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Erythemal and vitamin D weighted solar UV dose-rates and doses estimated from measurements in mainland France and on Réunion Island

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ABSTRACT

Solar UV radiation causes beneficial and detrimental changes in human health. International and national Health agencies recommend avoiding sun exposure when the solar rays are strongest (typically 2 h before and after solar noon). In this study we detail and refine such recommendations. We estimated biologically-effective radiation (inductive of erythema and pre-vitamin D) using spectral solar UV radiation measurements on a horizontal plane at three French sites equipped with spectroradiometers: Villeneuve d'Ascq (VDA) (North of France); Observatoire de Haute-Provence (OHP) (French Southern Alps); and Saint-Denis de La Réunion (SDR) on Réunion Island, in the Indian Ocean. These sites are very different: VDA is a semi-urban site in a flat region, OHP a rural mountainous site and SDR a coastal urban site on a small mountainous island. Biologically active radiation was analyzed by studying erythema induction and measuring pre-vitamin D synthesis. Dose-rates, doses and times for sunburn induction and vitamin D production were derived. Regarding the level of vitamin D dose considered here (1000 IU), we found that at mainland sites time required for vitamin D synthesis was relatively long, even around solar noon, in winter months this could be 2–3 h for phototype II individuals exposing their face and hands. In the tropics vitamin D could always be synthesized in a reasonable time (e.g. 20 min in winter). By contrast, in summer, the required duration times (exposing face, hands, arms and legs) are very short, approximately 2–4 min on the mainland and 1 min in the tropics for phototype II individuals. In all skin phototypes the duration of sun exposure required to induce erythema was generally longer than that to produce vitamin D. These quantitative results, obtained using an instrument measuring on a horizontal plane and with an unobstructed view, do not represent realistic values for human exposure. To account for realistic human body exposure, received doses and times of exposure were adjusted. Our study shows that, mostly in summer, the time periods where limited solar exposure is recommended should be extended, especially at low latitude locations.

1. Introduction

UV radiation has both beneficial and harmful effects on human health. These depend on environmental conditions, such as geographical location, season, time of day, atmospheric ozone and cloudiness, which determine radiation levels at the Earth surface, and personal factors, such as skin phototype, personal sun exposure behavior, age and gender. These effects can be immediate or delayed. Over-exposure to UV-B (280–315 nm) radiation leads to detrimental effects [1], while sufficient exposure to UV-B is required to trigger the vitamin D production necessary for mineral balance and skeletal maintenance and for other functions such as regulation of cell proliferation and differentiation [2].

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UV-B generates delayed skin pigmentation (3–5 days after exposure according to Juzeniene et al. [3]). UV-A (315–400 nm) generates both delayed and immediate skin pigmentation [3] and may influence melanoma occurrence [4] and immunosuppression [5].

Detrimental effects of UV-B are numerous, including erythema, eye damages (e.g. cataract), general and local immunosuppression, photoageing, melanoma and keratinocytic skin cancers (basal and squamous cell carcinomas) [1,3,6–9]. Occurrence of these diseases depends on several factors. According to Armstrong and Kricker [10] and Gandini et al. [11], intermittent exposure to solar radiation is associated with risk of basal cell carcinoma and/or cutaneous melanoma whereas chronic exposure to solar radiation favors squamous carcinoma. An extensive literature review by Hoel and de Gruijl [12] stated that increased melanoma risk is linked to severe sunburn while non-burning sun exposure is associated with a reduction of risk of melanoma. Age is also important. Early age sun exposure plays a greater role in melanoma occurrence than later exposure [13]. Detrimental effects are not limited to UV-B exposure, and UV-A has a role in melanoma occurrence [4,14,15], in cataract disease [3] and in photoageing [16].

Extent of skin pigmentation affects the erythema response to solar radiation, because melanin affords some protection from the damaging effects of solar radiation [16]. Patients inhabit various phototypes according to their susceptibility to burning upon first sun exposure [17], (Table 1). Erythema appears only if the radiant exposure (or dose, Jm^{-2}) exceeds a certain threshold. This defines a Minimal Erythemal Dose (MED) that is equivalent to the skin absorbed radiant exposure that induces a minimally perceptible skin erythema. MED values depend on the patient's genetic disposition, skin pigmentation and on the thickness of the cornified layer, and may differ by body site [18]. In addition, MED values depend on factors such as gender, meteorological factors and season, but not age [19]. Therefore they are not entirely accurate. Approximate values of threshold MEDs are reported in Table 1 [20].

There are beneficial effects from solar UV exposure, affecting blood pressure, circadian rhythms, depression, scleroses [3,12,21,22], and vitamin D synthesis. Vitamin D (a steroid hormone) is essential for human health, notably affecting bone growth and the maintenance of bone strength. Vitamin D also affects cell growth. The function of multiple genes is modulated by vitamin D metabolites, and many cells express vitamin D receptors [2,3,20,21,23]. Vitamin D may protect against skin cancer [24] and other diseases including internal cancers, hypertension, diabetes, asthma, etc. [21,23,25]. Following UV-B exposure, pre-vitamin D_3 develops in the skin from cell membrane 7-dehydrocholesterol and isomerizes toward vitamin D_3 (cholecalciferol) which further converts in the liver to the vitamin D reserve form, 25-hydroxyvitamin D (25(OH)D), that is further converted in the kidneys into the active form 1,25-dihydroxyvitamin D ($1,25(OH)_2D$). UV-B also regulates the amount of vitamin D synthesis in the skin. Pre-vitamin D_3 can absorb UV-B leading to conversion into lumisterol and tachysterol. These photoisomers can also absorb UV-B and are converted back to pre-vitamin D_3, creating an equilibrium [26]. Thus, longer sun exposure will not further increase vitamin D accumulation, but will increase skin cancer risk [2]. Age is also an important factor: skin capacity to synthesize vitamin D reduces with age [2,27].

The role of skin pigmentation on vitamin D production during solar radiation exposure remains unclear [28]. The authors found no significant correlation between vitamin D synthesis after UV-B exposure and skin pigmentation [29,30] contrary to others [3,18,27,31,36]. The radiant exposure required to achieve vitamin D production depends on the surface area of skin exposed. Holick [32] stated that exposing ¼ of the body skin to ¼ MED produces an adequate amount of vitamin D (1000 IU - International Unit), this has been referred to as Holick's rule. Using Holick's rule, Dowdy et al. [33] and CIE/WMO [20] defined the Minimum vitamin D Dose (MDD) as the minimum radiant exposure equivalent to the recommended daily oral dose of vitamin D. The recommended daily vitamin D dose is poorly agreed. Health organizations recommendations range between 200 and 2000 IU [27,28,34–38]. Following published recommendations [20] we consider for our study a value of 1000 IU and a full-body exposure for phototype II, which corresponds to a MDD of between 21 and 34 Jm^{-2}. This gives a mean value of 110 Jm^{-2} (range: 84–136 Jm^{-2}) for ¼ of body skin surface (hands, face and arms) exposure for skin phototype II. It should be further pointed out that cutaneous vitamin D production also depends on the existing vitamin D status [29,39]. Therefore, the value of 110 Jm^{-2} is equivalent to 1000 IU only if vitamin D status is low.

Mean values MDD_A for an exposed fraction A of the entire skin area were derived. The MDD_A required to induce this recommended vitamin D synthesis is reported in Table 1 for a ¼ of body surface area (BSA) for all phototypes. In the present study we assumed that melanin inhibits vitamin D synthesis and we scaled MDD with skin type like MED, as in [40]. If instead, melanin has a small effect on vitamin D production, type II results could apply to other types.

These $MDD_{1/4}$ values are approximately two times larger than those of Webb and Engelsen [40]. These authors made an assumption due to missing information on the solar spectrum. We chose to follow Dowdy et al. [33]. In contrast to erythema, which appears above a threshold, vitamin D is always produced.

To avoid detrimental solar effects, international and national health agencies recommend avoiding sunlight exposure when the level of UV radiation is high. The World Health Organization recommends limitation of exposure to the sun between 10:00 a.m. and 4:00 p.m., without considering season or geographical location (see https://www.who.int/news-room/q-a-detail/sun-protection). The French association Sécurité Solaire recommends avoiding sun exposure in summer between 12:00 a.m. (noon) and 4:00 p.m. summer local time in mainland France (see https://www.soleil.info/sante/se-proteger/les-10-conseils-essentiels.html). Reduction of solar UV radiation exposure might reduce the beneficial effects. It is useful to compare sunlight exposures that promote a harmful effect, i.e. erythema, and a beneficial effect, i.e. vitamin D synthesis.

Several factors modulate the UV radiation that reaches the Earth's surface: the solar zenith angle (SZA, which depends on geographical location, day, time of day), atmospheric ozone (characterized here by the total ozone column, TOC, mainly impacting UV-B), cloudiness, surface albedo and aerosols. The values of these factors and their variability at different sites throughout the year lead to very different measurable UV radiation levels. Spectral solar UV measurements of irradiance on a horizontal plane were routinely carried out in France at 3 sites for 10 years. At Saint-Denis on Réunion Island, close to the tropic of Capricorn, the UV radiation level around midday is high due to a high solar elevation, especially in summer, and low TOC. UV radiation at the mid-latitude sites in mainland France is lower compared to UV level at Saint-Denis because of their lower solar elevations in summer around midday and larger TOC.

This paper reports a precise evaluation of ground level erythemally

Table 1
Skin types according to Fitzpatrick [17]. Characteristics, approximate values of 1 Minimal Erythemal Dose (MED) and 1 Minimum vitamin D Dose for ¼ of body skin area (face-hands-arms) exposed ($MDD_{1/4}$) [20,40].

Skin type	Skin pigmentation	Skin reaction to sun exposure	1 MED, Jm^{-2}	1 $MDD_{1/4}$, Jm^{-2}
I	Pale white	Always burns, never tans	200	90
II	White (Caucasian)	Burns easily, tans minimally	250	110
III	Light brown	Sometimes burns, slowly tans	300	130
IV	Moderate brown	Burns minimally, always tans	450	200
V	Dark brown	Rarely burns, tans well	600	265
VI	Deeply pigmented dark brown to black	Never burns	1000	440

weighted UV and vitamin D weighted UV in these three sites, and documents that in most instances the sun exposure necessary to synthesize vitamin D is shorter than that required to produce erythema.

All measured spectra were weighted with the erythema and pre-vitamin D action spectra (AS) to estimate biologically-effective dose-rates and derive various biologically-effective doses. These effective quantities were used to estimate the times required to develop erythema and generate the recommended vitamin D dose. Since these doses are relative to a horizontal plane they are not representative of the UV doses received by a human body. For this reason, they have to be modified. We used conversion factors for daily doses proposed by Pope and Godar [41] to correct the times to develop erythema and to produce vitamin D.

Due to several uncertainties (vitamin D action spectrum, MED, MDD and also UV radiation measurements - as will be discussed hereafter), the computed times can only be considered as estimates.

The ground-based spectroradiometers are described in Section 2 along with techniques to derive biologically-effective radiation at the surface. Section 3 presents the results: ten-year climatologies of total ozone column and mean UV index (UVI) around solar noon at the 3 sites; erythemal and vitamin D effective 1-hour doses and daily doses; comparisons of these doses with the MED or the MDD; exposure times required to develop erythema and produce the recommended vitamin D synthesis; and estimates of exposure times accounting for the shape of the human body. All these results are discussed. Conclusions are reported in Section 4.

