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THE SUN AND THE SKIN: EVIDENCE-BASED PHOTOPROTECTION AND SUNSCREEN GUIDELINES

This supplement is produced as part of Dermatological Nursing Volume 15 No 2

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CONTINUING PROFESSIONAL DEVELOPMENT: SUNSCREEN SUPPLEMENT LEARNING OUTCOMES

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www.bdng.org.uk Dermatological Nursing 2016, Vol 15, No 2 (suppl)
It’s fair to say that in 2016 we are a society aware of the potential risks of excessive exposure to UV. Wrong.

As Co-founder of Teddington Trust, a charity that exists to provide support and education to patients of Xeroderma Pigmentosum (XP) (who require the most extreme and stringent photo-protection), I can say without a shadow of a doubt that, despite the wealth of knowledge out there, the message still isn’t getting through.

I am fortunate to be able to spend time in nursery and primary schools across the UK, talking about this rare genetic condition. When talking to the children we discuss what it might be like to live with such a complex condition, and we use pupil interaction to demonstrate this.

One of our standard questions — “Hands up who has ever had sunburn that really hurt” — never fails to take our and the teachers’ breath away, for up shoot hands typically representing 80–90% of the school. Anecdotally, in a group of pupils 4-11 years old, the vast majority have experienced sunburn at least once. Clearly something is going wrong but what and how can it be addressed? Undeniably the responsibility lies with parents, but are they failing to grasp the importance of sun safety or is it something else?

The truth is complicated and multifaceted. Parents today enjoyed long hot summers and ran naked in the garden; few wore sunscreen and a ‘nice tan’ was a sign of a healthy child. A tan was associated with health and vitality and this legacy remains. It’s often the first comment when seeing a friend’s child — “You look well” — or when a friend prompts their child to show their “little white bits” like a badge of honour. Media photos...? Wrong.

One aspect is confusion about what SPF really means. One aspect is confusion about what SPF really means: how long can my child be in the sun and what protection does it really offer? What does the UVA star rating and term broad spectrum ‘mean’? Then there’s the lack of clarity regarding water resistance.

Having navigated the minefield of assorted products, do parents who responsibly purchase their SPF 30+, top UVA-rated, water-resistant sunscreen realise that this only really applies to laboratory conditions? That actual application represents about one quarter of the protection stated; and that’s without factoring in sweat or swimming, which may reduce the effectiveness by 50% plus.

There is widely conflicting opinion regarding Vitamin D advice. Parents are bombarded with articles about the rise of rickets in UK children due to low vitamin D levels, reportedly due to less outside time and high use of sun safety measures. Yet conversely they are scared of the risk of exposure when enjoying outside time in an attempt to boost Vitamin D.

But we must not forget it is not all down to parents. Another big stakeholder in all of this is schools, where our children spend 6 hours a day, 5 days a week, for much of the year. Children often spend their breaks and extended learning outside during the hottest part of the day. They are when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when parents are being told to keep their children during the hottest part of the day. They are when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection, whereas when whole classrooms are being ushered inside and in the shade, with maximum protection.


Parents buy high-SPF sunscreen, UV protective clothing, sunhat and shades, but still it isn’t as simple as it looks.

Better suncare information for all

Living in the UK, it’s easy to think that sun safety is a matter for hot climates only or for patients who suffer from debilitating UV-sensitive conditions. But this is a mistake we make at our peril. With skin cancer rates rising and more of our population facing the consequences, it is of paramount importance that action is taken now.

Concise information on SPF, UVA ratings, coverage, effective application and guidance on skin types does exist, but industry needs to provide better signposting to it to ensure consumers can access this wealth of information and make informed buying choices.

Education for parents, schools and at individual pupil level must be high on the agenda. Without this we will see another generation flounder and fail to take seriously health risks associated with irresponsible or misguided sun exposure.

As a mother of one child with xeroderma pigmentosum, who requires complete protection from all UV sources, and of another child who does not, I am given a unique insight into both approaches.

