

# Tanning-Elicited Variations in the Ultraviolet Absorption Spectra of the Cutaneous Tissues: Skin Photobiology and Photomedicine Implications

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**Abstract**—When ultraviolet radiation is absorbed within the cutaneous tissues, it can trigger a number of phenomena that can have detrimental or beneficial consequences to an individual's health. Tanning is among the most visually noticeable of these phenomena. It may result in significant changes in skin pigmentation and thickness. These spectrally-dependent physiological responses, in turn, can elicit variations in the ultraviolet absorption profiles of the cutaneous tissues and, consequently, alter the occurrence of other ultraviolet-induced photobiological processes such as the breaking of DNA strands and the synthesis of previtamin D3. These tanning-elicited variations in the cutaneous tissues' absorption profiles is often tied to the increased presence of melanin throughout these tissues. However, during the tanning, shifts in the relative content of this pigment within certain skin layers can also be observed. In particular, the stratum basale, the innermost epidermal layer where melanogenesis takes place, can have its relative melanin content significantly reduced in comparison with other epidermal layers. Since the aforementioned photobiological phenomena are preferentially brought about within this layer, such pigmentation shifts may have a more pivotal role in skin photobiology than has been assumed to date. Accordingly, in this work, we investigate the impact of spectrally-dependent tanning-elicited physiological responses, with a particular focus on the inter-layer melanin distribution patterns, on the absorption profiles of the main cutaneous tissues. We also examine how variations in these absorption profiles may alter the outcomes of photo-triggered phenomena associated with the onset of different medical conditions. Our findings are expected to contribute to the advance of the current understanding about skin photobiology, which is indispensable for the success of photomedicine initiatives involving this highly complex organ.

**Index Terms**—skin, tanning, melanin, UVB, UVA, absorption, photobiological phenomena, DNA damage, previtamin D3.

## I. INTRODUCTION

A wide range of photobiological phenomena can be elicited by ultraviolet radiation (UVR) interacting with human skin, notably in the UVB (280-315 nm) and UVA (315-380 nm) spectral domains [1], [2]. While some of these phenomena can be clearly classified as either detrimental (*e.g.*, erythema (sunburn), photoaging and photocarcinogenesis) or beneficial (*e.g.*, vitamin D production and nitrite oxide induction) to human health [3], others cannot since their consequences are still not completely understood. Among the latter, we can highlight delayed tanning [4], the focal point of this investigation. Although the visible effects of

this phenomenon, henceforth referred simply as tanning, can last from hours to more than a year [5], the extent of its impact on skin photobiology remains an open question.

Upon penetration in the cutaneous tissues, UVR is subjected to attenuation (scattering and absorption) events. Its absorption may start or intensify the previously cited photobiological phenomena depending on the absorber and the wavelength of the impinging UVR. In the case of tanning, the resulting UVR-induced physiological responses are triggered by melanin, the strongest UV-absorbing pigment found in human skin [6]. Through a melanogenesis process, this pigment is synthesized by melanocyte cells located in the stratum basale (the innermost epidermal tissue), where it is preferentially concentrated [7]. As the epidermal cells move upward, melanin (in colloidal and aggregated forms) is distributed throughout the full thickness of the upper epidermal tissues [5], [8].

During tanning, there is an increase in the number and activity of melanocytes [9]. This leads not only to an increase in the amount of melanin found in human skin, but also in a shift in the distribution of this pigment within the epidermal tissues [10]. Additionally, the thickness of both epidermis and dermis is also subjected to change during tanning through a hyperplasia process [4], [11]. The magnitude of these responses depends on the amount and spectral distribution of the UVR interacting with the cutaneous tissues.

The minimum amount of radiant energy per unit area necessary to produce tanning responses is defined as the minimum melanogenesis dose [12], which is often provided in terms of the standard erythema dose (SED) [13]. According to CIE (Commission Internationale de l'Éclairage) [14], a unit of SED is defined as  $100 J/m^2$  at 298 nm. This wavelength was selected by CIE for being the most effective wavelength at eliciting photobiological responses, such as erythema and melanogenesis, to UVR exposure [13], [14]. In fact, it is employed as the normalization reference for the melanogenesis' action spectrum, which represents the relative effectiveness of different wavelengths of light in triggering this phenomenon [15]. Examples of physiological responses resulting from a tanning process induced by a specific UVR exposure dose schedule are presented in Fig. 1.