2. Measurements

The UV measurements used were obtained at Villeneuve d'Ascq (VDA) (50.61° N, 3.14° E, 70 m a.s.l. (above sea level)), Observatoire de Haute-Provence (OHP) (43.93° N, 5.70° E, 686 m a.s.l.), and Saint-Denis, Réunion Island (SDR) (20.9° S, 55.5° E, 85 m a.s.l.). All sites use a double-monochromator spectroradiometer (model DTMc300, Bentham) that measures irradiance on a horizontal plane. The instruments are thermally regulated and provide global spectral irradiance (in Wm^{-2} nm^{-1}) in the 280–450 nm wavelength range with a 0.5 nm sampling step. Scans are performed every fifteen minutes (at OHP since 2011 and at SDR since 2009), or every thirty minutes (at VDA since 2009 and at OHP in 2009–2010) from sunrise to sunset. Scan duration is approximately 5 min. All instruments are regularly calibrated with standard 1000 W lamps whose calibrations are traceable to the National Institute of Standards and Technology. After calibration the solar spectrum is corrected for wavelength misalignment and cosine error [42]. The three instruments are affiliated with the Network for Detection of Atmospheric Composition Change (NDACC).

Biologically-effective dose-rates (or irradiances, Wm^{-2}) were computed: the corrected spectra were weighted with action spectra for human skin from the Commission Internationale de l'Éclairage (CIE), i.e. the erythema action spectrum [43] leading to the erythemal weighted UV, UV_{ery}, and the action spectrum for the production of pre-vitamin D_3 [44] leading to the vitamin D weighted UV, UV_{vitD}. The UVI (unitless) was then derived by dividing UV_{ery} by 25×10^{-3} Wm^{-2} [45,46]. As mentioned in [45], this UVI is a simple tool to help people adopt safe behavior regarding UV radiation, specifically to avoid sunburn. The CIE vitamin D action spectrum is subject to large uncertainties. Indeed, it is derived from measurements at few wavelengths and is extrapolated to wavelengths longer than those measured [20]. Work by van Dijk et al. [47] discusses a possible shift in wavelength of approximately 1 nm and presents other AS. However, due to incoherence in these new AS, they do not represent a better choice than CIE AS. Thus, we used CIE AS in this study.

Irradiance uncertainty results from uncertainties in the absolute calibration (including spectral irradiance lamp uncertainty provided by the lamp supplier, imprecision of adjustments and wavelength misalignment) and in the field measurements (imprecision of diffuser horizontality, uncertainty on cosine correction and on wavelength shift correction). Resulting uncertainties of UV_{ery}, UVI and UV_{vitD} are approximately 5% (coverage factor $k = 2$) [42]. Regarding the UV_{vitD} uncertainty, 5% is a low estimate because, as mentioned above, the vitamin D AS is not well determined, but it is hard to say how much larger it actually is.

Fig. 1 shows erythema and pre-vitamin D action spectra and noon solar irradiance spectra measured with cloudless sky conditions (CS) at VDA near winter solstice and spring equinox and at VDA and SDR near summer solstices. These spectra span all possible situations that are encountered at these sites. At wavelengths shorter than (approximately) 315 nm, weighting functions are large whereas spectral irradiances are small due to ozone absorption and Rayleigh scattering.

Contributions of 5-nm wavelength intervals to erythemal and pre-vitamin D weighted irradiances are plotted in Fig. 2a. For both erythema and pre-vitamin D, when irradiance levels are high (green and brown lines, in summer), contributions are predominantly from short wavelengths while longer wavelengths contribute more when the irradiance level is low (magenta and black lines, i.e. winter and spring). The wavelength range relevant for the induction of erythema is wider than that for pre-vitamin D synthesis. Fig. 2b relates cumulative contributions versus wavelength. At high irradiance levels (green and brown curves), wavelengths shorter than 315 nm contribute about 70% of the erythemally weighted irradiance and about 90% of pre-vitamin D weighted irradiance. At low irradiance levels (magenta and black lines), these contributions reduce to ~40% and ~85% respectively. The contribution of wavelengths longer than 315 nm is always small for UV_{vitD} but important for UV_{ery} when solar elevations are small.

Various quantities were computed to characterize the biologically-effective radiation at the three sites, including 1-hour means around solar noon, diurnal variations of 1-hour doses and daily doses. For these observations, climatologies for 2009–2018 were established enabling examination of average seasonal variations. Times for sunburn and 1000 IU pre-vitamin D synthesis were estimated from the 1 MED and 1 $MDD_{1/4}$ values in Table 1.

3. Results

3.1. UVI and Modulating Factors

VDA is a flat semi-urban site, with presence of absorbing aerosols

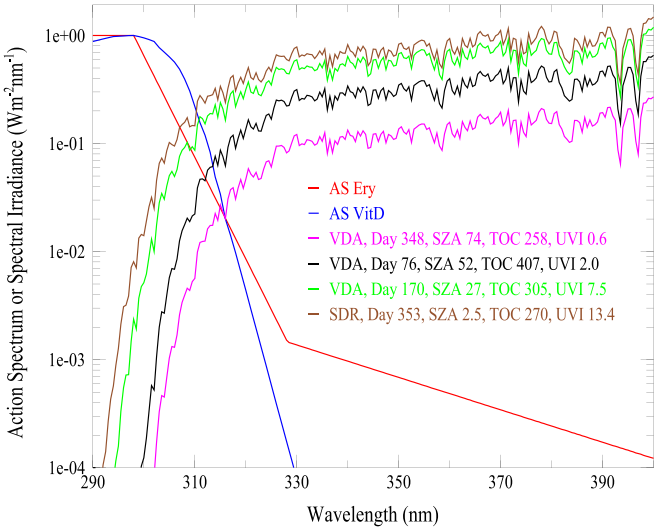

Fig. 1. Action Spectra for erythema (red) and pre-vitamin D_3 production (blue), normalized to unity, along with spectra measured close to solar noon with cloudless conditions at VDA close to winter solstice (magenta) and spring equinox (black) and at VDA (green) and SDR (brown) close to summer solstices. SZA in degree, TOC in DU.

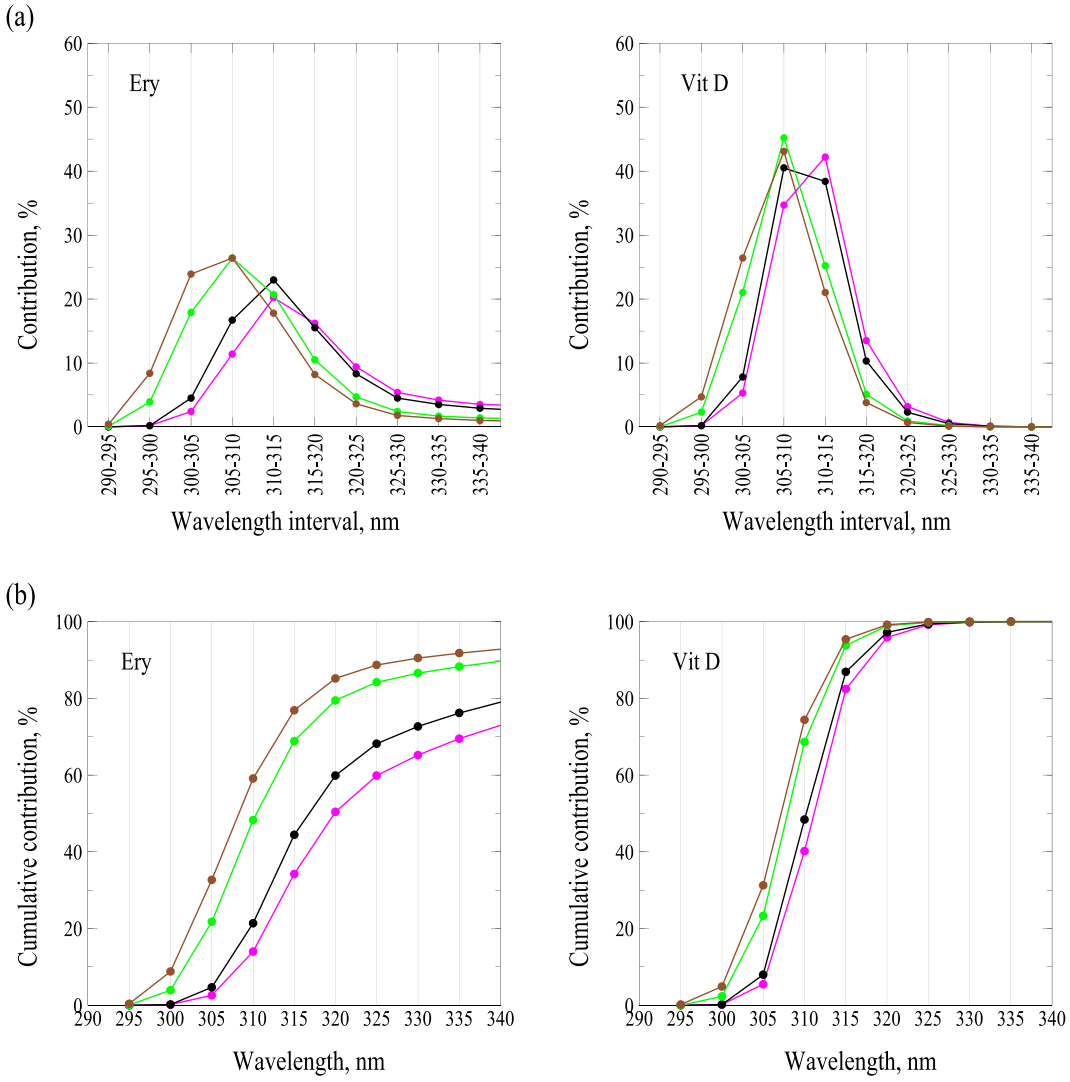

Fig. 2. Contribution of spectral weighted irradiance to the weighted irradiance (UV_{ery}) and to the pre-vitamin D_3 weighted irradiance (UV_{vitD}) for the measured spectra shown in Fig. 1 (same colour code). (a): Contribution of 5-nm wavelength intervals to UV_{ery} (left) and to UV_{vitD} (right). (b): Cumulative contributions to UV_{ery} (left) and UV_{vitD} (right).

from pollution. Fig. 3a exhibits seasonal variations of TOC between 2009 and 2018 (OMTO3 product from OMI, https://avdc.gsfc.nasa.gov/index.php?site=677055910&id=28) that lie, on average, in the 270–400 DU range, lower in autumn and larger in spring. OHP is a rural mountainous site characterized by a TOC similar to VDA (Fig. 3b) and the intermittent presence of absorbing aerosols. SDR is a coastal urban site on a mountainous island, characterized by a much smaller TOC than at mainland sites, on average in the 250–290 DU range (Fig. 3c). SDR has a complex topography, is frequently influenced by noontime orographic clouds and is subject to a small aerosol load. All sites show large year-to-year variability of TOC. We also report climatological (averaged from 2009 to 2018) seasonal variations (Fig. 3a,b,c).

Seasonal variations of mean UVI in a 1-hour window around solar noon are shown for all sites in Fig. 4, along with their climatology. There is large year-to-year variability mainly due to cloudiness and TOC variability. According to the UVI classification [44], at VDA UVIs are low in winter (≤ 2), moderate (in the 3–5 range) in spring and autumn, and high (6–7) in summer. At OHP the situation is similar, but with some very high values (8–10). At SDR the winter UVIs are low to moderate; in spring and autumn they are high to very high and in summer they are extreme (≥ 11). Differences between UVIs in mainland France and SDR can be broadly explained from several modulating factors. At SDR the average winter UVIs are ~4-5 compared to ~1 at OHP and ~0.5 at VDA. These differences reflect differences in noontime SZA (~44° at SDR, 67° at OHP and 75° at VDA) and differences in TOC (~260 DU at SDR, ~310 DU at the other sites). These UVI differences are about 7% lower than expected because of the difference in the Sun-Earth distance at winter solstice between the northern and southern hemispheres.

3.2. *Thresholds for Erythema and Sufficient Vitamin D Synthesis*

As mentioned by McKenzie et al. [48], the absolute values of the effective dose-rates UV_{ery} and UV_{vitD} are not directly comparable since the action spectra are normalized. We computed erythemal and vitamin D effective doses (dose-rates integrated over 1 hour) for comparison with the MED and $MDD_{1/4}$ respectively for any skin type.