Sometimes I wonder if perhaps the decision-making for a parent with a child with XP or other UV-sensitive condition might indeed be simpler. For them, it is not a matter of when and how much to protect, or a reliance on sunscreen; it’s a matter of total shielding. I am safe in the knowledge that my XP son is receiving no damage from UV due to his zero exposure lifestyle, but despite all the knowledge I have, can I be so confident in the exposure legacy I am affording my other son?
TOWARDS A BROADER SUN PROTECTION

Dominique Moyal, Sophie Seité

Human skin exposure to UVR from sunlight can cause many adverse effects. Photoprotection is required for both UVB and UVA radiation. Organic and inorganic filters are used in sunscreens. Sunscreen filters must be carefully combined to achieve aesthetically pleasant products with photostability and well-balanced UVB-UVA photoprotection, the latter defined by a UVA protection factor of at least one-third of the SPF value. Methods for assessing the SPF and the UVA protection have been standardised to afford the same information of efficacy to consumers. Recommendations need to be made on the choice of the adapted level of protection according to the phototype and sun exposure conditions and on the usage of the sunscreens during exposure. Recently visible light, infrared radiation and pollution have been associated with oxidative damage and premature ageing. New products are developed to integrate these new elements.

KEYWORDS
- Sun protection
- UVA protection
- Photoprotection beyond UVR
- Sunscreen formulation

Introduction
Human exposure to ultraviolet (UV) radiation from sunlight can cause many adverse effects. UVB radiation (290-320nm) is mainly responsible for the most severe damage: acute sunburn and long-term damage including skin cancer. It has a direct effect on cell DNA and proteins1. Unlike UVB, UVA radiation (320-400nm) is not directly absorbed by biological targets1 but can dramatically impair cell and tissue functions. UVA penetrates deeper into the skin than UVB. It particularly affects connective tissue where it produces detrimental reactive oxygen species (ROS), also known as free radicals. ROS cause damage to DNA, cells, vessels and tissues leading in particular to photoageing15. A good way to remember this is UVA — Ageing and UVB — Burning.

Adverse effects of UVA and UVB on health
As with UVB, UVA has been implicated in the depression of the immune system7-8 and in the development of skin cancer, principally melanoma and squamous cell carcinoma9-10. Photosensitivity reactions as well as photodermatoses are mainly induced by UVA11 and oxidation of pre-existing melanin is a key factor in the aggravation of pigmentation disorders such as melasma12. Within the UVA band, UVA1 rays (340-400nm) have been shown to be responsible for most of the UVA damage. It is also important to note that UVA radiation is at least 17 times higher in UV levels than UVB irradiance14.

In response to the growing awareness of the additional damage caused by UVA radiation, various UVA filters are now incorporated into sunscreen formulations in most parts of the world. For all these reasons, it is evident that sunscreens must contain both UVB and UVA filters to cover the entire range of UV harmful radiation.

The evolution of photoprotection methods to measure the level of UVB and UVA protection have also evolved. By the turn of the 21st century, it was already clear that skin damage was the result of both genetics and extrinsic factors including UVB/UVA. However, there is growing evidence that extrinsic factors should be broadened to include additional wavelengths, ie infrared (IR) and visible light, but also environmental xenobiotics from air pollution13.
sunscreen products in Europe should fulfil this criteria in order to ensure a well-balanced protection. A UVA logo (‘UVA’ in a circle) is used on the packaging so consumers know this requirement has been met (Figure 1).

Figure 1

All products should bear the UVA logo

To increase UV protection requires higher concentrations of UV filters in the formulation. However, this can reduce the cosmetic acceptability of the product. Therefore the challenge for formulators is to produce an aesthetically pleasant product with lower concentrations of UV filters, while maintaining the same level of protection. This also keeps the cost of the product down.

Higher UV protection and a satisfactory degree of cosmetic acceptability can be achieved using a combination of UV filters with different maximum absorbance peaks (UVB, short UVA and long UVA). It is also important to ensure that the resultant formulation is photostable, ie does not degrade (hence reducing its effectiveness) on exposure to sunlight.

UV filters regulation

All UV filters used in Europe have to be approved by the European authorities to ensure they are safe for consumers. Each UV filter should be used at a maximum concentration depending on its safety profile. The main UVB and UVA filters used in Europe are shown in Table 1. UV filters have been developed to not penetrate through the skin because they are more efficient when they stay at the top of the skin.

