Some considerations on the protection against the health hazards associated with solar ultraviolet radiation

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Abstract

The present report briefly reviews the basic features of the current strategy for the protection against the health harms caused by solar ultraviolet (UV, ~295 - 400 nm). The emphasis has been made upon the erythema as being the best-studied UV harm and the ability of UV irradiance to damage the deoxyribonucleic acid (DNA) molecules, which leads to carcinogenesis. The erythemally weighted UV irradiance that determines the ultraviolet index (UVI), represents a common measure of the solar UV radiation level at the Earth’s surface and the current protective messages have been made by using UVI as a basic parameter. However, such an approach seems insufficiently grounded to be used also in the messages against the skin cancer, bearing in mind the different nature of the erythema and DNA lesions. In this context, an enlargement of the strategy basis by including additional biological effects of UV radiation studied during the past years has been discussed. For instance, the weight of the spectral UV-A (315 - 400 nm) band that in practice had been neglected by UVI definition can be enhanced since it was found to play an important role in DNA damaging. In addition, features of the contemporaneous lifestyle can be taken into account together with some people habits.

Keywords: Solar UV irradiance, Skin cancer, Erythema, Protection against UV radiation

1. Introduction

Research performed over the last century recognized the solar ultraviolet (UV) radiation reaching the ground (~ 295 – 400 nm) as an environmental factor that is able to cause a variety of damage in the human organism like erythema, cataract and skin aging (Ambach and Blumthaler, 1993; Mullenders, 2018; WHO, 2002; 2003; Delbacq-Chainiaux et al., 2012). In addition, the exposure to solar UV irradiance can trigger carcinogenesis (Elwood and Jopson, 1997; Jhappan et al., 2003; Tucker, 2008; Green et al., 1985; Karran and Brem, 2016; Ambach and Blumthaler, 1993). On the other hand, UV irradiance stimulates the production of vitamin D, which enhances the resistance to various diseases, including cancer (Reichrath, 2014; McKenzie et al., 2009; WHO, 2003; Passeron et al., 2019). Thus, the exposure to solar UV radiation should not be completely avoided, but a balance between damage and benefit need to be sought. All of these circumstances led to the elaboration of rules assuring safety exposure to solar UV radiation and production of supplements, like sunscreen creams that improve an organism’s defence (WHO, 2002; 2003; Harrison et al., 2016). These preventions formed a protective strategy that was aimed at reducing the harmful effects of the solar UV radiation. An important requirement for that is the knowledge about UV irradiance at the surface and its diurnal and seasonal variations. Currently, various satellite devices (Tanskane et al., 2006; Schmalwieser et al., 2017) and ground-based instruments provide both operative information about the distribution of UV radiation and data for studying its behaviour (Bernhard et al., 2015; Bais et al., 2015; Petkov et al., 2014a; Seckmeyer et al., 2019).

However, despite the precautions to reduce the health hazards associated with solar UV radiation, statistics generally shows that the incidence of skin cancer has increased during the past decades although the improvements in the diagnostic approaches (Tucker, 2008; Apalla et al., 2017; Corona, 1996; Lai et al., 2018; WHO, 2003). Guy-Jr et al. (2015) reported a doubling of the melanoma incidence rate in the United States for the period from 1982 to 2011. Wallingford et al. (2015) found a nearly 2% increase of melanoma occurrences per year for young English people (at the age between 14 and 24 years). Meantime, the 6-12% decrease of Australian youth was assumed to be a result of the protective strategies. The cases of basal cell carcinoma (BCC) that is the most spread malignancy in Caucasian population were found to increase with a rate of about 2% per year in Australia followed by 0.4% per year in the United States and nearly 0.15% per year in Europe (Lai et al., 2018; Apalla et al., 2017). According to other estimates the BCC occurrences in the United States rose from 20% to 80% during the period between the mid 1970 and 2006, while the incidence of squamous cell carcinoma (SCC) increased with a rate of 3-10% per year for the same period (Wadhera et al., 2006). The number of SCC cases duplicated from 2004 to 2012 in Belgium (Callens et al., 2016) and a doubling for man and a tripling for women were registered in Denmark between 1978 and 2007 (Birch-Johansen et al., 2010).