Fig. 5a shows diurnal variations of erythemal and vitamin D 1-hour doses (dose-rates integrated over 1 hour, Jm^{-2}) for CS days on four typical dates at VDA (around summer and winter solstices, and spring and autumn equinoxes). Differences between different days are mostly explained by ozone and SZA. A Sun Earth distance (smaller around boreal winter solstice, larger around boreal summer solstice) reduces the differences between winter and summer values. Close to winter solstice (21 December) and spring equinox (20 March) UV received over a 1-

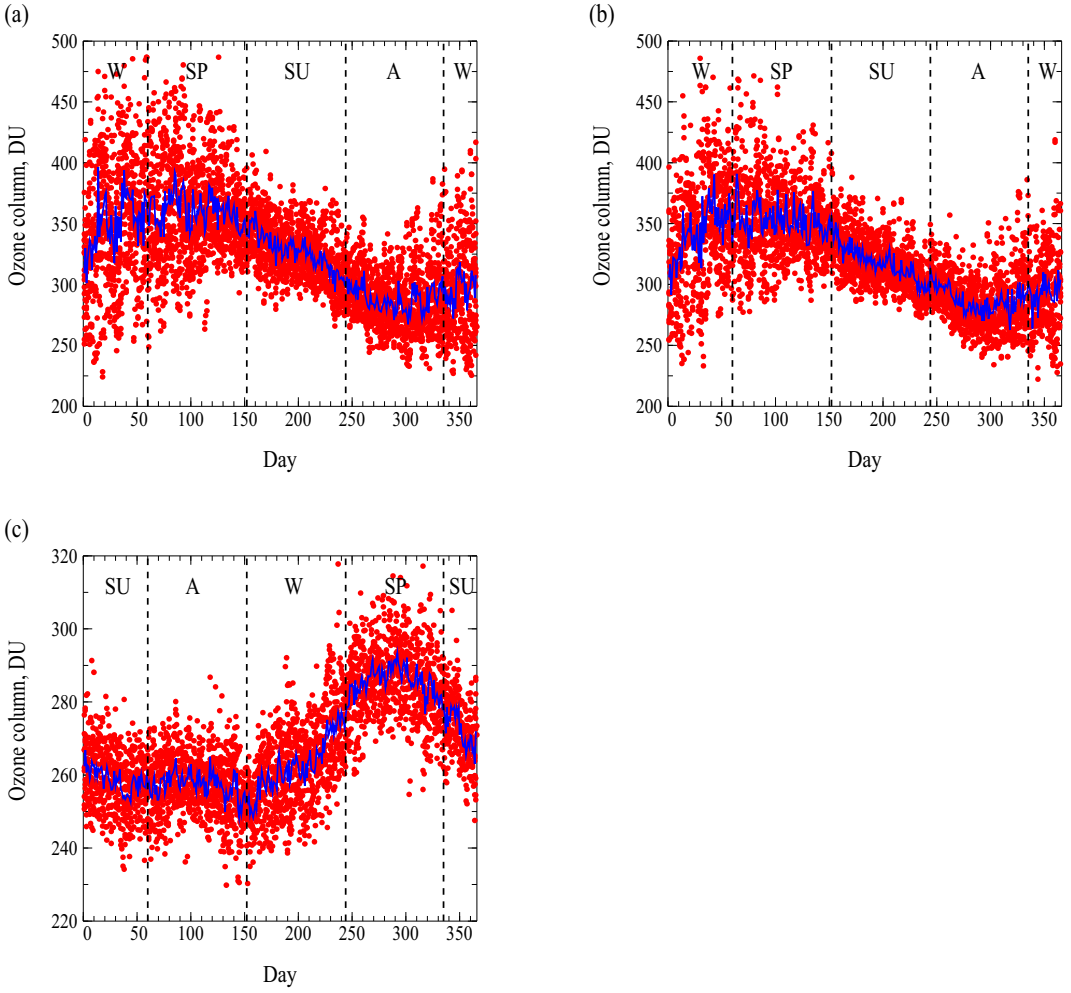

Fig. 3. Seasonal variations of the total ozone column (OMI) during the period 2009–2018. Red dots: daily values, blue solid line: climatology. (a) At VDA, (b) at OHP, (c) at SDR. Vertical black dashed lines indicate season-like limits: W: Winter (December–February for Northern hemisphere (NH), June–August for Southern Hemisphere (SH)), SP: Spring (March–May for NH, September–November for SH), SU: Summer (June–August for NH, December–February for SH), A: Autumn (September–November for NH, March–May for SH).

hour period is insufficient to induce erythema in individuals of any phototype. Close to summer solstice (21 June) a 1-hour exposure can induce erythema between 11:00 and 16:00 summer local time (SLT) in skin types I-IV, between ~12:00 and 14:30 SLT in skin type V. There is no risk for skin type VI (black dashed line missing). Close to autumn equinox (23 September) a 1-hour exposure leads to erythema only in skin types I-III between ~12:00 and 15:30 SLT.

Vitamin D (1000 IU) cannot be synthetized close to winter solstice by individuals of any phototype. Close to spring equinox, UV_{vitD} results in sufficient vitamin D synthesis in skin types I-IV with a 1-hour exposure between ~12:00 and 14:00 winter local time (WLT), whereas skin types V and VI cannot produce sufficient vitamin D under these conditions. Close to summer solstice sufficient vitamin D production can occur after a 1-hour exposure during most of the day (between ~10:00 and 17:00 SLT for type VI). Close to autumn equinox the time-window is shorter (~12:00–15:00 SLT for type VI).

Climatological (averaged 2009 to 2018) seasonal variations of 1-hour doses at different times at VDA are shown in Fig. 5b along with 1 MED and 1 $MDD_{1/4}$ for all phototypes. At each time point the 1-hour dose is the average of before and after noontime 1-hour doses (i.e. assuming solar noon at 12:00 UTC, the noon ±2 h plot corresponds to the average of dose-rates integrated between 9:30 and 10:30 UTC and between 13:30 and 14:30 UTC). Phototypes V and VI may never develop erythema. For other phototypes, a 1-hour exposure leads to erythema within a given day or month range and during a given number of hours from noontime: e.g. type II is at risk of erythema close to noon between days ~100 to 270 (~April to September) and up to 3 h from noon between days ~160 to 210 (~June–July).

All phototypes can synthesize vitamin D sufficiently during a 1-hour exposure at a certain period of the year and at a specific time: e.g. for photypes II and III the periods are ~March–October close to noon, and ~May–August at 4 h from noon. Table 2 gives the month ranges for each effect at different times of day.

Both effective 1-hour doses at OHP are larger than those at VDA by a factor of 1.2–1.5 in summer and autumn, and ~1.7–2 in winter and spring, however the results are not very different from results from VDA. They are displayed in Fig. S1 (in the supplementary material), and summarized in Table 3.

Results from SDR, (Fig. 6a,b) show significantly larger 1-hour doses than observed at mainland sites: larger than at VDA by a factor of 2–4 in summer/autumn, and up to 6–10 in winter/spring. Erythema risk is greater and vitamin D synthesis markedly more efficient than at mainland sites. Fig. 6a shows that, for all phototypes, around summer solstice (21 December) and autumn equinox (20 March), a 1-hour exposure around midday can induce erythema, and around the 4 selected dates vitamin D synthesis is sufficient during most of the day (Table 4).

The previous results for vitamin D were obtained, assuming 1-hour exposure of one quarter BSA ($MDD_{1/4}$ horizontal black dashed lines).

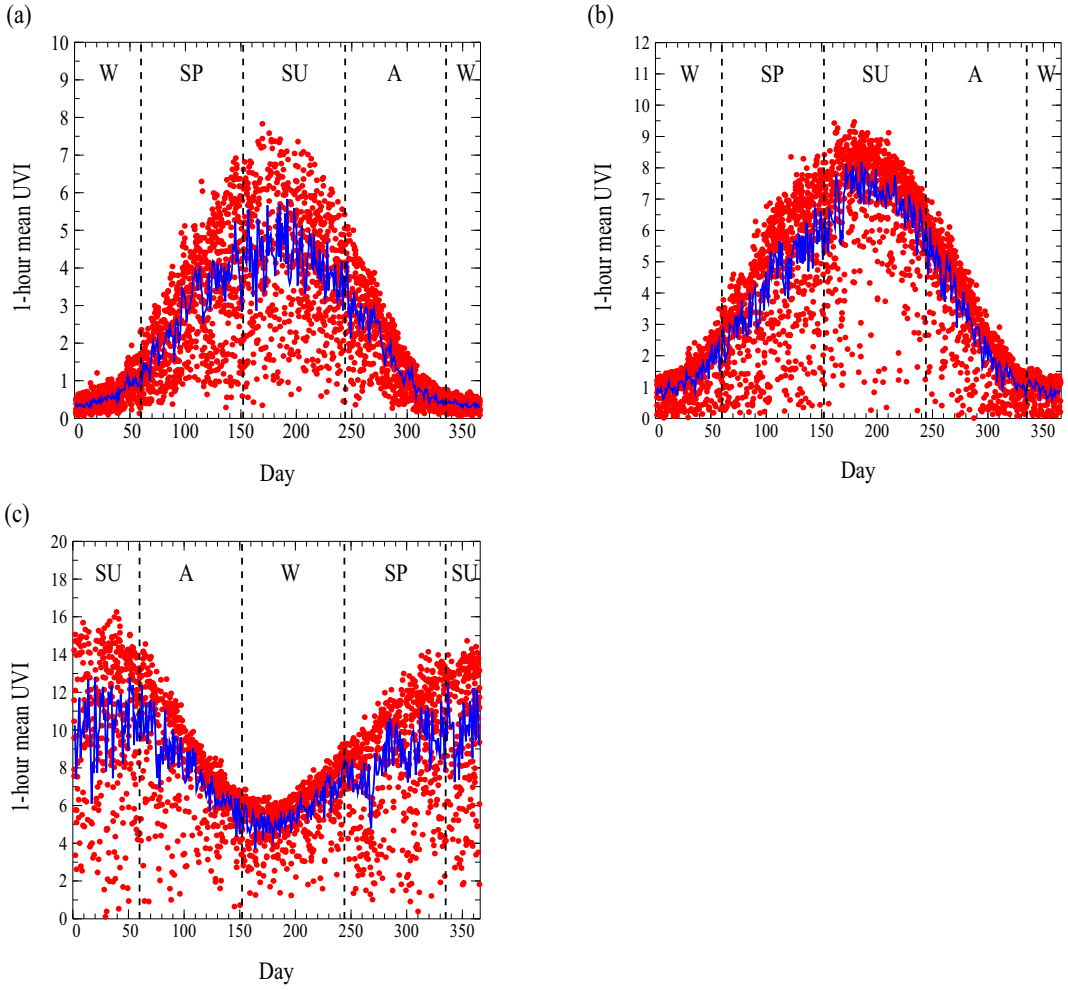

Fig. 4. Same as Fig. 3 but for seasonal variations of the mean UVI in a 1-hour window centered at local solar noon (± 30 min around solar noon).

If a larger area of skin is exposed to solar radiation, vitamin D synthesis will be favored (MDD_A smaller).

Seasonal variations of erythemal and vitamin D daily doses, along with relevant climatology, are shown in Fig. 7 with 1 MED and 1 $MDD_{1/4}$ for all skin phototypes. These doses facilitate estimation of exposure of outdoor workers or vacationers who spend long periods outdoors.

The VDA climatology (Fig. 7a), shows skin phototypes I-III to be at risk of erythema for a longer period relative to other phototypes. On average they are at risk from February to mid-November. They also achieve the recommended vitamin D dose during a longer period than other phototypes, on average from February to November. This means that a 'vitamin-D winter' [49], a period of insufficient vitamin D production, extends only from December to January for these phototypes. Monthly ranges of erythema risk and 'vitamin-D winter' for all phototypes are reported in Table 5.