We remark that, besides tanning, other important spectrally-dependent photobiological phenomena take place within human skin. For example, the peak of UV-induced DNA damage [17] and the optimum production of vitamin D3 (one of the forms of vitamin D) [18] are associated with exposure to UVB. Furthermore, exposure to UVA may contribute to single strand DNA breaks [9] and vitamin

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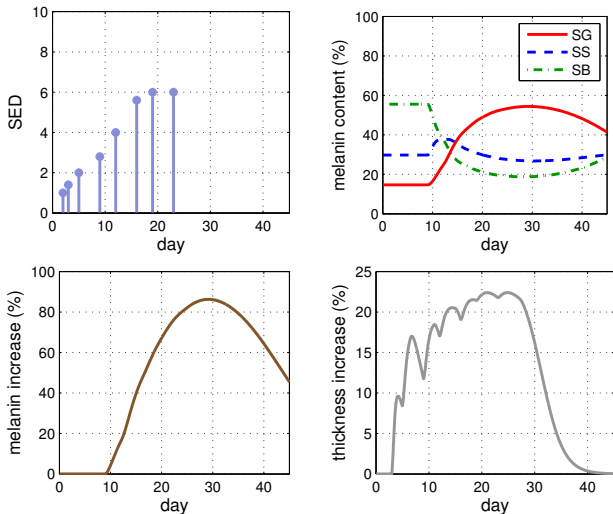


Fig. 1. Physiological responses resulting from a tanning process triggered by ultraviolet radiation. Top left: exposure dose schedule. Top right: variations in the melanin content of the stratum granulosum (SG), stratum spinosum (SS) and stratum basale (SB). Bottom left: variations in total melanin content. Bottom right: variations in skin thickness. These datasets were obtained using a physiologically-based simulation framework [5] whose predictive capabilities were evaluated through comparisons with measured data [16].

D3 photodegradation [19], [18]. It is also worth noting that epidemiological data indicates that the presence of melanin can provide some degree of photoprotection against DNA damage and other photocarcinogenic processes [9], [20], [21]. However, the connections between spectrally-dependent tanning-induced responses, such as variations in melanin content and distribution, and the cited photo-induced phenomena are still elusive. Indeed, experimental datasets to support the study of some of these connections, like the impact of such tanning-induced responses on vitamin D3 production, are unavailable in the literature to date [22].

The lack of quantitative evidence to strengthen the understanding about these connections can be attributed to the limitations of traditional laboratory procedures. For example, *in vitro* procedures employed to collect and prepare a tissue sample can change its optical properties, and *in vivo* procedures involving direct exposure to UVR may pose risks to a subjects' health. Also, recall that light absorption by melanin depends not only on the amount of this pigment present in the cutaneous tissues, but also on its distribution patterns [7], [23]. To the best of our knowledge, controlled (*in vivo* or *in vitro*) spectral measurements involving dynamic changes in the melanin content of specific epidermal tissues, without altering the biophysical characteristics of the specimen at hand, are still unfeasible.

In this work, we perform a detailed assessment on how tanning-elicited physiological responses can affect the cutaneous tissues' UVR-absorption capabilities and, consequently, the induction of photobiological phenomena associated with these capabilities. To overcome the above mentioned experimental limitations, we employed an *in silico* (computational) investigation approach [24] supported by measured data provided in the related literature. The results

of our controlled *in silico* experiments unveil changes in UVB and UVA absorption within the main cutaneous tissues (from top to bottom, stratum corneum, stratum granulosum, stratum spinosum, stratum basale, papillary dermis and reticular dermis) that may result from such responses. We also concisely address the biomedical ramifications of our findings from skin photobiology and photomedicine perspectives.

## II. IN SILICO EXPERIMENTAL SETUP

In this work, we considered the baseline and tanned states of a typical skin specimen with a moderate tanning ability. While the former state corresponds to its constitutive pigmentation level resulting from genetic factors, the latter state corresponds to its facultative pigmentation level resulting from a tanning process whose outcomes are illustrated in Fig. 1. More precisely, we employed the data associated with the peak of that process (day 30). Accordingly, we accounted for an  $\approx 86\%$  increase in the total melanin content and an  $\approx 16\%$  increase in the thickness of its cutaneous tissues. Moreover, we also incorporated in our simulations the specific variations in the melanin content of the stratum granulosum, stratum spinosum and stratum basale. Lastly, we assumed the specimen to be in normal physiological conditions. Thus, no melanin presence was considered in the stratum corneum, papillary dermis and reticular dermis.