Such a brief extract from the statistics of the skin cancer occurrences lead to the impression of gaps in the current protec-
tive strategy against the solar UV radiation since the exposure to UV irradiance was indicated as the main factor for increasing cancer incidence by most of the authors who provided the statistical data. The next sections discuss the basis of the present protective scheme and some new achievements in the researches related to the UV radiation effects in the organisms. An emphasis was made on the impact of UV radiation on the structure of the deoxyribonucleic acid (DNA) molecule and assumptions about an enlargement of the protective strategy basis have also been discussed.

2. Solar UV irradiance at the ground and its impact on the human organism.

Being the main energy source for the Earth, the solar radiation is one of the most important environmental factors that drives various chemical and dynamical processes in the atmosphere, hydrosphere and biosphere (Melnikova and Vasilyev, 2005). Emitted by the Sun electromagnetic radiation pertains to a large spectral range starting from X-rays (10⁻²⁻⁰⁴ m) and reaching to the radio waves 10⁻¹⁰ m (Brasseur and Solomon, 2005). UV irradiance occupies the range from 100 nm to 400 nm (from 1 · 10⁻⁷ to 4 · 10⁻⁷ m) and in turn, it is subdivided into UV-C (100 - 280 nm), UV-B (280 - 315 nm) and UV-A (315 - 400 nm) spectral bands (WHO, 2002). The radiation pertaining to the 400 - 700 nm wavelength interval, which can be felt by the human eye, composing the visible spectral band, while the wavelengths from 700 nm to 10⁹ nm are named as infrared radiation. The last two spectral ranges together with UV band contain about 99% of the solar irradiance reaching the Earth surface.

Figure 1 presents the spectral distribution of the solar UV radiation entering the terrestrial atmosphere and an example of the typical spectral radiation reaching the Earth’s surface. As can be seen the wavelengths below about 295 nm turn out to be strongly attenuated in the atmosphere that is due to the absorption mainly by the oxygen (until about 250 nm) and ozone in the stratosphere (Brasseur and Solomon, 2005). Figure 1 shows also that UV-A solar irradiance at the ground is more intensive than UV-B by about an order of magnitude.

UV radiation possesses enough energy to affect bioprocesses in the human’s organism, focusing its impact on the skin and eyes that realize contact with the environment. The upper skin layer, the stratum corneum that is built by dead keratinocytes absorbs significantly in UV-B spectral range (Bruls et al., 1984) reducing its penetration into the inner epidermis layers. It should be mentioned that the thickness of the stratum corneum could change depending on the skin hydration and solar UV irradiation (Fogawa et al., 2007; Bouwstra et al., 2003; Bruls et al., 1984). Nevertheless, a certain amount of UV-B together with UV-A radiation can reach the internal epidermis strata causing various damage effects, such as erythema, skin aging, photodermatoses and are able to trigger carcinogenic processes in the human skin (Ambach and Blumthaler, 1993; Yu and Lee, 2017; Elwood and Jopson, 1997; Jhappan et al., 2003; Setlow et al., 1993; Boukamp, 2005; WHO, 2003).

The erythema, which is one of the more frequently occurring skin damages is an inflammatory dermatological effect that appears several hours after the exposure to the UV radiation and it is characterised by red skin due to the enhanced blood flow in the stricken area (Agache et al., 1998; Flare, 1955; Nishigori, 2014; Roy and Gies, 2017). In light skin people (skin type II-IV, WHO (2002)), such a condition subsides within several days passing to the tanning that is determined by enhanced melanin concentration in the upper epidermis layers (D’Mello et al., 2016; Baker et al., 2017; Maddodi et al., 2012; Kaur and Saraf, 2011). It is usually considered that this enhancement is due to induced production of melanin by melanocytes, while recent researches indicated that solar UV irradiance stimulates its transport from the basal layer (Tadokoro et al., 2005). Together with the stratum corneum, the melanin is an additional epidermal shield that reduces the propagation of UV irradiance into the deeper skin layers because of its absorption in this spectral range (Anderson and Parriss, 1981; Brener and Hearing, 2008; Solano, 2014; Premi et al., 2015).