At OHP (Fig. 7b), daily doses of UV are higher than at VDA but the results are similar (Table 5).

Red dots in Fig. 7a,b indicate that cloudy situations decrease strongly the ability to produce sufficient vitamin D, extending the 'vitamin-D winter' period. Cloudless conditions increase the erythema risk period.

At SDR (Fig. 7c), in contrast to mainland sites, all phototypes risk erythema all year round and sufficient vitamin D is produced, meaning that there is no 'vitamin-D winter', even in cloudy situations (see red dots and Table 5).

3.3. Duration Times to Develop Erythema and to Synthesize the Recommended Amount of Vitamin D

From previous results from 1-hour UV effective doses (see Section 3.2), individuals of each phototype can choose a time of day that will allow them to avoid erythema and yet produce a sufficient amount of vitamin D during one-hour exposure. Determining adequate solar exposure is easier by considering the times associated with induction of erythema and synthesis of sufficient vitamin D. These times, t_{ery} and t_{vitD}, are calculated by computing erythemal and vitamin D effective doses (dose-rates integrated as a function of time) and comparing them with the MED and MDD_A respectively, for any skin type.

Fig. 8 shows exposure duration times versus time of initiation of sun exposure for the 4 cloudless days selected in Fig. 5 at VDA. A curve is absent or stops if the effective dose did not produce the biological effect. Two fractions of exposed skin are considered: 10% (face-hands, typical of late autumn, winter and early spring exposure in mainland) and 65% (face-hands-arms-legs, typical of late spring, summer and early autumn exposure in mainland and exposure throughout the year in the tropics). Two phototypes frequently encountered in all studied areas (plots (a) for phototype II and (b) for type V are considered. Other phototypes are available in supplementary material (Figs. S2-S3)). Near winter solstice, there is no erythema risk, except for phototype I. Individuals need to expose 65% BSA to synthetise sufficient vitamin D without burning. At other times, on the three other days, for 10% of BSA exposure, t_{ery} is longer than t_{vitD}, so sufficient vitamin D is produced before erythema occurs. For 65% BSA exposure, $t_{vitD}(65\%)$ is short (e.g. $< \sim$10 min and

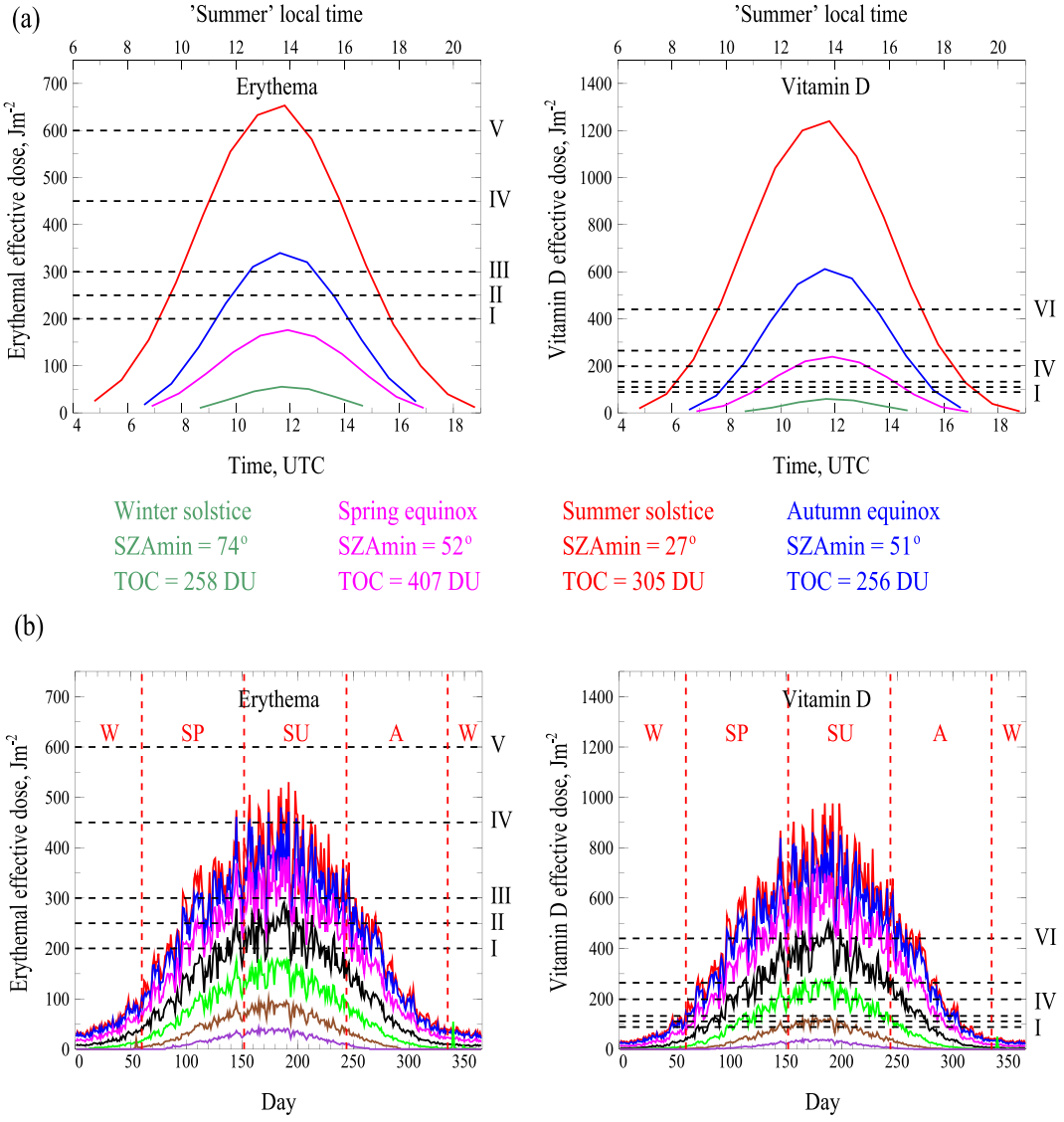

Fig. 5. 1-hour doses computed in a ± 0.5 h window centered at several times at VDA. Left plots for erythema, right plots for Vitamin D. (a) Diurnal variations for 4 cloudless days close to winter and summer solstices (green and red lines respectively) and to spring and autumn equinoxes (magenta and blue lines respectively). SZAmin (average over 1 hour) and TOC from OMI are indicated. The 'summer' local time (UTC + 2 h, SLT), available from approximately end of March up to end of October, is indicated on the top axes. (b) Seasonal variations of 1-hour doses centered at different times: Red: solar noon – Blue: noon ±1 h – Magenta: noon ±2 h – Black: noon ±3 h – Green: noon ±4 h - Brown: noon ±5 h – Violet: noon ±6 h. See text for more details. Dashed red vertical lines indicate season-like limits (as defined in Fig. 3). Horizontal black dashed lines indicate 1 MED (left plots) and 1 $MDD_{1/4}$ (right plots, face-hands-arms exposure) for all phototypes (see Table 1).

Table 2
Month ranges where effects produced by 1-hour exposure at different times of the day may occur at VDA. Vitamin D production corresponds to a 1000 IU dose obtained while exposing ¼ of body. The numbers refer to month numbers.

VDA	Effect	Noon	Noon ±1 h	Noon ±2 h	Noon ±3 h	Noon ±4 h
Type I	Erythema risk	[04–09]	[04–09]	[mid04–mid09]	[mid05–mid08]	Never
	Vit D	[mid02–mid11]	[mid02–mid11]	[03–10]	[mid03–mid10]	[04–mid09]
Type II	Erythema risk	[04–09]	[mid04–mid09]	[05–08]	[06–07]	Never
	Vit D	[03–10]	[03–10]	[03–mid10]	[mid03–09]	[mid04–08]
Type III	Erythema risk	[mid 04–08]	[mid04–08]	[mid05–mid08]	Never	Never
	Vit D	[03–10]	[03–10]	[mid03–mid10]	[04–09]	[05–08]
Type IV	Erythema risk	[06–07]	[06–07]	Never	Never	Never
	Vit D	[mid03–mid10]	[mid03–mid10]	[03–09]	[mid04–mid09]	[06–07]
Type V	Erythema risk	Never	Never	Never	Never	Never
	Vit D	[mid03–mid10]	[mid03–mid10]	[04–09]	[05–08]	Never
Type VI	Erythema risk	Never	Never	Never	Never	Never
	Vit D	[04–09]	[04–09]	[05–08]	[07]	Never

Table 3
Same as Table 2 but at OHP.

OHP	Effect	Noon	Noon ± 1 h	Noon ± 2 h	Noon ± 3 h	Noon ± 4 h
Type I	Erythema risk	[03–10]	[03–10]	[mid03–mid10]	[mid04–mid09]	[06–07]
	Vit D	[mid01–11]	[mid01–11]	[mid02-mid11]	[03–10]	[04–09]
Type II	Erythema risk	[03–mid10]	[mid03–mid10]	[mid04–09]	[04–08]	Never
	Vit D	[02–11]	[02–11]	[mid02–mid11]	[03–10]	[mid04–mid09]
Type III	Erythema risk	[mid03–mid10]	[04–mid10]	[mid04–09]	[mid05–08]	Never
	Vit D	[02–mid11]	[02–mid11]	[mid02–10]	[mid03–mid10]	[mid04–mid09]
Type IV	Erythema risk	[mid04–mid09]	[05–mid09]	[mid05–mid08]	Never	Never
	Vit D	[mid02–mid11]	[mid02–mid11]	[03–10]	[mid03–09]	[05–08]
Type V	Erythema risk	[mid06–mid08]	[mid06–08]	Never	Never	Never
	Vit D	[mid02–10]	[mid02–10]	[03–mid10]	[04–mid09]	[mid05–mid08]
Type VI	Erythema risk	Never	Never	Never	Never	Never
	Vit D	[mid03–mid10]	[mid03–mid10]	[04–09]	[mid05–08]	Never