The parameter values used to characterize the specimen's baseline and tanned states are provided in Table I and Table II. The selection of values for these parameters was based on physiologically valid ranges provided for specimens with a relatively low constitutive pigmentation level (since this investigation is centered at facultative pigmentation changes) in the related literature. The sources for these ranges are listed elsewhere [25], [26] for conciseness.

To perform our *in silico* experiments, we employed a first-principles model of light and skin interactions, known as HyLIoS (*Hyperspectral Light Impingement on Skin*) [25]. More specifically, we used HyLIoS to compute the directional-hemispherical reflectance and absorptance curves presented in this work. It is worth stressing that its predictive capabilities have been quantitatively and qualitatively evaluated through comparisons of its outcomes with actual

TABLE I  
SETS OF PARAMETERS EMPLOYED IN CHARACTERIZATION OF THE SELECTED SPECIMEN IN ITS BASELINE (B) AND TANNED (T) STATES.

Parameter	Value (B)	Value (T)
SC Thickness (cm)	0.001	0.00116
SG Thickness (cm)	0.0011	0.00128
SS Thickness (cm)	0.0011	0.00128
SB Thickness (cm)	0.0011	0.00128
PD Thickness (cm)	0.04	0.0465
RD Thickness (cm)	0.1	0.1162
SG Melanosome Content (%)	0.21	1.45
SS Melanosome Content (%)	0.43	0.72
SB Melanosome Content (%)	0.80	0.50
SG Colloidal Melanin Content (%)	1.03	7.09
SS Colloidal Melanin Content (%)	2.9	3.50
SB Colloidal Melanin Content (%)	3.9	2.46

Note: The acronyms SC, SG, SS, SB, PD and RD refer to the stratum corneum, stratum granulosum, stratum spinosum, stratum basale, papillary dermis and reticular dermis tissues, respectively.

TABLE II

SPECIMEN'S PARAMETERS KEPT FIXED DURING THE SIMULATIONS.

Parameter	Value
Ratio of Skin Surface Folds	0.1
Melanosome Dimensions ( $\mu m \times \mu m$ )	$0.41 \times 0.17$
Melanosome Eumelanin Concentration ( $g/L$ )	50.0
Melanosome Pheomelanin Concentration ( $g/L$ )	2.0
Melanin Refractive Index	1.7
Blood Content (%)	0.2
RD Blood Content (%)	0.2
Dermal Oxyhemoglobin Fraction (%)	90.0
Hemoglobin Concentration in Blood ( $g/L$ )	147.0
Methemoglobin Concentration in Blood ( $g/L$ )	1.5
Carboxyhemoglobin Concentration in Blood ( $g/L$ )	1.5
Sulfhemoglobin Concentration in Blood ( $g/L$ )	0.0
Bilirubin Concentration in Blood ( $g/L$ )	0.003
Extravascular Bilirubin Concentration ( $g/L$ )	0.0
Beta-Carotene Concentration ( $g/L$ )	$2.1E-4$
Epidermis Beta-Carotene Concentration ( $g/L$ )	$2.1E-4$
Blood Beta-Carotene Concentration ( $g/L$ )	$7.0E-5$
SC Water Content (%)	35.0
Epidermis Water Content (%)	60.0
PD Water Content (%)	75.0
RD Water Content (%)	75.0
SC Lipid Content (%)	20.0
Epidermis Lipid Content (%)	15.1
PD Lipid Content (%)	17.33
RD Lipid Content (%)	17.33
SC Keratin Content (%)	65.0
SC Urocanic Acid Density ( $mol/L$ )	0.01
Skin DNA Density ( $g/L$ )	0.185
Melanin Refractive Index	1.7
SC Refractive Index	1.55
Epidermis Refractive Index	1.4
PD Refractive Index	1.39
RD Refractive Index	1.41
PD Scatterers Refractive Index	1.5
Radius of PD Scatterers ( $nm$ )	70.0
PD Fraction Occupied by Scatterers (%)	22.0

Note: The acronyms SC, SG, SS, SB, PD and RD refer to the stratum corneum, stratum granulosum, stratum spinosum, stratum basale, papillary dermis and reticular dermis tissues, respectively.

measured data [25], and it has been instrumental in a number of biomedical investigations (e.g., [2], [27], [28], [29], [30]).

