, and JAMA Dermatology. He is an elected honorary member of dermatology societies in Austria, France, Spain, the Philippines, China, and the Baltics.

Thierry PASSERON
France

After his medical training Thierry PASSERON specialized in Dermatology in the department of Pr. Jean-Paul ORTONNE in Nice University Hospital. He also worked one year in Principal Hospital of Dakar, Senegal. In 2003 he published his thesis on the use of Excimer laser in Dermatology.

He worked as clinical assistant in the Department of Dermatology, University Hospital of Nice from 2003-2005. Concomitantly he developed fundamental researches in the laboratory of Dr Robert BALLOTTI (INSERM U895).

From 2005 to 2007, he worked in the laboratory of Dr. Vincent J. HEARING at the National Institute of Health, National Cancer Institute (Bethesda, USA) and characterized the role of SOX9 in melanocytes and in melanoma. He passed his PhD in 2008.

Since 2010, he is full Professor of Dermatology in the University hospital of Nice. He also heads the laboratory INSERM U1065 team 12, C3M dedicated to the research of melanocytic differentiation. He heads the University laser center in Nice. He has three international patents and more than 120 publications in scientific journals (h-index 28).

His fields of research include pigmentary disorders (including vitiligo and melasma), melanoma and lasers.

HYPERPIGMENTATION AND VISIBLE LIGHT: NEW CHALLENGES

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About 50% of the sunlight reaching the Earth's surface is visible light (400-700 nm). Other sources of visible light (VL) include lasers, light-emitting diodes and flash lamps. Photons from VL are absorbed by photoreceptive chromophores *(e.g., melanin, heme, and opsins)*, altering the skin function by activating and imparting energy to chromophores. Additionally, VL penetrates the full thickness of the skin and induces pigmentation and erythema.[1]

In recent years, studies have increasingly shown the negative impact of VL on skin, particularly in individuals with skin of color *(SOC; Fitzpatrick skin types IV-VI)*, including the exacerbation of hyperpigmentation disorders, such as melasma and post-inflammatory hyperpigmentation, as well as induction of the former.[2] **Differences between dark skin and light skin include:**

1. Larger, more melanized melanosomes, distributed individually within the keratocytes rather than in aggregates, in skin phototypes *(SPTs)* IV to VI;
2. Melanin in SOC can filter 2 to 5 times more UV than melanin in light-skinned individuals;
3. Epidermis of SPTs V to VI has an intrinsic sun protection factor *(SPF)* of 13.4 compared to a SPF of 3.3 in light phototypes.

The mechanism of VL-induced hyperpigmentation is thought to be different from that of UVA.[3] Free radicals can be generated in the skin by sunlight, and 50% of those are induced by visible light.[4, 5, 6] The induction of free radicals, leading to the generation of reactive species, is one driving mechanism of VL-induced skin pathologies, leading to the induction of melanogenesis and hyperpigmentation.

When comparing the effect of VL on the immediate pigmentation and delayed tanning of melano-competent skin with those induced by long-wavelength UVA *(UVA1)*, VL-induced pigmentation is darker and more sustained, and UVA1 and visible light can synergistically induce pigmentation in skin types IV-VI.[7] No pigmentation is observed in skin type II. Furthermore, VL induces an immediate erythema in light- and dark-skinned individuals.[7, 8]

In dark skin-type melanocytes, the blue light *(BL)* component of VL is sensed by opsin *(OPN)3*, which ultimately leads to sustained upregulation of the key melanogenic enzymes tyrosinase and dopachrome tautomerase.[3] Moreover, the stimulation of OPN3 by BL induces tyrosinase complexes that stabilize tyrosinase activity, which explains the long-lasting hyperpigmentation observed after VL exposure.[3] This multimeric tyrosinase/tyrosinase-related protein complex is mainly formed in dark-skinned melanocytes and induces a sustained tyrosinase activity.[3, 9]

The VL + UVA1 combination may play a role in conditions worsened by sun exposure, such as post-inflammatory hyperpigmentation and melasma, especially in dark-skinned individuals. The combination of VL + UVA1 induces erythema in light-skinned individuals. The photobiologic effects of sunlight are given in Table 1. Currently available organic *(chemical)* UV filters are not sufficient to protect the skin from the effects of VL. However, data are emerging on the efficacy of topical and oral antioxidants, tinted sunscreens, and novel filters against VL-induced effects.