Synergy of protection between organic and inorganic UV filters

One example of synergy (ie enhanced effectiveness/sun protection) between organic UV filters is terephthalyldene dicamphor sulfonic acid (Mexoryl® SX), a short UVA filter; drometrizole trisiloxane (Mexoryl® XL) and bis-ethylhexyloxyphenyl methoxyphenyl trizine (Tinosorb® S), two broad UVB/UVA filters. All three filters are photostable and part of Mexoplex® technology.

A further strategy is to use nanosized titanium dioxide (an inorganic UV filter) in combination with organic UV filters.

Photostability

Another very important aspect to ensure continued protection during exposure, without increasing the concentration of UV filters to reach the expected level of protection, is the photostability of the filtering system.

The UV filter avobenzone (butylmethoxydibenzoylmethane or BMDBM) has a high potency in the UVA1 range (340-400nm), peaking at 358nm; these types of UVA1 rays are known to increase the development of most of the UVA damage. However, on UV exposure they undergo significant degradation, leading to a decrease in their protective UVA efficacy. Some potent photostabilisers of avobenzone have been identified, such as octocrylene, a UVB filter17 or a biodegradable oil, Eldew® SL-205, a derivative of a natural amino acid sarcosine, used in Mexoplex® technology.

Table 1.

Main UVB and UVA filters used in Europe with their peak of absorption.

<table>
<thead>
<tr>
<th>Filter</th>
<th>Peak of Absorption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Octocrylene</td>
<td>UVB (303nm)</td>
</tr>
<tr>
<td>Homosalate</td>
<td>UVB (306nm)</td>
</tr>
<tr>
<td>Ethylhexyl salicylate</td>
<td>UVB (305nm)</td>
</tr>
<tr>
<td>Ethyl hexyl trizone</td>
<td>UVB (314nm)</td>
</tr>
<tr>
<td>Butyl methoxydibenzoylmethane Avobenzone</td>
<td>UVA (357nm)</td>
</tr>
<tr>
<td>Terephthalyldene dicamphor sulfonic acid (Mexoryl® SX)</td>
<td>UVA (345nm)</td>
</tr>
<tr>
<td>Drometrizole trisiloxane</td>
<td>UVB/UVA (303nm, 344nm)</td>
</tr>
<tr>
<td>Disodium Phenyl Dibenzylmazole Tetrasulfonate</td>
<td>UVB/UVA (335nm)</td>
</tr>
<tr>
<td>Dihydroxybenzyl Hexylbenzate</td>
<td>UVA (354nm)</td>
</tr>
<tr>
<td>Methylene bis-benzotriazolyl tetramethyl-butyphenol</td>
<td>UVB/UVA (305nm, 360nm)</td>
</tr>
<tr>
<td>Bis-ethylhexyloxyphenyl trizine</td>
<td>UVB/UVA (310nm, 343nm)</td>
</tr>
<tr>
<td>Titanium dioxide</td>
<td>295nm</td>
</tr>
</tbody>
</table>

Polymer and spreadability

Increasing efficacy, improving the compliance of consumers for all ages and skin types, is a key factor: It is important to develop products with good spreadability, aesthetically pleasing skin feeling and transparency on the skin. An homogeneous distribution of UV filters onto skin is essential for maximising efficacy.

Film-forming polymers are very often used to obtain more even spreading of the product and to enhance efficacy and resistance to different stresses, such as water; towelling and sweating.

Clinical studies

Research suggests that combinations of highly efficient and photo-stable filters provide an optimally balanced protection against both UVA and UVB. Studies have shown the protection against UV-induced skin damage, provided by sunscreen products with same SPF but different UVA protection factor, is markedly different, emphasising the importance of high-UVA protection in preventing damage. Only well-balanced sunscreen products characterised by a UVAPF/SPF protection factor ratio of at least one-third, with absorption over the entire UV spectrum of sun radiation, have been shown to prevent the adverse effects of UV exposure on skin biologic functions. Comparisons between products having the same SPF but different UVAPF have been made. Better protection was always found when the UVAPF was higher. The difference between the products’ efficacy was demonstrated for many types of sun...
damage including photodermatoses (polymorphous light eruption), DNA damage, photoinmunosuppression, photocaging markers and pigmentation. Moreover, a higher level of protection was obtained when the UVA protection was increased, meaning an SPF/UVAPF ratio of ≤2 instead of 3².3.