We note that, within the HyLIoS' ray-optics algorithms, a ray interacting with the tissues of skin specimen can be associated with any wavelength ( $\lambda$ ) within a spectral region of interest. For consistency, we adopted a spectral resolution of 5 nm in all reflectance and absorptance curves presented in this work. In their computation, we considered an angle of incidence of  $0^\circ$  and employed  $10^6$  sample rays (per  $\lambda$ ).

To enable the reproduction of our findings and the future extensions of our investigation by the research community, we made HyLIoS available on online [31] along with the supporting biophysical datasets (e.g., refractive index and extinction coefficient curves) used in this work. Such extensions could involve, for example, the consideration of specimen characterization datasets with distinct degrees of variability (and/or random noise) with respect to the ones presented in Tables I and II.

It is expected that variations in the selected specimen's melanin content and thickness may affect its appearance, particularly considering the peak of a tanning process. The skin swatches presented in Fig. 2 (bottom row) illustrate this aspect. Their chromatic attributes were obtained from

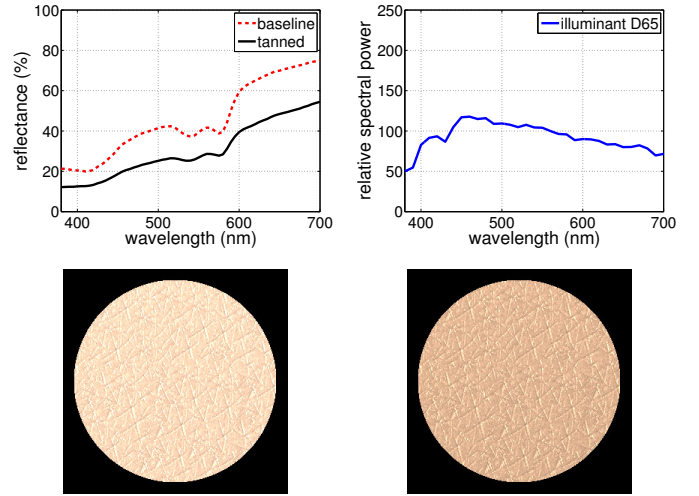


Fig. 2. Components of the convolution process employed to generate skin swatches for the selected specimen in its baseline and tanned states. Top left: reflectance curves obtained using HyLIoS [25] and the data presented in Tables I and II. Top right: relative spectral power distribution of the CIE standard D65 (daylight) illuminant [32]. Bottom left: Resulting swatch for the specimen in its baseline state. Bottom right: Resulting swatch for the specimen in its tanned state.

the convolution of the reflectance curves computed for the specimen in its baseline and tanned states (Fig. 2 (top left)), the CIE D65 illuminant's relative spectral power distribution (Fig. 2 (top right)) and the human photoreceptors's broad spectral responses [32]. This last step was carried out using a standard CIEXYZ to sRGB conversion procedure [33].

Finally, to further the analysis of our *in silico* experimental results with respect to the two spectral regions of interest, UVB and UVA, we calculated the mean relative differences between the absorptance curves computed for selected specimen's cutaneous tissues considering its baseline and tanned states. This quantity (given in terms of %) is expressed as:

$$MRD = \frac{1}{N} \sum_{i=1}^N \frac{|\alpha_t(\lambda_i) - \alpha_b(\lambda_i)|}{\alpha_b(\lambda_i)} \times 100, \quad (1)$$

where  $\alpha_b$  and  $\alpha_t$  correspond to the absorptance curves computed for the specimen in its baseline and tanned states, respectively, and  $N$  is the total number of wavelengths.