Sunlight	Effect	Most prominent
UVB, UVA2	Erythema Photocarcinogenesis	SPT I-III
UVA1	Tanning, photoaging Photocarcinogenesis	SPT IV-VI
Visible light	Erythema Tanning	All skin types SPT IV-VI

Table 1. The photobiologic effects of sunlight

Recently, there has been increased recognition regarding tinted sunscreens, as they offer good protection against the effects of VL. Public interest in sunscreen searches using Google trends increased greatly between 2016 and 2021, particularly for tinted and mineral *vs.* chemical sunscreens.[10] A practical guide to tinted sunscreens, including a comprehensive list of 53 tinted sunscreens with SPF ≥ 30 containing iron oxide, has recently been made available on Mendeley *(http://dx.doi.org/10.17632/dtb4y9b684)*.[11]

These tinted sunscreens are listed according to average price per ounce. There may be a role for antioxidants *(AO)* and free radical quenchers in skin protection, and initial clinical studies have demonstrated the effectiveness of topical sunscreen with antioxidant. **Topical AO *(2%)*, compared to control, decreased erythema in SPT I-III and reduced pigmentation *(immediate pigment darkening, persistent pigment darkening, and delayed tanning response)* in SPT IV-VI caused by VL+ UVA1.[12] In another study, sunscreen SPF 50 with 5 antioxidants had the same protective effect as tinted sunscreen SPF 20 on skin exposed to VL + UVA1.[13]**

Polypodium leucotomos extract (PLE) has properties that may offer protection against VL. In subjects with Fitzpatrick skin phototype IV-VI who received a 28-day oral supplementation of PLE *(480 mg daily)*, a statistically significant difference in the amount of relative pigment was observed when comparing persistent pigment darkening and delayed tanning pre- and post-PLE.[14] In addition, PLE was shown to prevent UV- and VL-induced extracellular matrix degradation by: 1. Stimulating tissue inhibitor of metalloproteases; 2. Inducing expression of collagen and elastin; 3. Inhibiting the expression and enzymatic levels of matrix metalloproteinases (MMPs); 4. Functioning as an antioxidant.[15]

PLE could therefore potentially be used as an adjuvant to traditional means of photoprotection, to protect against the effects of VL.

Different sunscreens are needed for different phototypes, a concept termed "personalized photoprotection". Using an artificial intelligence-based algorithm validated by dermatologists, marked differences were observed in the skin ageing process between 2 very large groups of European and Chinese women, both in the prevalence of each facial ageing sign and their kinetics.[16] Thus, photoprotection should be recommended according to skin phototype and dermatoses. **Protection against ultraviolet (UV)B is especially important for light skin as there is a high risk of sunburn, DNA damage and skin cancers. Darker skin may be naturally better protected against UVB, but it is more prone to hyperpigmentation induced by visible light (VL) and UVA.**

Protection against UVA, VL and infrared A can be helpful for all skin phototypes, as they penetrate deeply and cause photoaging. Long-wave UVA1 plays a critical role in pigmentation, photoaging, skin cancer, DNA damage and photo-dermatoses. **Adapting the formulation and texture of the sunscreen to the type of skin and dermatosis is therefore essential.**

Practical recommendations from an expert panel for suitable sunscreens for Fitzpatrick prototype I to VI were issued in 2021 to support the clinician in daily practice.[17] The absorption profile of sunscreen recommended for healthy individuals with different skin phototypes for the prevention of skin cancers and photoaging is shown in Figure 1.[17] The latitude of where the individual lives should also be taken in consideration.[17]

Fitzpatrick phototype	Description	Individual Typology Angle (ITA)	Skin color (ITA classification)	UVB protection (SPF)	UVA protection (UVA-PF)	High energy visible light protection (VL-PF)
I	Always burns, never tans	ITA°>55°	Very light	SPF50+	UVA-PF +++ (>1/3 labelled SPF)	
II	Burns easily, sometimes tans	41°<ITA°< 55°	Light			
III	Sometimes burns, always tans	28°<ITA°<41°	Intermediate			
IV	Rarely burns, tans easily	10°<ITA°<28°	Tan			
V	Rarely burns, tans easily; moderately pigmented	-30°<ITA°<10°	Brown			
VI	Rarely burns, tans promptly and intensely; highly pigmented	ITA°<-30°	Dark	SPF30+	UVA-PF +++ (>2/3 labelled SPF)	VL-PF +++

Figure 1. Spectral absorption profiles of sunscreens suitable for different skin phototypes.