**Evaluation of the efficacy of sunscreen products against UVB and UVA radiation**

**Determination of sun protection factor (SPF)**

The efficacy of a sunscreen against UVB damage is measured by the sun protection factor (SPF), which is a globally accepted index of protection from erythema after a single exposure to solar simulated radiation (SSR). SPF is determined under conditions described by ISO standard 24441, or the US Food and Drug Administration (FDA)².³.

The SPF is the ratio of the UV dose able to induce a skin erythema with product (also called Minimal erythema dose with product (MEDp)), to the UV dose able to induce an erythema on the unprotected skin (MEDu); SPF = MEDp/MEDu.

Sunburn is mainly induced by UVB (80-85%); short UVA contributes about 15-20%. In practice the SPF means the factor of multiplication of the UV dose to get sunburn. This is also often interpreted as a factor of multiplication of time, however it is important to keep in mind that sunscreen products are designed not to increase the time of exposure but to protect the skin from damage.

SPF is determined using a standardised amount of product applied on the skin, which is 2mg/cm² in order to enable reproducible results within a laboratory and from one laboratory to another. Lower amount of product leads to lower SPF with a high variability. Studies have shown that consumers apply, in one single application, from 0.5 to 1.2 mg/cm² depending on the texture and site of the body. This is why very high SPF (50 and above) has been developed. Nevertheless, it is very important to recommend frequent re-application, in particular after swimming, sweating and towelling. In effect, lower amounts of the sunscreen applied will lead to lower SPF protection with higher variability — whatever the level of SPF.

**Determination of UVA protection level**

Because SPF only relates to UVB protection, there was a need to develop a method of assessing protection against UVA.

The EU commission issued a recommendation²⁷ to use the Persistent Pigment Darkening (PPD) method similar to the JCIA (Japanese Cosmetic Industry association, 1995) method or any in vitro method able to provide equivalent and reliable results. The PPD endpoint is sensitive to both short and long UVA (not to UVB) allowing an evaluation of efficacy against all UVA wavelengths.

In addition, the critical wavelength, which is an evaluation of the width of the absorbance of the sunscreen product, must be at least 370nm.

The in vivo (devised with people as subjects, rather than laboratory technique) UVA method has been standardised by ISO TC217 and published in 2011 under the reference ISO 24442, and is now adopted or accepted in many countries.

The UVA protection factor is determined using a method very similar to the SPF method, however the endpoint is the pigmentation (tanned skin) induced by UVA instead of the skin erythema (skin burning) induced by UVB and a UVA, UVAPF is the ratio of the Minimal pigmenting dose with product (MPDp) to the unprotected minimal pigmenting dose (MPDu). As with SPF, UVAPF is a factor to be multiplied before reaching the minimal dose able to induce damage on the skin (PPD endpoint is representative of UVA damage). MPDu is obtained in about 1 hour in the sun. Depending on the amount of product applied, the UVAPF can decrease.

**Determination of water resistance**

Water resistance in sunscreens is measured by comparing the SPF without and with water baths. The protection should remain at least 50% after two baths of 20 minutes (in standardised conditions of temperature and water agitation) for a water resistance claim, and after 4 baths of 20 minutes for a very water-resistant claim. In practical terms for a product to claim water resistance, it will remain at least SPF 50% after 40 minutes’ immersion in water; and for a very water resistant claim, at least SPF 50% after 80 minutes.

**Recommendation of a sunscreen product**

Recommendations for sun protection levels, taking into account the skin phototypes and the sun exposure conditions, have been published by different parties. One example is given in Table 2.