### III. RESULTS AND DISCUSSION

In Fig. 3, we present the results of our *in silico* experiments with respect to the stratum corneum, the outermost skin tissue. The main UVR absorbers acting in this tissue are DNA and keratin, which are characterized by a stronger absorption capability in the UVB domain [25]. Nonetheless, the tanning-elicited UVR absorption variations were more prominent in the UVA domain. More specifically, one can observe a more noticeable reduction in UVA absorption within this tissue. This can be attributed to the fact that a large amount of UVB is absorbed in the stratum corneum upon penetration in this tissue, while a large amount of UVA is propagated to the underlying tissues. Thus, the latter is more strongly affected by tanning-elicited changes in the UVR attenuation within those tissues.

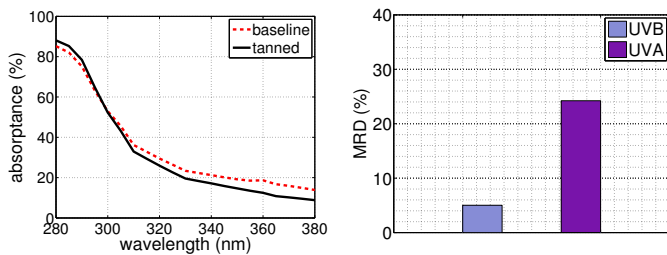


Fig. 3. Absorbance curves (left) and MRD values (right) computed for the stratum corneum of the selected specimen in its baseline and tanned states.

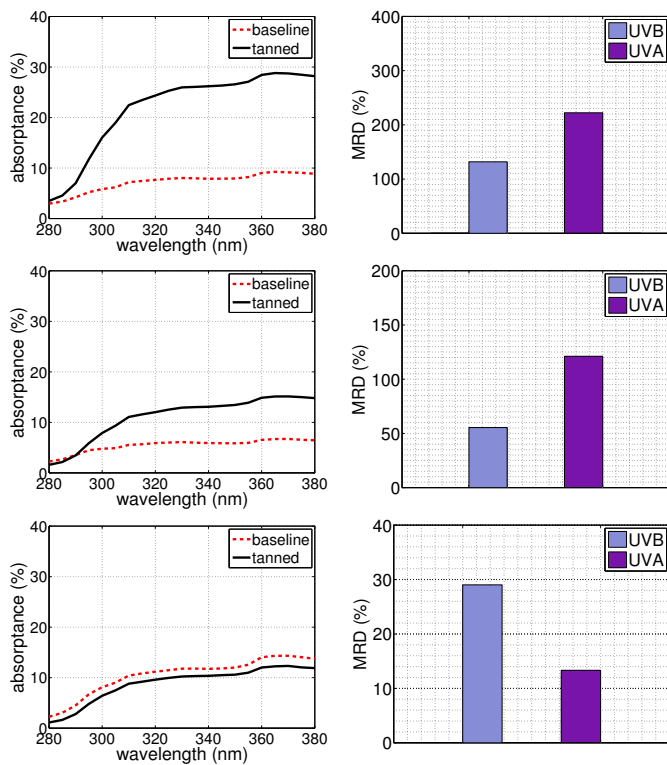


Fig. 4. Absorbance curves (left column) and MRD values (right column) computed for the epidermal tissues of the selected specimen in its baseline and tanned states. Top row: stratum granulosum. Middle row: stratum spinosum. Bottom row: stratum basale.

In Fig. 4, we present the results of our *in silico* experiments with respect to the epidermal tissues, from the outermost to the innermost, stratum granulosum, stratum spinosum and stratum basale. We remark that the largest tanning-elicited intra-tissue increase in the melanin content took place in the stratum granulosum (Fig. 2 (top right)). This resulted in the corresponding largest increase in UVR absorbance (Fig. 4 (top left)) in comparison with the other cutaneous tissues. Again, since most of the UVR penetrating in the stratum granulosum is in the UVA domain, a larger impact of tanning-elicited changes in its UVR absorbance is observed in this domain (Fig. 4 (top right)).

Similar observations can be made with respect to UVR attenuation in the stratum spinosum. However, the increase in its absorbance (Fig. 4 (middle left)) was relatively lower than that verified in the stratum granulosum. This can be explained by the lower increase of melanin presence in the stratum spinosum. Recall that its relative melanin content

was slightly reduced by the tanning responses (Fig. 2 (top right)). In addition, the impact on its UVA absorbance in comparison with its UVB absorbance (Fig. 4 (middle right)) was stronger than that verified for the stratum granulosum.