UV radiation is able to impact also the sub-cellular structures such as the DNA molecules causing an array of genomic mutations (Ravanat et al., 2001; Mullenders, 2018) that can lead to cancer formation. The human organism possesses various mechanisms able to repair the lesions in DNA caused by UV irradiance and the efficiency of these mechanisms is very important for the human health (Sinha and Hader, 2002; Rastogi et al., 2016; Mouret et al., 2008; Smith et al., 2000; Bustamante et al., 2019). It was established that the cell’s inability to repair the DNA damages is one of the factors that could lead to carcinogenesis (Jhappan et al., 2003; Lehmann et al., 2011; Yu and Lee, 2017).

The propagation of the solar UV irradiance through the atmosphere is characterised by enhanced scattering from the gas molecules (Rayleigh scattering) that is much more intensive for the shorter wavelengths. As a result, the diffuse component of the solar UV irradiance at the ground turns out to be higher than the corresponding components in the visible and infrared bands. Figure 2 shows time-patterns of the direct and diffuse solar irradiance at three wavelengths for clear sky conditions as were evaluated by the Tropospheric Ultraviolet-Visible (TUV) radiative transfer model (Madronich and Flocke, 1997). It is seen that the diffuse component is almost as intensive as the direct one for UV-B wavelengths, which relation gradually changes towards longer wavelengths. The diffuse radiation becomes nearly half as much than direct for UV-A and an order of magnitude less intensive for visible and infrared (the case not shown in the figure) spectral bands. Bearing in mind these features it can be concluded that an efficient protection against the visible or infrared solar radiations would be achieved by reducing the corresponding direct components, or staying in shadow, for instance. However, this is not a reliable protection against the UV irradiance since the diffuse component that could arrive from any direction is not completely stopped. In addition, it should be pointed out that we cannot feel the level of solar UV irradiance as we could have an idea about intensity of the visible and infrared radiation through the eyes and sensation of heat by the skin, respectively. These circumstances would not allow us
to have a foreboding of the peril in some specific conditions like those in high mountain, above about 2000 m, where the coming UV irradiance is higher than at sea level (Siani et al. 2008). Additional forcing factors could be a surrounding of snow covered surfaces and the presence of haze that reduces the visible irradiance, making an impression of low irradiation. Actually, such environment can contribute to appreciable enhancement of the diffuse UV irradiance since the snow reflects more than 70% of UV and the haze, especially if it is composed by very small particles could additionally scatter the light according to Rayleigh law. These conditions are very dangerous for the skin and the eyes since they lead to erythema and/or photokeratitis. Similar damage could occur after a long staying under an umbrella on the beach, where the umbrella creates the feeling of protection. However, the elevated reflection capacity of the sand in UV band (about 20%) together with the reflection from the water and Rayleigh scattering enhance the diffuse component of the solar UV radiation.

Above extreme cases, as well as the usual everyday UV exposure imply the knowledge of the UV irradiance level as an important requirement for effective protection. For that reason continuous observations of solar UV radiation have been developed within the past few decades and nowadays the information about the solar UV irradiance is available for almost all over the world (Tanskanen et al. 2006; Schmalwieser et al. 2017; Mims-III et al. 2019).

### 3. Basic features of the present solar UV protective strategy.

Current messaging around sun protection to avoid the harmful effects of exposure to solar radiation requires the monitoring of the solar irradiance at the Earth’s surface. Such activity allows the estimations of the solar energy (dose) absorbed by a unit horizontal surface and, as a result, the elaboration of recommendations about exposure to solar irradiance. Since the photobiological effects are spectral sensitive, the dose, related to a certain damage has its specific meaning that is the subject of the next subsection.

#### 3.1. Biologically active UV doses and estimation of the irradiation times.

The effects produced by solar UV irradiation in human organism depend on the wavelength $\lambda$, so that the radiation at different wavelengths has different capacity to produce a certain effect and the function determining this ability is named as action spectrum $A_{\text{eff}}(\lambda)$. Weighting the spectral solar irradiance $I(\lambda, t)$ at time $t$ by the action spectrum we obtain the radiation $I_{\text{eff}}(t)$, able to produce the considered effect in humans:

$$I_{\text{eff}}(t) = \int_{280\text{nm}}^{400\text{nm}} I(\lambda, t) A_{\text{eff}}(\lambda) \, d\lambda.$$