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**Table 2. Recommendation of level of protection depending on the skin phototypes and sun exposure conditions.**

<table>
<thead>
<tr>
<th>Phototype</th>
<th>Moderate sun exposure</th>
<th>High level of exposure</th>
<th>Extreme level of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phototype 1</td>
<td>High Protection SPF 30-50</td>
<td>Very high protection SPF 50⁺</td>
<td>Very high protection SPF 50⁺</td>
</tr>
<tr>
<td>Phototype 2</td>
<td>Medium protection SPF 15-20-25</td>
<td>Medium protection SPF 30-50</td>
<td>Very high protection SPF 50⁺</td>
</tr>
<tr>
<td>Phototype 3</td>
<td>Low protection SPF 6-10</td>
<td>Medium protection SPF 15-20-25</td>
<td>High Protection SPF 30-50</td>
</tr>
<tr>
<td>Phototype 4</td>
<td>Low protection SPF 6-10</td>
<td>Low protection SPF 6-10</td>
<td>Medium protection SPF 15-20-25</td>
</tr>
<tr>
<td>Phototype 5</td>
<td>Low protection SPF 6-10</td>
<td>Low protection SPF 6-10</td>
<td>Medium protection SPF 15-20-25</td>
</tr>
<tr>
<td>Phototype 6</td>
<td>Low protection SPF 6-10</td>
<td>Low protection SPF 6-10</td>
<td>Medium protection SPF 15-20-25</td>
</tr>
</tbody>
</table>
Fitzpatrick defined six phototypes based on the ability to develop skin erythema and tanning, as described in Table 3.

It is also noticeable that photoprotection afforded by melanin in darker skin types (phototypes IV-VI) is no more than 5 times higher than for lighter skin types and they can develop skin cancers, supporting the need for them to adopt sun protection.

Visible light and infrared radiation effects and how to prevent them
Visible light (400-700nm) accounts for approximately 50% of the total solar spectrum but has lower energy compared to UV radiation. Visible light generates ROS. Initially, as with UVA radiation, visible light causes oxidation of pre-existing melanin, resulting in pigment darkening in people with Fitzpatrick skin type IV-V but not those with lower skin types. Visible light is also suspected of being an aggravating factor in melasma.

In order to protect against visible light, pigmented products have been developed. Opaque pigments such as iron oxides are able to reflect the visible light thus reducing the generation of ROS. Differences in the prevention of pigmentation can be obtained when comparing sunscreens with or without opaque pigments. Antioxidants can also be used to act against the ROS induced by visible light and consequently can contribute to the reduction of pigmentation.

Table 3.
Skin phototypes.
The Fitzpatrick skin phototype definitions are based on the first 30-45 minutes of sun exposure after a winter season of no sun exposure, i.e.

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Always burns easily; never tans</td>
</tr>
<tr>
<td>Type II</td>
<td>Always burns easily; tans minimally</td>
</tr>
<tr>
<td>Type III</td>
<td>Burns moderately; tans gradually</td>
</tr>
<tr>
<td>Type IV</td>
<td>Burns minimally; always tans well</td>
</tr>
<tr>
<td>Type V</td>
<td>Rarely burns; burns profusely</td>
</tr>
<tr>
<td>Type VI</td>
<td>Never burns; deeply pigmented</td>
</tr>
</tbody>
</table>

In vitro as well as ex vivo studies have shown that powerful antioxidants are efficient against MMP-1 induction by IRA. Antioxidant complexes also improve sunscreen efficacy against oxidative stress induced by UVB, as demonstrated when sunscreen formulations with and without antioxidants are compared using the in vitro method (human skin fibroblasts) or the in vivo method (prevention of UV-induced hyperpigmentation).

Pollution skin effects and how to prevent them
Urban life exposes our skin to various pollutants such as exhaust fumes, ozone and cigarette smoke. Exposure to these environmental factors has been associated with accelerated skin ageing, increased pigmented spots and wrinkles. Activation of certain factors such as aryl hydrocarbon receptor (AhR) found in various skin cells including keratinocytes, melanocytes, fibroblasts and Langerhans cells, both by UV radiation and by pollutants, results in the expression of gene coding for proteins involved in oxidative stress reactions, inflammation, immunosuppression, pigmentation and premature ageing.

Interactions between UV radiation and pollutants have also been studied in different models. A synergistic increase in squalene peroxidation (a marker of oxidative stress and inflammation) by a combination of UVA and smoke pollutants compared to either agent alone has been demonstrated. Sunscreen products containing an efficient UVA filtering system and an antioxidant complex have been shown...
to prevent this peroxidation induced by UVA radiation and pollution.  