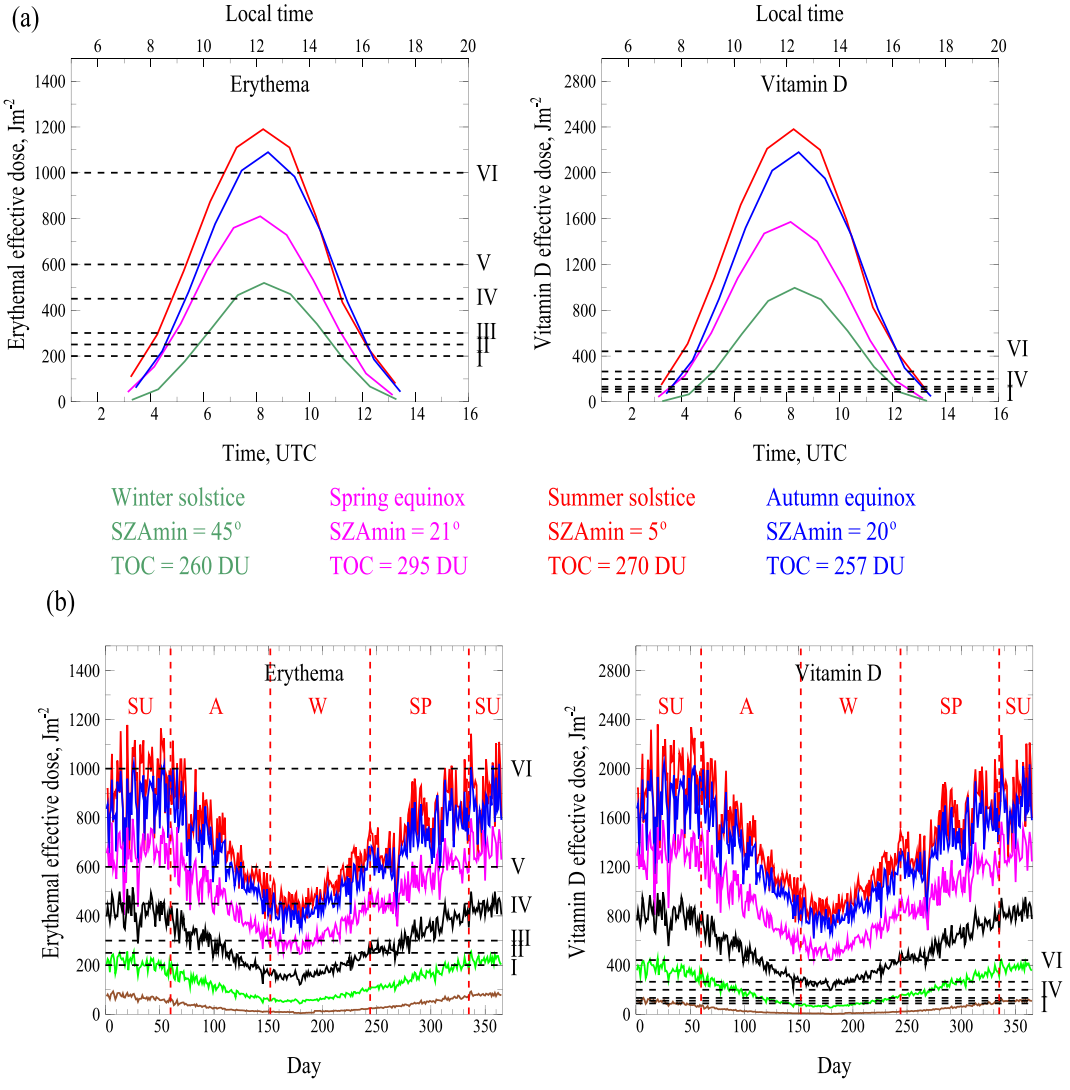

Fig. 6. Same as Fig. 5 but at SDR. The local time (UTC + 4 h) is indicated on the top axes of (a).

25 min for types II and V respectively). The flat curves indicate a wide period around solar noon favorable for relatively rapid vitamin D production without burning.

OHP results for the days presented in Fig. S1are exhibited in Figs. S4-S5-S6. Exposure durations are shorter than at VDA by a factor 2–3 in winter/spring and about 1.5 in summer/autumn.

Predictably, the SDR duration times (Fig. 9 and Figs. S7-S8) for the 4 days highlighted in Fig. 6a, are much shorter than on mainland France: in winter the difference is about a factor 8 compared to VDA, in spring/autumn ~4 and in summer ~2. Factorial differences reflect ozone and SZA differences between sites (see Figs. 5–6 and S1). At SDR, t_{ery} is always longer than t_{vitD} and in winter $t_{vitD}(10\%)$ is short.

Time durations were computed as previously for each day of the 2009–2018 period, for phototypes and the two previously utilized BSA exposed at several initiating times. Climatological seasonal variations of duration times are shown in Fig. 10 for exposure initiating at solar noon

Table 4
Same as Table 2 but at SDR.

SDR	Effect	Noon	Noon ± 1 h	Noon ± 2 h	Noon ± 3 h	Noon ± 4 h
Type I	Erythema risk	Always	Always	Always	[mid08–mid05]	[12–mid02]
	Vit D	Always	Always	Always	Always	[mid08–mid05]
Type II	Erythema risk	Always	Always	Always	[09–04]	Never
	Vit D	Always	Always	Always	Always	[09–04]
Type III	Erythema risk	Always	Always	[mid07–05]	[10–mid04]	Never
	Vit D	Always	Always	Always	Always	[09–04]
Type IV	Erythema risk	Always	[mid07–05]	[09–mid04]	[12–mid02]	Never
	Vit D	Always	Always	Always	Always	[10–mid03]
Type V	Erythema risk	[mid08–mid05]	[09–mid04]	[11–mid03]	Never	Never
	Vit D	Always	Always	Always	[mid07–05]	[11–mid03]
Type VI	Erythema risk	[12−02]	Never	Never	Never	Never
	Vit D	Always	Always	Always	[09–mid04]	Never

for phototypes II and V (other phototypes are available in Figs. S9-S10).

Fig. 10a,b and Figs. S9a,b - S10a,b show that for exposure from noon at VDA, from approximately November to February, there is no erythema risk and sufficient vitamin D synthesis is not achievable from exposure of 10% of BSA. From March to October t_{vitD}(10%) is shorter than t_{ery}. Exposing 65% of BSA strongly decreases t_{vitD} all year round, and there is no risk of burning with these t_{vitD} durations. Erythema risk is high in summer for all phototypes, including type VI whose risk is weaker (t_{ery} ~3 h) albeit still important since summer is favorable to long sun exposure. Spring is not safe as phototypes I-III are erythema susceptible, and may develop erythema in less than one hour from April to September (Table 6).

At OHP (Fig. 10c,d, Figs. S9c,d - S10c,d and Table 6) inductive durations are shorter than at VDA by a factor of 1.5–2.5 in spring, summer and autumn, and up to ~6 in winter.

At SDR (Fig. 10e,f and Figs. S9e,f - S10e,f), inductive durations are shorter than at VDA with factors in the 2–10 range, depending on season. During the entire year it is possible for all phototypes to synthesize sufficient vitamin D without burning, exposing as little as 10% of BSA. When 65% skin surface area is exposed, t_{vitD} is very short, even in winter. Erythema risk is very high except in winter for types V-VI (Table 6).

On the mainland, for an exposure initiated 3 h before noon (Figs. S11a,b,c,d - S12a,b,c,d - S13a,b,c,d), duration times in summer are longer than at noon by a factor of about 1.2–2. For an exposure initiated 3 h after noon (Figs. S14a,b,c,d - S15a,b,c,d - S16a,b,c,d), summer duration times are also longer than at noon by about 2–2.5. So, as expected, for these two exposure periods, erythema risk is less than near to noon. Risk does exist for some phototypes since summer favors long sunbathing. All phototypes can synthetize vitamin D in less than ~1 h after exposing 65% of BSA. (See Tables S1-S2 in supplementary material).

At SDR, for an exposure initiated 3 h before noon (Figs. S11e,f - S12e, f - S13e,f), duration times are longer than at noon by a factor of about 1.5–2.5 depending on season. It is possible for all phototypes to synthesize sufficient vitamin D without burning after exposure of 10% of BSA. Though less than close to noon, erythema risk is not negligible, even in winter, for phototypes I-III ($t_{ery} < 1$ h). 3 h after noon (Figs. S14e, f-S15e,f-S16e,f), sufficient vitamin D synthesis is possible in winter in less than ~1 h (type VI excepted) while exposing 60% of BSA. Erythema risk is still high with $t_{ery} < 1$ h for types I-II in summer. (See Tables S1-S2).

Note that, since prior analyses involved averaged values of time durations over 10-years, they do not represent extreme cases (cloudless and overcast) and this may lead to large variations in results.

As explained in Section 3.1, mean UVI in a 1-hour window may be a simple and convenient indicator of erythema risk, highly relevant for hourly UVI forecast (Fig. 4, around noon). Fig. 11 displays the 1-hour UVI 3 h before noon. At VDA (Fig. 11a), UVI is on average in the [0–3.5] range, i.e. low/moderate but with a large variability (summer peak at ~5). For a UVI of 3 (seen in VDA in summer), by using the MED values adopted in this study, phototypes I-II may burn in less than ~1 h, confirming previous results (Table S1). At OHP (Fig. 11b) UVI is also low/moderate ([0.2–5] range, peak at ~6). For a UVI of 3, erythema risk at less than 1 h exposure exists in late spring/summer for phototypes I-II. At SDR (Fig. 11c) the average range is [2.5–7] (i.e. moderate/high with a very high summer peak at ~9). For a UVI of 3, phototypes I-II are at risk of erythema after less than 1 h exposure, except in winter. Since UVI is often higher than 3, other phototypes may also be at risk after less than 1 h exposure at these times.

Results for 3 h after noon are displayed in Fig. S17. At VDA (Fig. S17a), UVI are almost always lower than 3, UVI is larger than 3 in June/July at OHP (Fig. S17b), and at SDR (Fig. S17c), this situation occurs in late spring, summer and early autumn.

Results for 4 h before noontime (Fig. 12 and Fig. S18) are similar to those 3 h after noon.

3.4. Realistic Body Exposure

The previous doses and times were derived from solar measurements on a horizontal plane that does not represent the shape of the human body. Exposure duration times must be adjusted to compensate for this. According to Pope and Godar [40], (referred to as P&G hereafter), the corrections to change plane irradiances to human body irradiances depend on variables such as the SZA, the orientation of the body part (inclination relative to the horizontal plane and orientation relative to sun direction) and on atmospheric conditions. P&G considered a semi-cylinder model to represent face, hands, shoulders and feet, and a full cylinder model to represent neck, trunk, arms and legs. P&G computed geometric correction factors (GCF, ratios of irradiance on a body-proxy to irradiance on a horizontal plane) for several SZA and orientations of the two cylinder models. For a human body standing/sitting/lying, all different parts can be represented by the average of many semi-cylinder/cylinder orientations. P&G computed erythemally and pre-vitamin D_3 weighted solar UV factors (GCF_{ery} and GCF_{vitD} respectively) for clear sky and overcast conditions.

Our measurements were performed during each day for various SZA and variable atmospheric conditions, but estimating new irradiances at each time would be impracticable.

We limited our study to an estimate of the factors for 1-hour doses at noontime in summer. Examination of Figs. 2 and 3 of P&G indicates that, at VDA (SZA ~30°), GFC are ~0.52 and ~0.35 for semi-cylinder and full-cylinder models respectively, at OHP (SZA ~25°) they are ~0.51 and ~0.34 and at SDR (SZA ~5°) they are ~0.50 and ~0.32. The 3 sites have thus similar correction factors. In summer, 65% of the body is often exposed so both models must be used together leading to an averaged GCF of ~0.42. On average, during one hour close to noon on a CS day in summer, individuals receive less than half the dose measured by the spectroradiometers resulting in duration times more than twice those t_{ery} and t_{vitD} derived from spectroradiometers measurements. Since the GFC are quite similar for erythema and vitamin D, we still have $t_{ery} > t_{vitD}$.