On the other hand, completely distinct observations can be made with respect to the stratum basale. As shown in (Fig. 4 (bottom left)), its overall UVR absorbance was reduced. Moreover, the tanning responses had a more significant impact on in UVB absorbance (Fig. 4 (bottom right)). The magnitude of these tanning-elicited variations, however, was significantly smaller than those observed for the other cutaneous tissues. This can be associated with the fact that the lowest tanning-elicited intra-tissue increase in melanin content took place in this tissue (Fig. 2 (top right)).

In Fig. 5, we present the results of our *in silico* experiments with respect to the papillary and reticular dermis. In both tissues, it was observed a decreased in their UVR absorbance (Fig. 5 (left)). We remark that there is no melanin present in these tissues under normal conditions. Hence, the decrease in their UVR absorbance can be attributed to the significantly lower amount of UVR penetrating these tissues due to an increased in UVR attenuation in the epidermal tissues. The impact observed in the UVR absorbance of these tissues was slightly higher in the UVB domain (Fig. 5 (right)).

In short, our findings indicate that, at the peak of a UVB-induced tanning process, there are significant quantitative and qualitative variations in the cutaneous' tissues absorbance. More importantly, they suggest that these variations are associated not only with an overall melanin increase, but also with a shift in its inter-tissue distribution patterns. This was illustrated by the observed increases in the absorbance of the top epidermal layers, and reductions in the absorbance of the stratum basale and the dermal tissues. Furthermore, while the increases were more prominent in the UVA domain, the reductions were more prominent in the UVB domain.

As an aggregated effect of these variations, if the cutaneous tissues are further exposed to UVR just after the

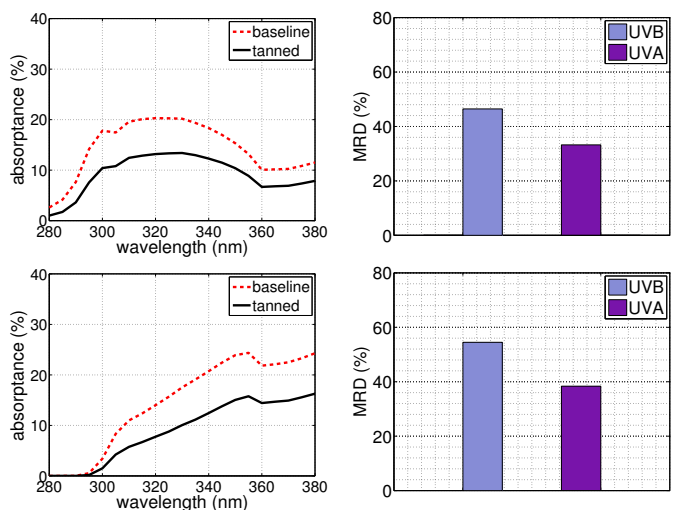


Fig. 5. Absorbance curves (left column) and MRD values (right column) computed for the dermal tissues of the selected specimen in its baseline and tanned states. Top row: papillary dermis. Bottom row: reticular dermis.

tanning peak has been reached, then relatively less UVR (notably in the UVB domain) is likely to be available (in comparison with a baseline state) to trigger other relevant photobiological phenomena that take place primarily in the stratum basale. In the remainder of this section, we discuss potential biomedical ramifications of this aspect. Our discussion is focused on two phenomena with significant impacts on human health, namely DNA-related photocarcinogenesis and previtamin D3 (preD3) photosynthesis.

We note that the same UVR radiation that can trigger tanning can also initiate a photocarcinogenesis process. For instance, the strong UVB absorption by DNA [25] may result in DNA damage and mutations that may give rise to tumours [17], [2]. We remark that, in this investigation, we are interested in what can happen after a UVB-induced tanning process has reached its peak, *i.e.*, the impact of tanning-elicited physiological responses on photobiological phenomena that may be triggered by further UVR (UVB and/or UVA) exposure.