IN CONCLUSION

It is important that dermatologists and the public are aware of the impact of VL on skin, especially in patients with skin of color, and understand the available options for VL protection. Numerous skin protection approaches are available, including organic and non-organic UV-filters, but also topically applicable antioxidants, DNA repair enzymes and compatible solutes, as well as oral photoprotective strategies. The impact of the different wavelengths of sunlight on the skin demonstrates the need for a tailored prescription of sunscreen according to SPT.

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AN ORIGINAL SCALE AND METHOD TO ASSESS THE PROTECTION AGAINST VISIBLE LIGHT

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Over the last ten years, several studies have shown that besides the well-known damaging effects of ultraviolet radiation *(UV)*, **both visible (VL, 400-700 nm) and infrared light (> 700 nm) also cause damage which contributes to photoageing.**[1] **UV is the main cause of skin pigmentation, but VL is also an important contributor, especially in melano-competent subjects.**[2] Furthermore, there is increasing evidence on the role of VL in pigmentary disorders in dark-skinned and immediate erythema in light-skinned individuals.[3,4] Thus, photoprotection from VL light may help to improve several hyperpigmentation disorders.[5]

Broad-spectrum sunscreens protect against UV but do not adequately protect against VL. A sunscreen that protects efficiently against VL, must be visible on the skin.[6] **Mineral filters, especially, zinc oxide and titanium dioxide, are currently used as nanoparticles in sunscreens to minimize the chalky and white appearance on the skin. However, they do not protect against VL.** Tinted sunscreens containing opaque pigments with large particle sizes, such as iron oxides, are the only currently available, efficient topical photoprotection products against visible light-induced hyperpigmentation.[7] Tinted sunscreens use different formulations and concentrations of iron oxides and of pigmentary titanium dioxide. Iron oxides and mineral filters have different absorbance profiles, and many shades of tinted sunscreens are available by combining different amounts of iron oxides and pigmentary titanium dioxide to cater to all skin phototypes.[6] With tinted sunscreens having different formulations and concentrations of iron oxides and pigmentary titanium dioxide, it is critical that the tinted sunscreen color matches the skin color of the patient. **A broad-spectrum sunscreen is necessary to protect against VL, UVA, long-wave UVA, and UVB.** Thus, recommending the right type of sunscreen can be challenging.

Recommendations to assess VL protection of sunscreens have been issued.[8] These recommendations confirm that:

1. Cell-based *in vitro* studies are only suitable for screening compounds;
2. Methods based on reactive oxygen species *(ROS)* should not be used to assess the protective effects against VL -induced hyperpigmentation;
3. The *in vitro* pigmentation protection factor *(PFF)* based on transmission reduction of VL appears to be the most adequate *in vitro* method for topical formulations;
4. *In vivo* models should always be considered the gold standard.[8]

A study assessing the transmission spectra of 10 broad sunscreens and involving subjects with a Fitzpatrick skin phototype III to V reported that the most effective protection was against high-energy visible light[9]. A strong correlation between *in vivo* visible light protection factor and *in vitro* transmittance measurements was observed, with the highest correlation factor at 420 nm and in the spectrum covering from 400 to 469 nm.[9] Transmittance measurements were found to be a good predictive tool to evaluate the VL photoprotection efficacy of a given sunscreen allowing to select formulations for final *in vivo* testing.[9]

In that same study, the reference visible light photoprotection factor VL-PF was also expressed as a percentage using the formula *(1-(1/VL-PF))×100*, called the pVL-PF, in order to facilitate comprehension by dermatologists and patients Therefore, the pVL-PF is a new interpretation of the original VL-PF to compare more intuitively from 0% to 100% the performance of different formulations on VL-induced pigmentation. For this reason, *in vitro* methods using transmittance measurements from 400 to 469 nm are predictive tools to evaluate VL photoprotection efficacy of sunscreens.