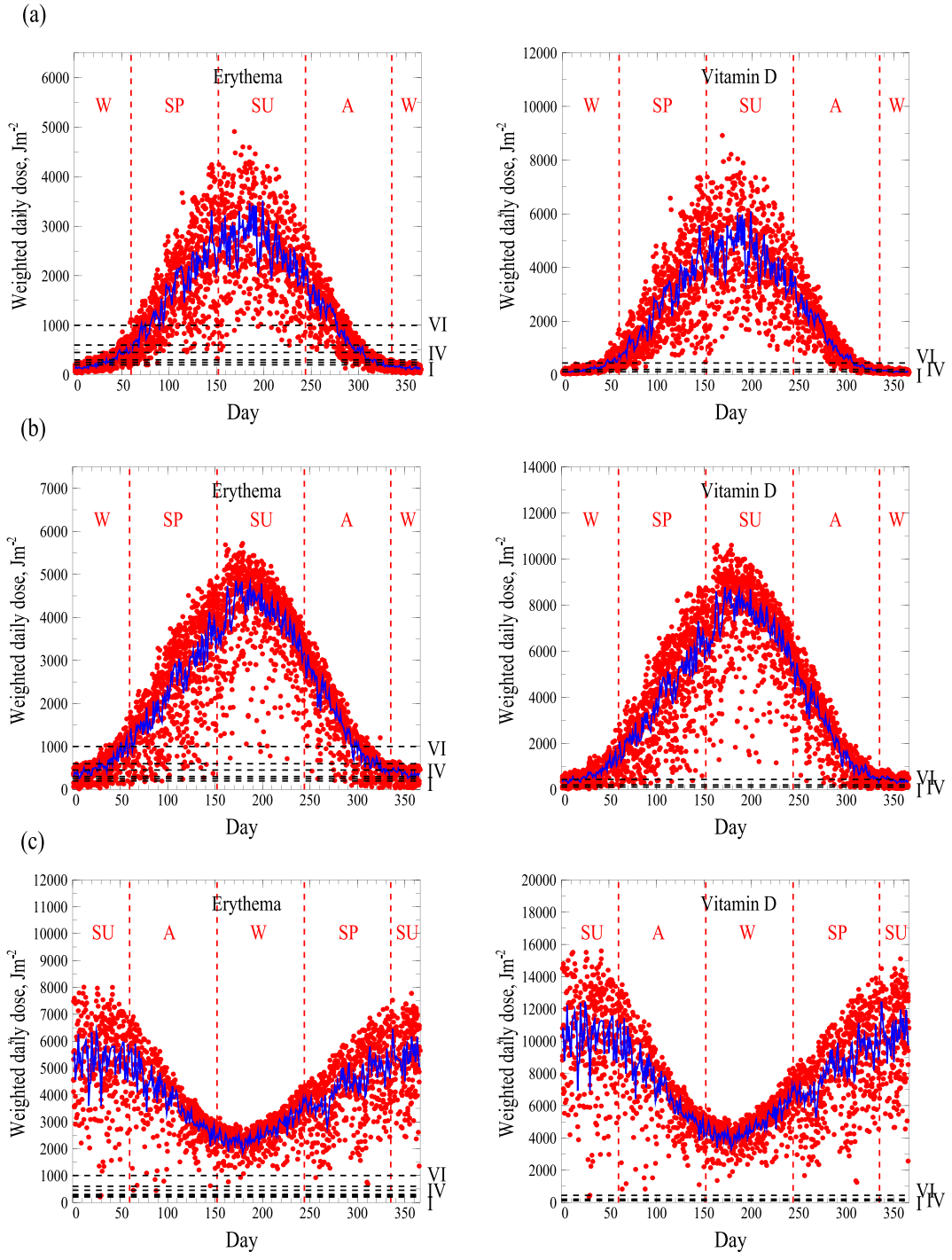

Fig. 7. Annual variations of daily doses at VDA (a), OHP (b) and SDR (c). Left plots for erythema, right plots for Vitamin D. Red dots: daily values, blue solid line: climatology. Horizontal black dashed lines indicate 1 MED (left plots) and 1 $MDD_{1/4}$ (right plots) for phototypes I, IV and VI.

Figs. 4 and 5 in P&G show that, for both cylinder models and both atmospheric conditions, GCF_{ery} and GCF_{vitD} daily values are similar thus an average value may be used. This mean daily GCF varying slowly with time, P&G calculated monthly factors that they separated in two groups corresponding to summer and winter seasons and provided values at several latitudes. There are only small differences between cloudless and overcast values, thus a mean value may be used for climatology studies. Table 7 summarizes the mean GCF at our sites.

We used them to estimate corrections to apply to our daily doses.

At VDA and OHP in summer, when 65% of BSA is often exposed, as previously, both models must be used together leading to a seasonally averaged GCF of ~0.55. In winter, the semi-cylinder model can be used alone (10% of BSA) leading to a GCF of ~0.70. The daily doses measured on a horizontal plane must be multiplied by these GCFs to provide realistic daily doses, and the derived duration times are therefore divided by these coefficients.

At SDR in winter, 65% of BSA is quite frequent, so all year around one has a seasonally averaged GCF of ~0.51. In case of 10% of BSA exposure the GCF would be ~0.64. Thus, the coefficients are not much different from the ones in mainland France, leading to the same conclusions.

On average, in summer at the three sites, individuals receive

Table 5
Month ranges during which effects produced by daily exposure may occur (1000 IU vitamin D for ¼ of BSA) at VDA (top of Table), OHP (middle) and SDR (bottom). Results are based on daily dose climatology. Numbers refer to months.

	Type I	Type II	Type III	Type IV	Type V	Type VI
VDA						
Erythema risk	[02–mid11]	[02–mid11]	[02–mid11]	[mid02–10]	[03–mid10]	[mid03–09]
Vit D	[02–11]	[02–11]	[02–11]	[02–11]	[mid02–mid11]	[mid02–mid11]
'Vitamin D winter'	[12–01]	[12–01]	[12–01]	[12–01]	[mid11–mid02]	[mid11–mid02]
OHP						
Erythema risk	Always	Always	Always	[02–11]	[mid02–11]	[03–10]
Vit D	Always	Always	Always	Always	Always	[02–11]
'Vitamin D winter'	Never	Never	Never	Never	Never	[12–01]
SDR						
Erythema risk	Always	Always	Always	Always	Always	Always
Vit D	Always	Always	Always	Always	Always	Always
'Vitamin D winter'	Never	Never	Never	Never	Never	Never

approximately half of the daily dose measured by a spectroradiometer and therefore the duration times are approximately twice t_{ery} and t_{vitD} relative to a horizontal plane, always with generally $t_{ery} > t_{vitD}$. In winter on the mainland, durations are about 1.5 times the t_{ery} and t_{vitD} derived for a horizontal plane.

Use of GFC values that are averages of various body's parts inclinations for full-cylinder/semi-cylinder models, as we did above, must be interpreted with caution. Indeed, as P&G mentioned in their discussion, a body part might receive more irradiance than a horizontal plane. This occurs when the body surface is oriented toward the sun. This leads therefore to a higher risk of sunburn.

4. Conclusion

Due to location of study sites, erythemal and vitamin D effective dose-rates and doses, as well as exposure times necessary to develop erythema and synthesize desired vitamin level, at the two mainland sites (Villeneuve d'Ascq (VDA) and Observatoire de Haute-Provence (OHP)) differ markedly from Réunion Island site (Saint-Denis de La Réunion (SDR)). SDR is close to the tropic of Capricorn, so high UV radiation levels around midday reflect high sun elevation, especially in summer, and a low total ozone column (TOC). The mid-latitude location of the mainland sites determines lower UV levels than seen in the tropics (lower summer sun elevation around midday and larger TOC). As expected, for the same conditions (day, season, and time of day when relevant), the dose-rates, 1-hour doses and daily doses were significantly larger at SDR than at mainland sites and the two induction times were much shorter at SDR.

Results from solar irradiance measurements on a horizontal plane are summarized below:

- At mainland sites, 1-hour of solar exposure close to solar noon induces erythema in lighter pigmented phototypes (I-III) during most of the year. In the tropics, phototypes IV and V are also at risk of erythema.
- At all sites, 1-hour of solar exposure close to solar noon permits synthesis of desired amount of vitamin D by all phototypes during most of the year.
- Daily vitamin D inducing UV doses are generally well above the $MDD_{1/4}$ values. A 'vitamin-D winter', as defined for ¼ of BSA, is observed at VDA for all phototypes in winter. At OHP such a situation occurs only for type VI in winter. In contrast to the other sites, SDR has no 'vitamin-D winter'.
- Even if only 10% of BSA is exposed, t_{vitD} is always $< t_{ery}$, except in winter at VDA.
- Erythema risk exists for several phototypes for exposure initiated 3 h before noon, mainly in summer, even at VDA. This risk is also present with exposure initiated 3 h after noon in summer at OHP and during most of the year in the tropics.

From Section 3.4, erythemal and vitamin D doses computed from measurements on a horizontal plane do not accurately represent doses received by a human body. Using geometric correction factors (GCF) [40] to represent a more realistic human body, the previous conclusions may be amended as follows:

- The 'vitamin-D winter' is approximately 1–2 months longer than previously assessed for mainland sites. SDR has no 'vitamin-D winter'.
- On average, exposure durations that induce erythema or produce sufficient vitamin D are approximately twice those estimated from spectroradiometer data.

However (Section 3.4), use of a GCF factor for erythema is not appropriate for body parts oriented toward the sun. It is therefore difficult to determine the maximum duration of exposure enabling to avoid erythema at any body site.

Computed times t_{ery} and t_{vitD} are estimates rather than exact values. Indeed: (i) biologically-effective doses are affected by uncertainties due to biologically-effective dose-rates UV_{ery} and UV_{vitD} uncertainties (Section 2, [3,19,42,47,48]); (ii) there exist uncertainties on MEDs values [19]; (iii) MDDs used in the present study are uncertain [20] since they are based on a vitamin D dose of 1000 IU (Section 1) which is highly uncertain [26,29,34,35,37,38]. t_{ery} and t_{vitD}, uncertainties are difficult to calculate since most contributions cannot be precisely determined. A low estimate of these uncertainties maybe derived from UV_{ery}, UV_{vitD}, MED and MDD uncertainties. The resulting uncertainty is at least 30% for both t_{ery} and t_{vitD}.

The 1000 IU recommended vitamin D level we chose enables any reader to easily derive other t_{vitD} for another vitamin D dose choice. We assumed in our calculations that MDD depends on skin type in the same way as MED does. This leads to a strong influence of the skin phototype on vitamin D induction times. If further research shows that this assumption is false, results for skin type II would be more appropriate for other phototypes. Note that time duration values correspond to climatologies, and may be much longer or shorter under specific atmospheric conditions.

WHO [45] provides sun protection recommendations based on UVI values which indicate only erythema risk. Summer protection recommendations outside the tropics when $3 \leq UVI \leq 7$, are to seek shade during midday hours and use clothes/hats/sunglasses and sunscreen on uncovered body parts. The term "midday hours" is insufficiently precise and needs further specification. At VDA we showed that, in summer, an $UVI \geq 3$ may be observed 3 h before noon. The same is true at OHP, but for longer period (late spring/summer/early autumn). At SDR, the period of increased hazard is even longer (spring/summer/early autumn). At OHP, $UVI \geq 3$ occurs 4 h before and 3 h after noon in summer. At SDR, this extended hazard is present in late spring, summer and early autumn. Even when $UVI < 3$, longer exposure may lead to