Figure 3(a) exhibits the action spectra of the (i) erythema $\Delta E_{\lambda} (\lambda)$ (McKinlay and Diffey 1987); (ii) DNA damage $\Delta E_{\lambda} (\lambda)$ (Setlow 1974); (iii) skin cancer in albino hairless mice $A_{\text{PCF}}(\lambda)$, which was corrected for human skin (Grujil et al. 1993); and (iv) vitamin D action spectrum $\Delta E_{\lambda} (\lambda)$ (MacLaughlin et al. 1982). Figure 3(b) shows the irradiances $I_{\text{eff}}(\lambda, t)$ for different wavelengths that indicate the wavelengths contributing corresponding $I_{\text{eff}}(t)$. It can be seen that the UV-B band presents a dominant role in all selected weighted irradiances. In fact, as Fig. 3 indicates, the weight of the UV-A band determined by the action spectra is even lower than 2 order of magnitude.

For the purposes of the protective messages the dimensionless UV index (UVI) was determined as $\text{UVI}(t) = I_p(t)/(0.025 \text{ Wm}^{-2})$ (WHO 2002; Lucas et al. 2018).

The solar energy associated with a certain effect and absorbed during the time interval $\Delta T_{\text{eff}}$, or the corresponding dose $D_{\text{eff}}$ can be evaluated as:

$$D_{\text{eff}}(\Delta T_{\text{eff}}) = \int_{\Delta T_{\text{eff}}} I_{\text{eff}}(t) \, dt.  \quad (2)$$

In a particular case when the action spectrum is $A_{\lambda} = 0$, for $280 \leq \lambda \leq 315 \text{ nm}$ and $0$, for $315 < \lambda \leq 400 \text{ nm}$ or $A_{UV-A} = (0$, for $280 \leq \lambda \leq 315 \text{ nm}$ and $1$, for $315 < \lambda \leq 400 \text{ nm}$), Eqs. (1) and (2) give the UV-B and UV-A irradiances and doses, respectively.

If we know the solar irradiance coming on the skin together with the action spectrum $A_{\text{eff}}(\lambda)$ and the minimal dose $D_{\text{eff}, \text{min}}$ that is able to cause a certain effect, the Eqs. (1) and (2) allow the estimation of the exposure time $\Delta T_{\text{eff}, \text{min}}$ needed to achieve the dose $D_{\text{eff}, \text{min}}$. In case of damage effect the irradiation above the time $\Delta T_{\text{eff}, \text{min}}$ enhances the probability of the damage occurrence and this time appears to be a parameter quite appropriate for protective messages.

Among all the known damages caused by UV radiation, the erythema, which has been examined from the beginning of the last century (Coblentz and Stauff 1934; Luckiesh et al. 1930; Anders et al. 1995; Dornelles et al. 2004) turns out to be the deepest studied. As a result, the erythema action spectrum $A_{\lambda} (\lambda)$ together with the threshold dose $D_{\text{E}, \text{min}}$, named as minimal erythema dose (MED) have been reliably determined. Bearing in mind the different sensitivities of the people to UV radiation, six types of skin were determined (Fitzpatrick 1988; Roy and Gies 2017). Measurements of the corresponding MED (Dornelles et al. 2004; Bielauksiene et al. 2019) allowed the estimation of the exposure period $\Delta T_{\text{E}, \text{min}}$ within which the erythema can be avoided (McKenzie et al. 2009). Actually, such an estimation can be currently made only for the erythema because $A_{\text{eff}}(\lambda)$ and $D_{\text{eff}, \text{min}}$ for other damages caused by solar UV radiation have not been adopted yet. While the erythema was studied on voluntaries, the examinations of the DNA damages were made on animals or through in vitro experiments that creates some restrictions for applying them to humans (Setlow 1974; Grujil et al. 1993; He et al. 2006; Wischnermann et al. 2008). For that reason, the erythematous solar radiation, especially UVI has become the basic parameter for the protective strategies.
3.2. Protection against the damages induced by solar UV radiation.

The estimation of irradiation time allows the elaboration of protective messages that suggest some rules about the exposure to solar light depending on the current UVI \cite{WHO2002}. Such rules, aimed to avoid erythema include the duration of the exposure for different skin types, depending on the season and geographical location, recommendations about the clothes, etc. In case of need, one could estimate the individual irradiation time for both erythema damage and vitamin D production knowing his skin type, solar irradiance and the sun protection factor (SPF) of the sunscreen used \cite{Antoniou2008, McKenzie2009, Passeron2019}. A method for more precise estimation of the doses that takes into account the specific shape of the human body was also elaborated \cite{Seckmeyer2013} together with empirical approaches connecting the doses really absorbed by the skin with the doses on horizontal surface that are usually provided by the solar irradiance instruments \cite{Siani2008}.