We have also recently obtained evidence that UVR and UVA I alone and ambient relevant pollution particles have additive effects on human keratinocytes. Interestingly, we tested the La Roche Posay Thermal Spring Water with well-known anti-inflammatory and antioxidant properties and showed a significant protective effect against the induction of interleukins IL I, IL6, which are markers of inflammation, and POMC (pro-opiomelanocortin), which is a precursor of the melanotropin α MSH in melanocytes, therefore used as marker of pigmentation [unpublished data].

**Conclusion**

Integrating all these new elements is changing the way we think about skin protection. In addition to the well-balanced and broad UVB and UVA filtering system to prevent UVB and UVA damage, antioxidants such as LRP Thermal Spring Water are very helpful to neutralise the pro-oxidative influence of IRA radiation and pollutants.

> Figure 2 illustrates a modern sunscreen product with a formulation affording a broader sun protection.

**References**


www. BDNG.org.uk
A SUMMARY OF NEW NICE GUIDANCE ON SUNLIGHT EXPOSURE: ENCOURAGING BEHAVIOURAL CHANGE IN PHOTOPROTECTION

Andrew Thompson, Julie Van Onselen

Healthcare professionals can play an important role in preventing skin cancer via raising awareness of the effects of sun exposure and supporting people to revise behaviours that place them at increased risk. The recent revision to NICE guidance on Sunlight Exposure provides up-to-date, evidence-based information that practitioners should seek to pass onto the public with regards to sensible sunlight exposure. The first half of the article gives a brief summary of the at-risk groups, the environmental factors that require consideration, and the myths and misconceptions that persist in relation to sunlight exposure which need to be challenged. The evidence suggests that simply providing information on risk is usually not sufficient to change behaviour in those who actively seek to tan; consequently the second half of this article describes some simple behaviour change techniques for use in dermatology practice.

Introduction
Challenging myths on sun exposure and motivating people to change unhealthy sun behaviour is an important role and responsibility for dermatology healthcare professionals and for anyone who works in a care or educational setting or who is an employer with staff responsibility. The new NICE guidance on Sunlight Exposure gives evidence-based public health information to help prevent skin cancer, but also to ensure how sensible sunlight exposure is important for vitamin D production. The first half of the article gives a brief summary of at-risk groups, environmental aspects and myths and misconceptions of sunlight exposure, as stated in the new NICE guidance. The second half of the article explores how healthcare professionals can challenge myths towards sunlight exposure and introduces some simple behaviour-change techniques for dermatology healthcare professionals.

New NICE guidance on sunlight exposure
In 2016, NICE published new guidance on sunlight exposure risks and benefits, aimed at healthcare professionals, NHS commissioners and managers, local authorities (and the wider public, private, voluntary and community sectors), as well as specifically people working in early-years educational settings, residential care, employers and the general public.

This guideline replaces recommendations 1-5 in skin cancer prevention 2011 NICE guideline PH32 and should be read together with NICE’s guideline on vitamin D: increasing supplement use among at-risk groups and any recommendations made by the Scientific Advisory Committee on Nutrition (SACN) on vitamin D.

‘At risk’ groups
The NICE guidance identifies several groups of people who are at risk of sun exposure; which may be over- or underexposure. NICE recommends that public health activities should focus on these groups, which are listed in Table 1.

Environmental considerations
There are several factors that need to be considered when advising patients and the public on exposure to the sun. In order to help with behavioural change, facts such as the effect of environmental factors on sun exposure and an understanding of the variation in intensity of sunlight is important to include in patient/public education.
Table 1.
Group of people at risk of over- or underexposure to sunlight.