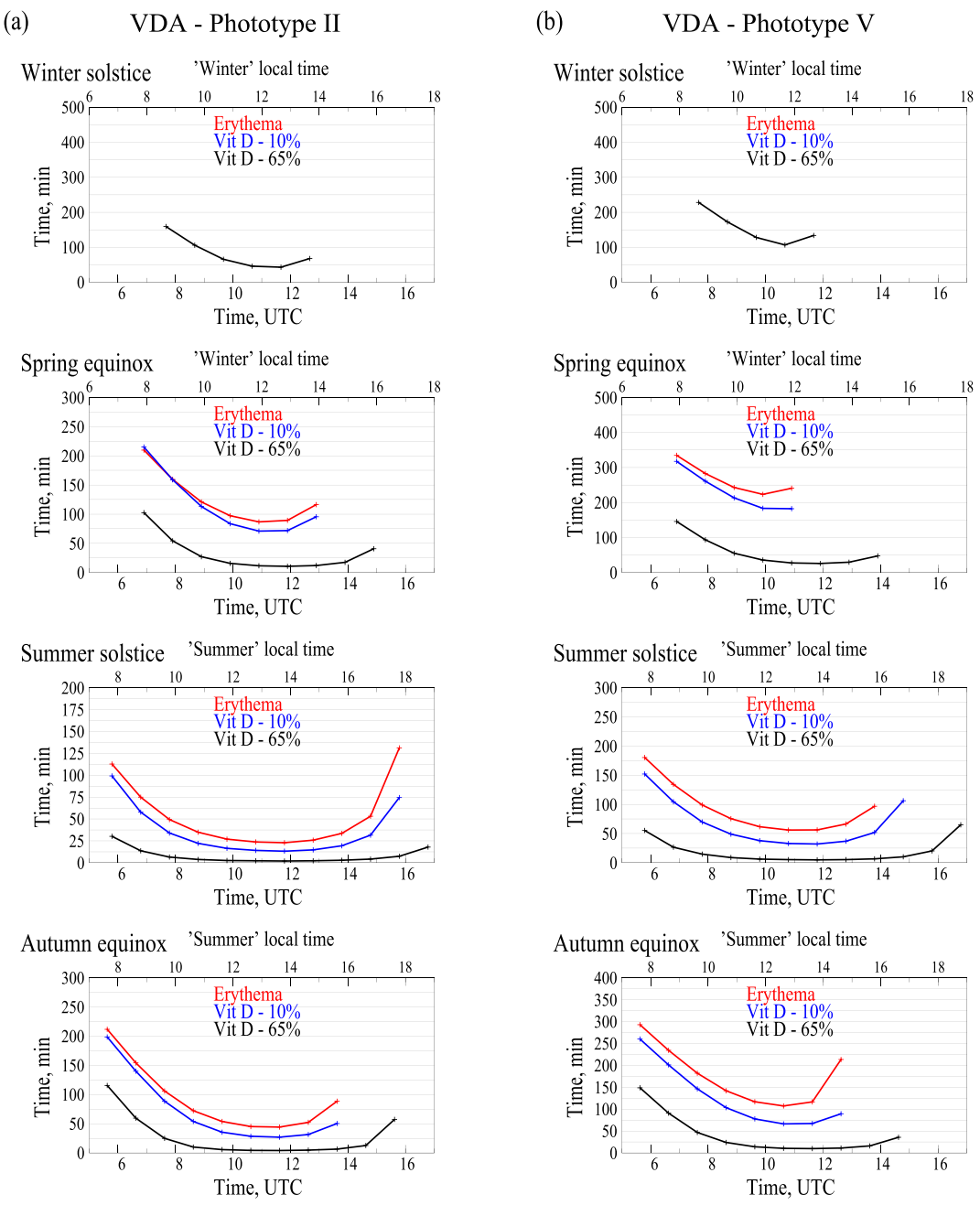

Fig. 8. Time to develop erythema and generate the recommended vitamin D quantity (1000 IU) versus time of initiation of exposure at VDA (local time on top axes), for the 4 cloudless days selected in Fig. 5a. Two BSA are considered for vitamin D: about 10% (face-hands) and about 65% (face-hands-arms-legs). (a) for phototype II, (b) for phototype V.

sunburn, e.g. with a UVI = 2, erythema can appear in less than 1.5 h for phototypes I-III, compared to less than 1 h for UVI = 3. WHO [45] also recommends avoidance of outdoor activities around midday hours when summer UVI ≥ 8. In mainland France, such a high UVI may occur at OHP 1 h before noon (data not shown). At SDR, high UVI is frequent 1 h before or after noon in summer, late spring and early autumn, and in summer even 2 h before noon (data not shown).

Health agency websites (Introduction) recommend limiting solar exposure between 10:00 a.m. and 4:00 p.m. (i.e. 10:00 and 16:00) (WHO), or between 12:00 a.m. and 4:00 p.m. (i.e. 12:00 and 16:00 SLT) (Sécurité Solaire). From our measurements, it is arguable that, in summer, these time periods should be extended. For example solar exposure should be limited, especially for phototypes I-III, and all usual safety measures employed (protective clothing/hats/sunglasses, sunscreen, careful use of shade) from 11:00 to 16:00 SLT at VDA, from 10:00 to 17:00 SLT at OHP and from 8:00 to 15:00 LT at SDR. Summer months are the period of maximum sunburn risk, but websites should mention that spring and autumn are also seasons that need prudence as they too carry some risk of sunburn.

Use of clothes/hats and sunscreen reduces erythema risk (sunscreen increases t_{ery}) but decreases the ability to produce vitamin D (by increasing t_{vitD}), though this latter effect of sunscreen is debated [34,39]. When a sufficient vitamin D production cannot be reached, a solution would be to expose a larger skin area to solar radiation. This approach has the advantage of reducing erythema risk, but is impractical in winter since individuals seldom expose more than face and hands in that season.

It is not necessary to receive the minimum UV_{vitD} dose every day

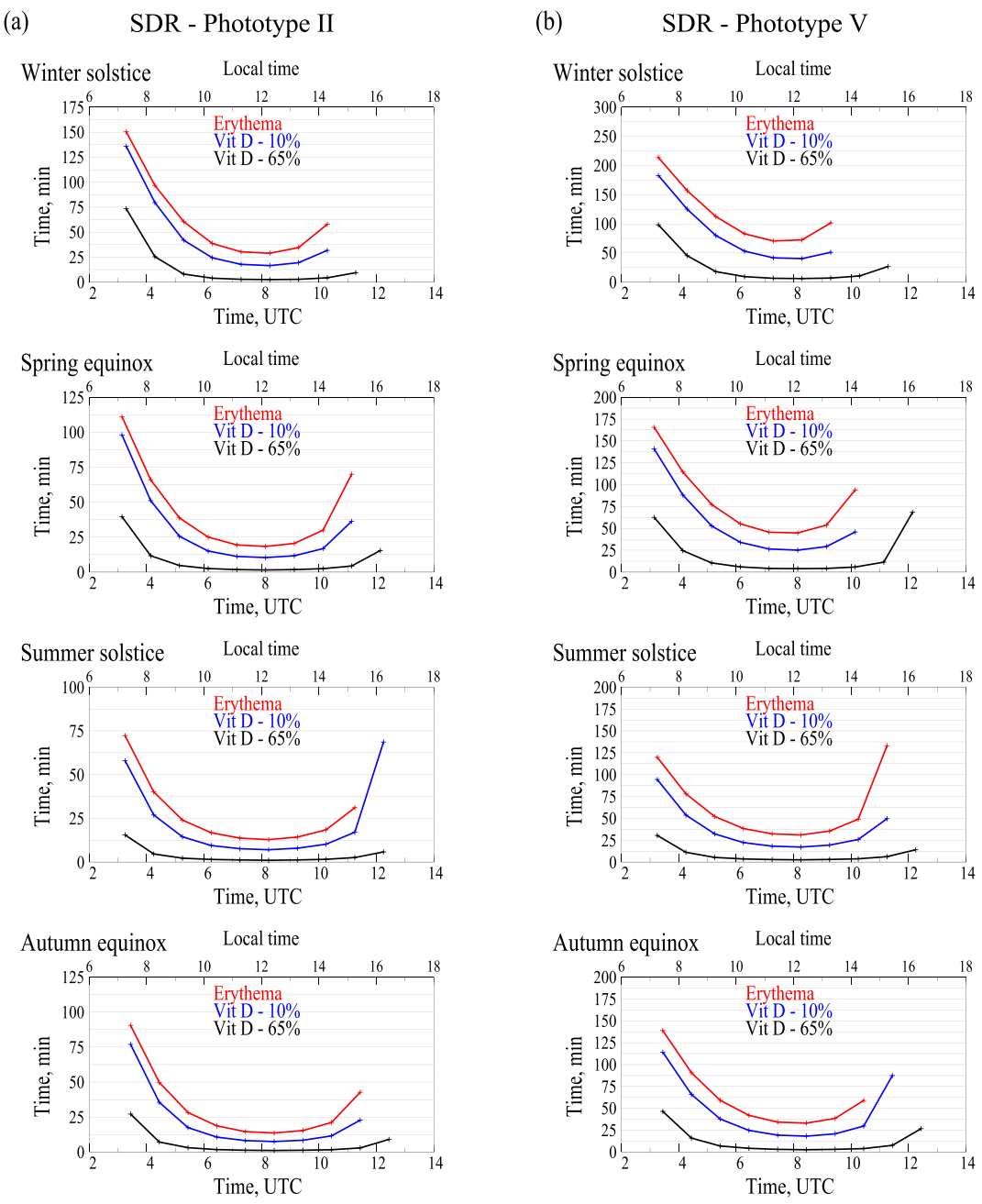

Fig. 9. Same as Fig. 8 but at SDR, for the 4 cloudless days selected in Fig. 6a.

because body fat stores vitamin D [3,48,50], however, the storage time of vitamin D is not well known. Nevertheless, during 'vitamin-D winter', vitamin D supplements may be necessary [3,48].

Note that when considering the various skin area exposed we did not consider that the skin in the different parts of the body is variably pigmented and thus does not synthesize vitamin D with similar efficiency [19,31]. Also, age influences the capacity to synthetize vitamin D [27].

Individual may assess their personal exposure to solar radiation by using software applications developed for smart phones (e.g. UVIMate (for Android and Apple), UVLens (for Android), UVreunion (for Android and Apple)). These take account of a personally selected skin type, and indicate t_{ery} and possibly t_{vitD} as a function of real-time UVI estimated from predicted TOC and cloudiness. However, indicating "burning time" may affect behavior and result in a prolongation of sun exposure. Furthermore, two issues must be considered: (i) UVI is computed for a horizontal plane, therefore the calculated time durations are unrealistic. For example t_{vitD} should be multiplied by approximately 2. Conversely, scaling of t_{ery}, is not advised because of the high risk of burning for body parts facing the sun; (ii) actual cloudiness may be quite different from the predicted one, and solar radiation may be obstructed by vegetation and/or buildings [51], factors not accounted for in the apps.

Use of personal dosimeters has been advocated to precisely determine the UV dose actually received. However, such dosimeters may only record part of the global UV radiation (direct and diffuse) due to their small aperture (usually 110°), orientation and position on the body. To obtain a better estimate of received doses an individual would have to wear many dosimeters distributed over the body, which is of course difficult to achieve for an average person. Therefore the "personal dosimeter" solution is unrealistic and should be reserved for scientific research.

Finding a balance between the risks and benefits of UV solar exposure is complex because the effects depend on many factors (phototype,