The tanned fair skin assumes an enhanced protective capacity of the organism despite that it can assure only a limited protection corresponding to SPF of no more than 4 \cite{WHO2002, Wood1999}. Hence, the caution about solar UV exposure should not be ignored after tanning.

Generally speaking, above the description presents the core of the current protective strategy against the damages associated with the solar UV radiation. It was constructed on the basis of erythema, since it uses UVI and $D_{E, min}$ for protective messages. However, an important question can be asked: Is this strategy really able to provide as reliable defence against the possible triggering of skin cancer as the defence against the erythema? Indeed, the DNA absorption spectrum is very similar to the erythemal action spectrum \cite{Kiefer2007} but this is not a sufficient ground for a positive answer. The erythema is an immediate effect of the UV irradiation and it was assumed that the risk of long-term cancer formation decreases if the sunburning can be avoided \cite{Wood2017, Nishigor2014, Green1985, Elwood1997}. Such assumption was based mainly on epidemiological studies and it could hardly give any quantitative relationships, which in turn would allow the elaboration of concrete rules as in the case of erythema.

The progress in the research about the ability of UV irradiance to trigger carcinogenesis could be used to improve the protective strategy against the harms of solar radiation. Following the current approaches such improvement could be achieved by introducing additional parameters that describe the recently examined effects and by assessing their impact on exposure time. Similarly to the role of the skin types for erythema the parametrization of a certain effect requires a common classification of the main individual features that would allow to launch protective messages to public. In other words, the basis of the strategy should be enlarged considering not only erythema.

4. Additional effects that could be used for protective messages.

As pointed out in the previous section, the current protective strategy concerns in practice only UV-B solar radiation. However, despite that the absorption of DNA molecule in UV-A band is negligible the latest researches clearly showed that UV-A irradiance is an important factor for carcinogenesis as well \cite{Agar2004}. This is mainly due to its ability to affect DNA indirectly through its capacity to induce the generation of reactive oxygen species (ROS). Such oxidative stress damages the DNA nucleobases and inhibits the nucleotide excision repair that restores the DNA lesions caused by UV-B irradiance \cite{Karran2016, McAdam2016, Ravanat2001, Mullenders2018, Bhampan2003, Delineasios2018}. In addition, \cite{He2006} found that chronic irradiation of keratinocytes by UV-A irradiance can induce apoptotic resistance and as a result to enhance the probability of carcinogenesis. Despite that UV-A radiation was considered to cause predominantly non melanoma cancer, last studies revealed pathways also for melanoma carcinogenesis \cite{Premi2015}.

Recent researches showed specific responses of the human’s organism to the exposure to solar UV irradiance that also could be taken into account. For instance, some studies indicated that together with its protective role, the melanin is able to enhance the probability for triggering cancer processes through various biochemical pathways induced by UV radiation \cite{Takeuchi2004, Premi2013}. Also, the circadian rhythm together with the life style were found to impact the erythema and DNA harms \cite{Gaddameedhi2015, Sarkar2018} that assumes an adaptation of the permanent habitant’s organism to the local irradiative conditions. However, nowadays transport methods allow very fast movement over the world, even within one day. Long-distance moving across latitudes leads to an appreciable phase shifting of the sleep-wake cycle with respect to the day-night cycle. This occurrence could disturb the circadian rhythm and as a result to impact the organism’s response to the solar UV irradiation. On the other hand, the movement across the latitudes causes a sharp change in the solar UV radiation levels. In case of traveling towards more irradiated geographical regions, such stress could affect the usual intensity of bioprocesses including DNA lesion-repair equilibrium. These conditions contribute to increasing of damages and some of the alterations to the DNA may remain as permanent mutations \cite{Roy2017}. To illustrate the solar irradiance variations as a function of latitude, Fig. 4 exhibits the diurnal time patterns of solar UV-B and UV-A radiations evaluated by TUV model for clear sky conditions at three different latitudes of the northern hemisphere. It can be seen that the maximum of UV-B irradiance at local noon at 60°N is about 68% lower than the maximum at 40°N and nearly twice lower than at 20°N. The corresponding differences for UV-A irradiance are smaller presenting ~ 28% lower value at 60°N with respect to 40°N and ~ 36% with respect to 20°N. The increase of radiation maximum passing from mid-latitudes (40°) to tropics (20°) is ~ 17% for UV-B and ~ 6% for UV-A.
Similar relations take place for the UV-B and UV-A doses evaluated over a half-hour period during the local noon. This example shows an appreciable increase in UV-B and UV-A doses, when one moves from the polar regions to mid-latitudes or tropics.