IN CONCLUSION

It is possible to protect the skin against VL-induced pigmentation by suggesting the most appropriate sunscreen. Increasing evidence on the impact of the different wavelengths of sunlight on the skin confirms the need for a tailored prescription of sunscreen according to skin phototype and dermatoses, which has been made possible due to advances in the filters and formulations of sunscreen.[10] To date, the most efficient sunscreens to protect the skin from VL contain iron oxide and are tinted. Transmittance measurement between 400 and 469 nm is a good way to predict protection against VL-induced pigmentation. The pVL-PF is an easy way to know the protective properties of a sunscreen against visible light.

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INNOVATIVE SOLUTIONS IN HYPERPIGMENTATION

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Facial hyperpigmentation disorders include melasma, post-inflammatory hyperpigmentation (PIHP) and solar lentigines. They are common, difficult to treat and may have an important impact on the subjects' quality of life.[1] Key challenges in the management of pigmentary disorders, are their resistance to treatment, tendency to recur after treatment, and the risk of exacerbating hyperpigmentation when using many treatment modalities.[2]

For many years, the gold standard for the dermatological treatment of hyperpigmentation has been the Kligman's trio, consisting of hydrocortisone, retinoic acid, and hydroquinone, each with a different effect *(anti-inflammatory, cell renewal accelerant, and melanin decrease in the epidermis, respectively)*.[3] This triple combination has shown its efficacy overtime. However, its use is limited to 2 months twice a year, leaving subjects without treatment 2/3 of the year. Moreover, after treatment, relapses and hypersensitivity, such as irritation or hyperpigmentation, have been observed.[4]

NAOS is the pioneer in ecobiology, a unique scientific approach that uses the in-depth knowledge in skin biology to protect its ecosystem. By observing, understanding and mimicking the natural mechanisms of the skin, ecobiology favours biomimetic ingredients that help the skin strengthening and replicating itself and stimulates its natural rebalancing and regeneration mechanisms, thus allowing to balance and regenerate itself.

Within this context, BIODERMA developed a specific skin care, Pigmentbio®, a complete line of products that allows to manage hyperpigmentation issues all over the year.

The product range Pigmentbio® include two types of ingredients: one to reduce dark spots, the Lumireveal™ technology, and a combination of vitamins acting as skin barrier support to optimize skin recovery.

The Lumireveal™ technology mimics the Kligman's trio mode of action, with glabridin as an anti-inflammatory, while Epidermactiv™ accelerates cell renewal, and the combination of andrographolide plus azelaic acid decreases the melanin level in the epidermis. Skin barrier support is provided by a combination of vitamin C and vitamin E, an antioxidant that prevents skin ageing and melanin darkening, and niacinamide which reinforces the natural skin barrier.[5]

In addition to the Lumireveal™ technology and to 2% fresh vitamin C which was added to reduce dark spots, Pigmentbio® C-Concentrate contains 2% salicylic acid and 8% glycolic acid, for a "peeling-like" effect, and 2% niacinamide plus 0.5% vitamin E for skin hydration and to prevent visible signs of ageing shown by a clinical study in women with melasma.[6] In this study, the melasma area and severity index *(MASI)* score had decreased by 10% after 2 months of daily use and kept on decreasing while the subjects were only using Pigmentbio® Daily-care SPF 50+ plus Pigmentbio® C-Concentrate. After five months, the MASI score had decreased by 25% compared to baseline. In another study involving 41 subjects with melasma, the MASI score was divided by 2 after 3 months of sun care protection and application of Pigmentbio® C-Concentrate.[7] When used alone, Pigmentbio® C-Concentrate increased skin hydration in 90% of subjects, acted as an 8-hour moisturizer, and was very well tolerated in all subjects.[7-9]