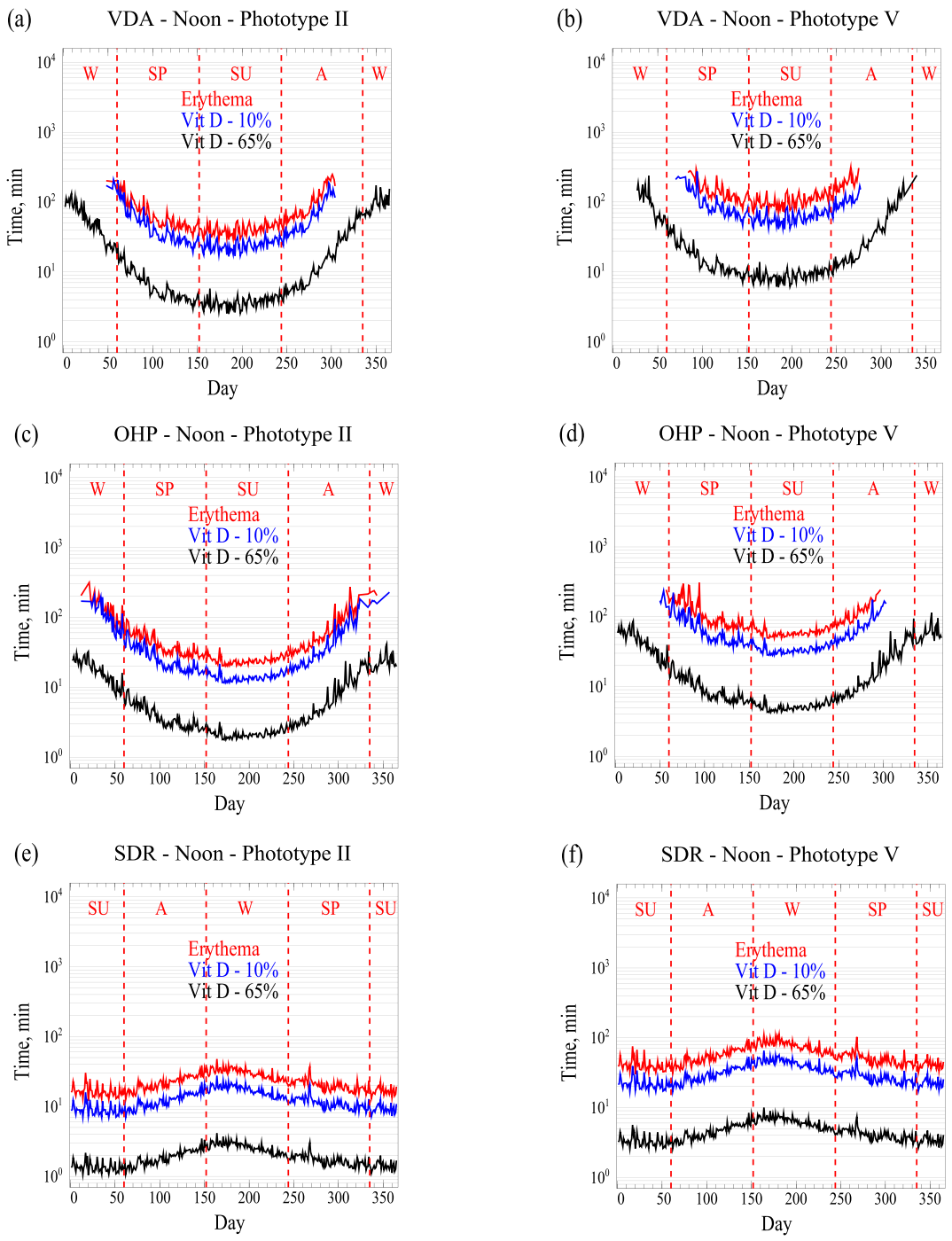

Fig. 10. Climatology of time duration to develop erythema and produce the recommended vitamin D dose (1000 IU) for an exposure initiating at solar noon. At VDA: (a) and (b); at OHP: (c) and (d); at SDR: (e) and (f). (a), (c) and (e) for phototype II, (b), (d) and (f) for phototype V. Two BSA are considered for vitamin D: about 10% and 65%.

age, meteorology, activities, use of protections…), therefore further research (improvement of vitamin D action spectrum, better estimate of the recommended vitamin D dose/day, role of skin pigmentation on vitamin D level, influence of sunscreen on vitamin D production…) is necessary.

Here we only addressed erythema risk and vitamin D synthesis from solar UV exposure, but many other risks and benefits exist. Sun exposure may be a cause of eye damage, immunosuppression, cancer, and it allows formation of compounds such as e.g. nitric oxide, serotonin, melatonin, which have beneficial effects [12,21,22,25]. Vitamin D supplementation does not correct these latter effects [12,52], so in these instances sun exposure is actually needed.

Health agencies/associations have a primary role in prevention of UV-induced damage by delivering messages to populations, media, teachers, physicians, etc., which emphasize that solar radiation exposure is associated with numerous health benefits, but also with significant detrimental effects. The absence of burning does not guarantee the absence of other damage. Sunburn has a UV-dose threshold level, but effects such as induction of skin cancer do not. Recommendations should not be too restrictive in order to favour their acceptance and effectiveness. Whereas public health messages are necessarily rather general, recommendations should ideally be both individually and locally

Table 6
Monthly ranges during which synthesis of recommended amount of vitamin D (1000 IU) is possible after 10% (face-hands) and 65% (face-hands-arms-legs) BSA exposure without burning for an exposure initiated at noon. Between brackets are the shortest and longest t_{vitD} within the month range. The t_{ery} range (summer-winter) and the month range with $t_{ery} < 1$ h are also indicated. Data from VDA are at the top, data from OHP in the middle and data from SDR at the bottom. Results are based on t_{ery} and t_{vitD} climatologies. Numbers refer to months.

% of BSA exposed	Type I	Type II	Type III	Type IV	Type V	Type VI
VDA						
t_{vitD}10%	[03–10] [15 min–2 h]	[03–10] [20 min–2.5 h]	[03–10] [25 min–3 h]	[mid03–mid10] [35 min–3 h]	[04–09] [45 min–3 h]	[mid04–09] [1.5 h–4 h]
t_{vitD}65%	[01–12] [2 min–1.5 h]	[01–12] [3 min–2 h]	[01–12] [4 min–3 h]	[mid01–mid12] [5 min–3 h]	[02–11] [7 min–3 h]	[mid02–mid11] [10 min–3 h]
t_{ery} range	[25 min–3 h]	[30 min–3 h]	[35 min–3 h]	[50 min–3 h]	[1.5 h–4 h]	[2 h–4 h]
$t_{ery} < 1$ h	[04–09]	[04–09]	[mid04–mid09]	[06–07]	[–]	[–]
OHP						
t_{vitD}10%	[02–11] [10 min–1.5 h]	[02–11] [12 min–1.5 h]	[02–11] [15 min–2 h]	[03–10] [20 min–2 h]	[03–mid10] [30 min–3 h]	[mid03–mid10] [50 min–3 h]
t_{vitD}65%	[01–12] [1 min–20 min]	[01–12] [2 min–25 min]	[01–12] [2 min–30 min]	[01–12] [3 min–40 min]	[01–12] [4 min–1 h]	[01–12] [8 min–1.5 h]
t_{ery} range	[15 min–3 h]	[20 min–3 h]	[25 min–3 h]	[40 min–3 h]	[50 min–3 h]	[1.5 h–3 h]
$t_{ery} < 1$ h	[03–10]	[03–10]	[mid03–mid10]	[05–mid09]	[mid06–07]	[–]
SDR						
t_{vitD}10%	[01–12] [6 min–20 min]	[01–12] [7 min–25 min]	[01–12] [8 min–30 min]	[01–12] [12 min–40 min]	[01–12] [20 min–1 h]	[01–12] [30 min–1.5 h]
t_{vitD}65%	[01–12] [1 min–3 min]	[01–12] [1 min–4 min]	[01–12] [2 min–4 min]	[01–12] [2 min–6 min]	[01–12] [3 min–9 min]	[01–12] [4 min–12 min]
t_{ery} range	[10 min–30 min]	[12 min–40 min]	[15 min–50 min]	[25 min–1.5 h]	[30 min–2 h]	[1 h–4 h]
$t_{ery} < 1$ h	[01–12]	[01–12]	[01–12]	[01–05;08–12]	[01–04;09–12]	[01–02;12]

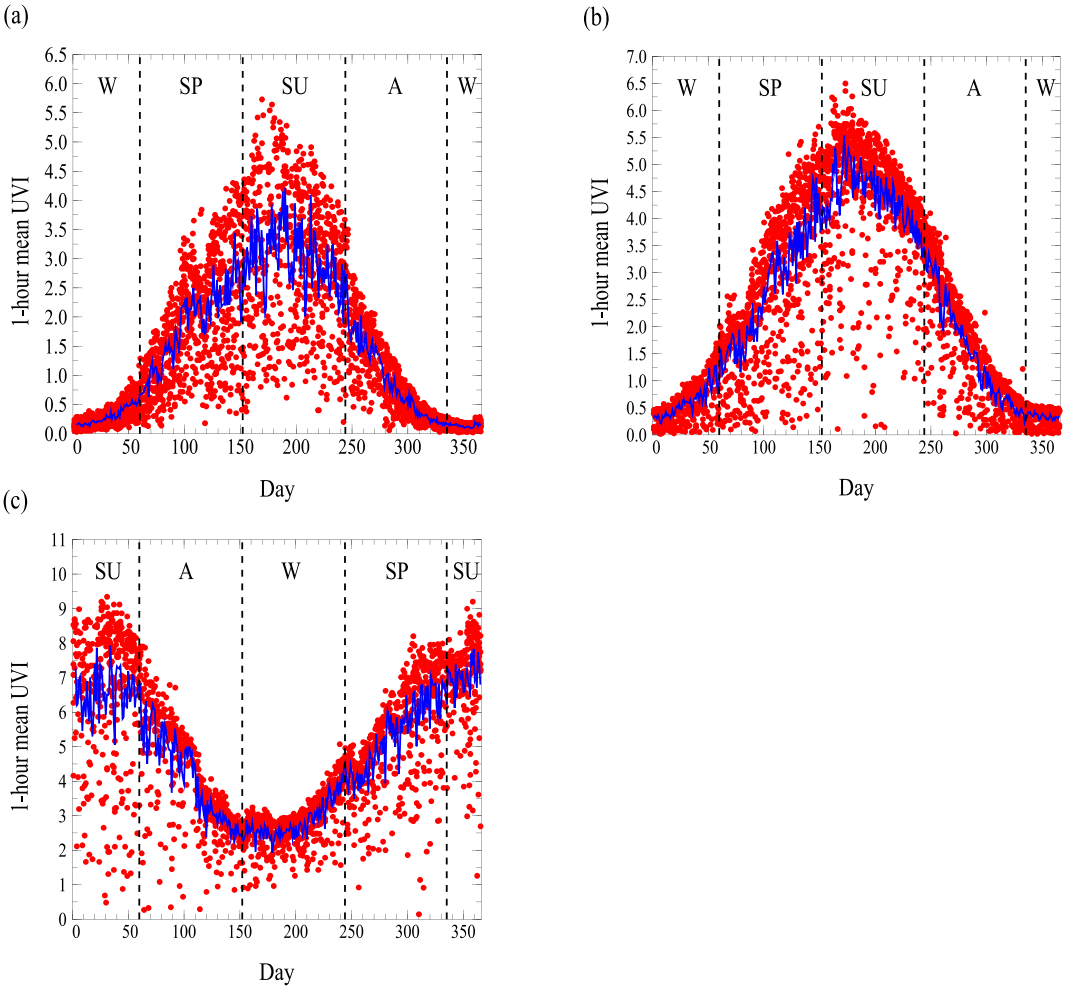

Fig. 11. Same as Fig. 4 but for the mean UVI in a 1-hour window initiated 3 h before noon. (a) At VDA, (b) at OHP, (c) at SDR.

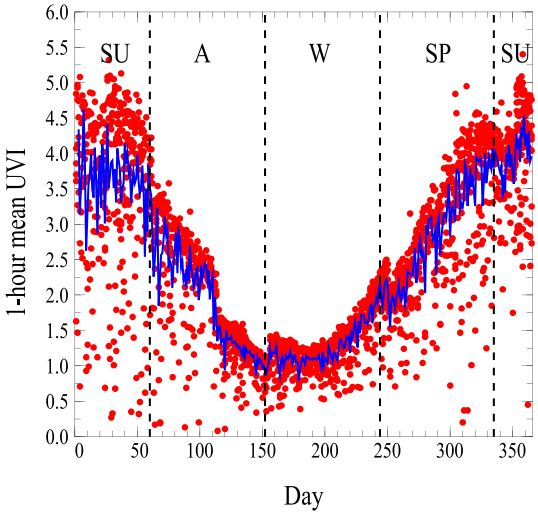

Fig. 12. Same as Fig. 4c but for the mean UVI in a 1-hour window initiated 4 h before noon at SDR.

Table 7
Mean daily GCF (mean of clear sky and overcast conditions's GCF) for the two cylinder models. Semi-cylinder model can represent the face, hands, shoulders and feet, whereas full-cylinder model can represent the neck, trunk, arms and legs.

GCF	Semi-cylinder model		Full-cylinder model	
	Summer	Winter	Summer	Winter
VDA	0.64	0.70	0.46	0.52
OHP	0.63	0.70	0.45	0.51
SDR	0.61	0.64	0.42	0.46

Adapted from Tables 1, 2, 4 and 5 of Pope and Godar [41].

tailored. Thus, knowledge of local UV radiation climatology can be helpful.

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Data Availability

Spectroradiometer measurements are currently available at http://www-loa.univ-lille1.fr/index.php/observation/sites.html and at ftp://ftp.cpc.ncep.noaa.gov/ndacc/station/

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Declaration of Competing Interest

None.

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Appendix A. Supplementary data

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