<table>
<thead>
<tr>
<th>At risk of overexposure to sunlight</th>
<th>People at risk of skin cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups of people who should take extra care to avoid skin damage and skin cancer</td>
<td>Children (particularly babies) and young people</td>
</tr>
<tr>
<td></td>
<td>People who tend to burn rather than tan — people with lighter skin, fair or red hair, blue or green eyes, or who have lots of freckles</td>
</tr>
<tr>
<td></td>
<td>People with many moles</td>
</tr>
<tr>
<td></td>
<td>People who are immunosuppressed</td>
</tr>
<tr>
<td></td>
<td>People with a personal or family history of skin cancer (even if their natural skin colour is darker than that of the family member who had cancer)</td>
</tr>
<tr>
<td>Groups who spend a lot of time in the sun and so are at increased risk of skin cancer</td>
<td>Outdoor workers</td>
</tr>
<tr>
<td>Groups with high, but intermittent, exposure to sunlight and who are therefore at increased risk of skin cancer</td>
<td>People who sunbathe and take holidays in sunny countries</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>At risk of underexposure to sunlight</th>
<th>People at risk of low vitamin D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups who have little or no exposure to the sun</td>
<td>Cultural reasons — skin is not exposed</td>
</tr>
<tr>
<td></td>
<td>People who are housebound or otherwise confined indoors for long periods. For example, people who are frail or in institutions, or people who work indoors all day</td>
</tr>
</tbody>
</table>

Table 2.
The strength of sunlight at different times of day.

<table>
<thead>
<tr>
<th>Time of day</th>
<th>Strength of sunlight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between 11am and 3pm</td>
<td>Sunburn is most likely. Most people can make sufficient vitamin D by going out for short periods and leaving only areas of skin that are often exposed uncovered (such as forearms, hands or lower legs). Longer periods may be needed for those with darker skin.</td>
</tr>
<tr>
<td>Before 11am and after 3pm</td>
<td>It takes longer to synthesise sufficient vitamin D. The risk of sunburn is less</td>
</tr>
<tr>
<td>In the UK, sunlight is strongest between 11am and 3pm between March and October</td>
<td></td>
</tr>
</tbody>
</table>

Geographical location: solar UV levels increase nearer to the equator and at higher altitudes.

Time of year: from March to October, UVB rays help people produce vitamin D, but excessive exposure can also cause sunburn. Solar UV levels are highest during the summer (and most intense in late June).

Time of day: solar UV levels are highest around the middle of the day when the sun is highest in the sky.

Weather conditions: solar UV levels are reduced by cloud cover but they can still be intense enough to cause sunburn (even if it is not warm).

Reflection: sunlight reflects off surfaces such as snow, sand, concrete and water. This can increase the risk of sunburn and eye damage, even in shaded areas.

UVA penetrates glass (although more weakly than direct exposure) and over long periods of exposure will cause skin damage. However, the vitamin D-inducing UVB does not penetrate glass.

Myths and misconceptions about sun exposure that may influence behaviour:

There are several misconceptions about sun exposure that may influence people’s behaviour in how they expose themselves to the sun or are unaware of the dangers of over- or underexposure.

A key fact is that increased frequency and time spent in the sun increases the potential risks of sunlight exposure. However, skin type also affects the potential risks and benefits from sunlight exposure. Table 3 outlines some common myths and misconceptions regarding sun exposure that people may express.

Changing sun exposure behaviour is identified by NICE as a public health responsibility for primary care practitioners along with those working in specialist dermatology services. Dermatology professionals can play a particularly important role in behaviour change in relation to sun exposure as they will encounter and have expertise in identifying people who will be at an increased risk because of their skin condition, treatment, or skin type. However, psychological research has consistently shown that simply raising awareness of the potential harm that can arise from carrying out certain activities (smoking, unprotected sex, heavy drinking, tanning etc) is often insufficient to change behaviour on its own. Unhealthy behaviours tend to be resistant (although not entirely) to the provision of new information and are often habitual, automated, and reinforced by a range of individual and cultural variables.

Healthcare professionals should seek to share knowledge about the risks of sun exposure, however they also need to be
Table 3.
Common myths and misconceptions regarding sun exposure.