To enlarge the basis of protective strategy by including additional effects like previously indicated, a proper parametrization of such effects need to be performed. A similar attempt was made by Petkov et al. (2011) who evaluated the exposure times $\Delta T_{UV-A,min}$ to solar UV-A irradiance that could lead to doses $D_{UV-A,min}$, which were found to trigger SCC in laboratory experiments with HaCaT keratinocytes (Colombo et al. 2017; Wischermann et al., 2008; He et al., 2006). For this purpose, weighting functions that replace the action spectrum in Eq. (1) were constructed by using both spectrum of the UV-A lamps applied in laboratory and the solar spectrum. Taking the doses found to cause SCC in experiments as $D_{UV-A,min}$, these weighting functions allowed the evaluation through the common approach presented by Eqs. (1) and (2) of the times $\Delta T_{UV-A,min}$ in real environmental conditions. Exposure periods $\Delta T_{UV-A,min}$ up to a month were obtained adopting diverse diurnal irradiation regimes and assuming a cumulative effect of subexposures (Wischermann et al., 2008; Lavker et al., 1995; Lai et al., 2018). It should be noted that these preliminary estimations just extrapolated the results achieved in laboratory to real environmental conditions. Their reliability that closely depends on the ability of HaCaT cells to represent the DNA behaviour in the human organism needs additional study. The example above illustrates a possible way for parameterizing the results about bioprocesses in order to be used for discussed enlargement of the protective strategy basis.

5. Conclusions.

The current protective strategy against the health hazards associated with the exposure to solar UV radiation has been constructed by using the erythema as a basic effect. Beside a brief description of the strategy, the present study discussed whether the messages about protection from the erythema had the same importance for carcinogenesis triggered by solar radiation. It was assumed that the risk of cancer formation decreases if the sunburning has been avoided. However, such assumption was made on the basis of epidemiological (statistical) studies without bearing in mind the variety of pathways for DNA damages. Similar relationship between erythema and carcinogenesis cannot provide any parametrization of the second effect as in the case of the former. A parametrization expressed by the action spectrum and minimal dose of weighted UV radiation is needed to make a reliable estimation of the exposure time for protective messages. Moreover, the different nature of the erythema and the lesions in DNA caused by UV radiation strengthens the doubt that above scheme could represent the probability of cancer development. On the other hand, the weight of UV-A radiation in erythema is quite low, while the role of UV-A for the cancer formation is well evidenced in the latest studies. In addition, the efficiency of the preventions would enhance if they take into account also the particularities of the contemporaneous life, like the possibility of fast transport from a place with low solar irradiation to a place characterized by much higher irradiation. These considerations suggest an enlargement of the protective strategy basis by seeking a method to including also the DNA damages beside erythema.

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Figure 3: Upper panel (a) presents the solar spectral UV irradiance $I(\lambda, t)$ as was measured in Bologna, Italy on 21 May 2009, 12:00 UTC together with the erythema ($A_E(\lambda)$), SETlow DNA damage ($A_S(\lambda)$), skin cancer ($A_{SC}(\lambda)$) and vitamin D formation ($A_{VD}(\lambda)$) action. The lower panel (b) exhibits the effect of irradiance weighting expressed by the irradiances $I_{eff}(\lambda, t)$.

Figure 4: Time patterns of the solar UV-B (left) and UV-A (right) radiations coming to a horizontal area of the Earth surface at 20°N, 40°N and 60°N, respectively on 30 June 2019. The corresponding doses absorbed by a horizontal surface and evaluated for a half-hour interval between 12:00 and 12:30 local time (shadowed areas) are also indicated in the graphs.