Preventing hyperpigmentation is an important issue. Blue light is directly involved in melasma. BIODERMA has developed Photoderm® M SPF50+, a blue light protection sunscreen with anti-recurrence efficacy, high coverage, and a matte finish for dark spots and melasma. The Sun Active Defense technology in Photoderm® M SPF50+ is based on the combined effects of SPF50+, UVA39, and a clean filtering association. By combining pigments *(10.7%)* and iron oxide, 61 to 66% of blue light reaching the skin is blocked *(Figure 1)*.[10]

Clinical investigations showed a significant decrease in the MASI index *(-31.8%, p<0.001)* in subjects with melasma using Photoderm® M SPF50+ for 4.5 months. [11] In addition, the cosmetic qualities of Photoderm® M SPF50+ generated better subject compliance. In other studies, 97% of the subjects had an improved quality of life, due to the high coverage of pigmentary spot, 89% of subjects stated that Photoderm® M SPF50+ leaves a pleasant powdery finish, and 93% considered that it held well throughout the day.[12-14]

Through their ecobiological approach, Photoderm® M SPF50+ and Pigmentbio® C-Concentrate, present an combining high efficacy, high tolerance, and high compliance for both the well-being of the skin and that of the subject. By combining high technology and sensory appeal, daily application is promoted, thus increasing the chances of success in preventing hyperpigmentation and its recurrence.

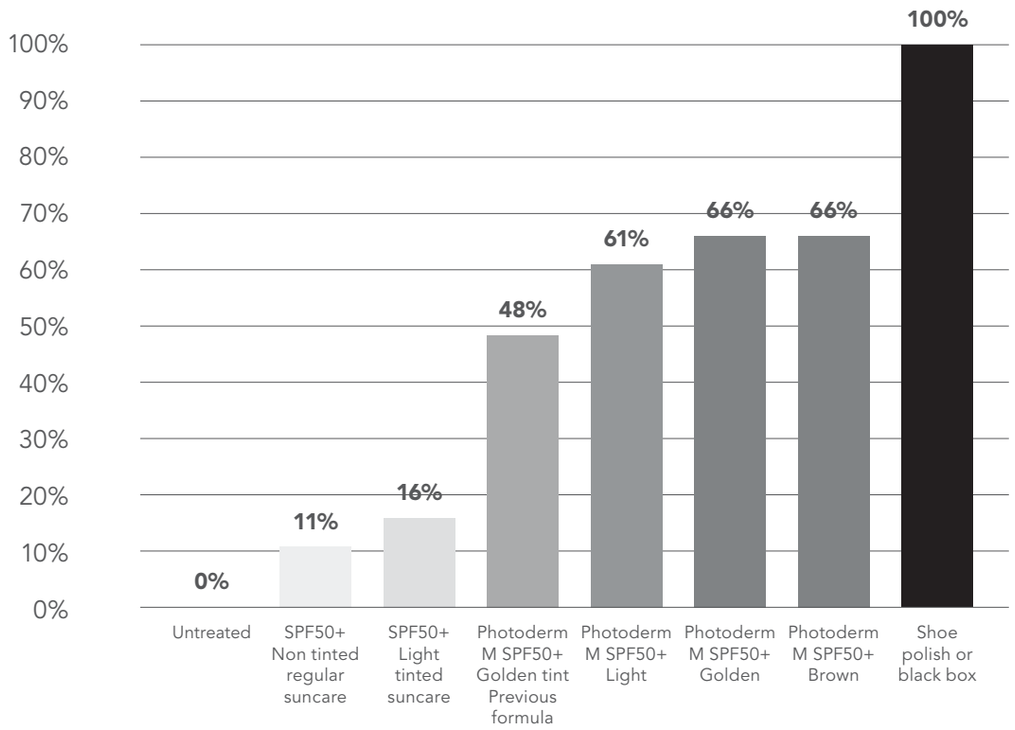

Figure 1. In vivo *protection against visible light - Photoderm® M SPF50+*

20 subjects, phototype III b to V

Clinical study report CPC-3596, STb20061 CPCAD, Nice, 2021 - Assessment of the protective effect of BIODERMA sunscreen products on visible light-induced pigmentation compared with an untreated control zone. Data on file.

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NOTES

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