It has been recognized that the stratum basale is a critical area for photocarcinogenesis since its cells can persist and pass on their UVR-induced mutations to daughter cells [2], [34]. Recall that the results of our *in silico* experiments show a reduction in UVR absorbance in this tissue (on average  $\approx 30\%$  in UVB and  $\approx 14\%$  in the UVA). It is worth stressing that it had  $\approx 56\%$  of the total melanin in the baseline state and only  $\approx 19\%$  during the peak tanned state, while the situation was reverse for the stratum granulosum (Fig. 2 (top right)). Nonetheless, the absorbance variations in the former were markedly smaller than those verified in the latter. These observations indicate that a higher relative amount of UVR (in comparison with the baseline state) is absorbed within the top epidermal tissues, reducing the relative amount of UVR traversing the stratum basale. This, in turn, could be translated to a certain degree of transient photoprotection against DNA damage within this tissue in the event that the specimen be subjected to further exposure to UVR.

The exposure to UVR radiation is also known to elicit the photochemical transformation of 7-dehydrocholesterol (7-DHC) into preD3. This is then thermoisomerized to vitamin D3 (cholecalciferol) [35]. It has been demonstrated that this vitamin plays a key role in the maintenance of normal bone mineral density and muscle functions [18], [36]. Moreover, different studies have shown that it may be a mitigation factor for a number of illnesses, including cardiovascular (*e.g.*, hypertension), autoimmune (*e.g.*, multiple sclerosis) and oncologic (*e.g.*, colon cancer) conditions [36], [37]. In the specific case of melanoma, the most serious type of skin cancer, it has been demonstrated that the different forms of vitamin D can have a potential role in reducing DNA damage and increasing DNA repair [38]. Recently, it has also been suggested that they can be employed as adjuvant therapies for COVID-19 infections [39], [40].

It is worth mentioning that we did not explicitly account for UVR absorption by 7-DHC and preD3 in our *in silico* experiments. However, this aspect had no influence in the resulting absorbance curves for two reasons. First, the rel-

atively tiny amount of these substances found in the human skin (on the order of nanograms per  $cm^2$  [41]). Second, their molar extinction coefficients are several orders of magnitude ( $10^{-6}$  [35]) smaller than those of other absorbers found in human skin, notably the melanins [29].

We remark that the optimum wavelengths (action spectrum) for the photosynthesis of preD3 within human skin are in UVB domain, more precisely between 295 and 300  $nm$ , with an apparent maximum near 297  $nm$  [35]. Also, although 7-DHC can be found throughout human skin ( $\approx 3 : 1$  ratio between epidermal and dermal tissues), its highest concentration per unit of area is in the in stratum spinosum and stratum basale [18]. Consequently, these tissues have the highest capacity for UVB-induced preD3 production.

Recall that our *in silico* experiments considering the specimen in the tanned state show lower UVB absorbance values for the stratum basale and dermal tissues. This can be tied not only to relatively lower amount of melanin present in the stratum basale (no melanin is present in the dermal tissues), but also to the increase in UVB absorption in the upper epidermal tissues. In other words, a smaller amount of UVB radiation becomes available for the photosynthesis of preD3 from 7-DHC within the stratum basale and the dermal tissues. This suggests that, at its peak, tanning-elicited physiological responses may contribute to a reduction in the photosynthesis of new preD3 (albeit to a lesser extent than that resulting from a high level of constitutive pigmentation characteristic of darkly-pigmented specimens [18], [36]), not just by increasing the amount of melanin present in the epidermal tissues, but also by altering its relative distribution within these tissues. However, the smaller amount of UVR propagating within the stratum basale and dermal tissues is also likely to reduce the probability of photodegradation of the vitamin D3 [18], [19] already present in these tissues.

#### IV. CONCLUSION

Our findings suggest that tanning-elicited physiological responses, in particular the shift of melanin distribution in the epidermal tissues, may contribute to significant changes in the amount and spectral distribution of UVR absorbed in the cutaneous tissues. In particular, they indicate that absorption of UVR may be reduced in the innermost cutaneous tissues even at the peak of a tanning process characterized by significant overall increases in skin melanin content and thickness. These changes, in turn, may affect other UVR-induced photobiological phenomena with far-reaching implications for human health such as DNA related photocarcinogenesis and previtamin D3 photosynthesis.

As with any *in silico* investigation, the biomedical implications of our findings still need to be assessed through actual experiments when the technology to safely perform them becomes available. Statistical explorations of our simulations' parameter space, albeit potentially insightful and worth future work, would not replace direct comparisons with actual measured data. Thus, further advances in this area will likely require synergistic cooperations between research groups employing traditional and *in silico* experimental approaches.

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