<table>
<thead>
<tr>
<th>Myth/misconception</th>
<th>Reality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunlight misconception</td>
<td>I would not get sunburnt on a cool or cloudy summer day. Even if it is cool or cloudy it is possible to burn in the middle of the day in summer. It is also possible to burn at other times of the day and year.</td>
</tr>
<tr>
<td>Sunlight myth</td>
<td>A tan from sunlight is healthy as it protects against sun damage. There is no safe or healthy way to get a tan from sunlight. Getting a tan provides little protection against later exposure to sunlight and the resulting skin damage outweighs any later protective effect.</td>
</tr>
<tr>
<td>Vitamin D myth</td>
<td>I should be able to absorb enough vitamin D sitting by a window with the sun streaming in. It is not possible to get enough vitamin D by sitting next to a closed sunny window.</td>
</tr>
<tr>
<td>Vitamin D misconception</td>
<td>Vitamin D can be absorbed from winter sunlight. It is not possible to get enough vitamin D from sunlight between October and March in the UK.</td>
</tr>
</tbody>
</table>

Developing behaviour change interventions is a challenging and complex process drawing on theories that explain the underlying barriers to change, while at the same time drawing on research that identifies the mechanisms required for that change to occur. Before behaviour change interventions can be fully rolled out into practice they require that the component techniques have been subject to effectiveness and economic testing. The Centre for Behaviour Change at University College London (UCL: www.ucl.ac.uk/behaviour-change) emphasises the importance of behaviour change interventions being underpinned by understanding the motivations, opportunities and capabilities that influence the target behaviour. Motivations, for example, might consist of both conscious intentions and goals, such as deciding to reduce exposure to the sun, as well as automatic motivations including habits associated with sun exposure, for example lying contentedly in the sun.

The NICE guidance raises the importance of behaviour change, however it provides little in the way of advice as to how this might be actually undertaken in dermatology practice. Consequently, our aim here is not to provide a definitive guide to developing or even using behaviour change techniques, but rather to describe some simple approaches to supporting behaviour change that show promise in reducing risky sun exposure and that might also lend themselves for use in brief primary care or dermatology consultations.

Subtly challenging myths towards being in the sun

The evidence on the effectiveness of strategies to communicate complicated messages such as those concerned with sun protection is limited. Consequently it is not clear how healthcare practitioners such as dermatology nurses might best convey messages concerning the risks of over- or underexposure to the patients that they encounter. Nevertheless, NICE makes clear recommendations for the need to raise public awareness of the environmental risk factors and myths associated with sun exposure that are described above and there is evidence that many people still lack knowledge about sun exposure. There is also evidence that advice provided face-to-face by healthcare practitioners might be more effective than information provided by mass media, particularly if it’s framed in relation to the specific patient’s risk issues and motivations. Consequently we have provided (Table 4) a list of useful web resources that can be used to provide patients with straightforward and sensible advice.

While attitudes are just one of the many determinants of behaviour change, researchers in this area have advocated the importance of understanding the individual patient’s motivations towards tanning and particularly the benefits they perceive that they achieve from engaging in this behaviour. For example, is seeking a tan associated with enhancing appearance? Or is it viewed as a way of ‘treating oneself’, or of reducing stress? While there are common motivations, there will be a range of individual differences and different groupings of people who see a tan that may require different behaviour change approaches.

As a result of this, Robinson et al in 2016 suggested that healthcare practitioners should take a patient-focused approach during history taking, asking open questions such as: how does having a tan make you feel? And, what do you get out of having a tan? They suggest that this approach, if done in a noncritical, exploratory fashion, can enable the healthcare practitioner to establish the specific motivations for individual patients in engaging in risky tanning. This in turn enables the tailoring of advice to the specific patient. Patients whose motivation is to relax or see sun exposure as stress reduction can be advised of the stress associated with illness and guided towards using other approaches to stress reduction. This might simply involve discussions of alternative ways of pampering or looking after themselves, such as increasing other activities that they personally enjoy and that are associated with lower risks.

Some examples of simple behaviour change techniques

Using a range of behaviour change techniques in combination has been shown to be useful. For example, Mahler et al reported that the combination of...
providing cosmetic sunless tanning product alongside a personal UV facial photograph showing sun damage resulted in reduced sun exposure in comparison to a participant group that were provided with a UV photograph alone, without advice on low-risk means of altering skin tone. This approach shows how the motivation of enhancing appearance is tackled by both finding a low-risk alternative to appearance enhancement which allows for conforming with social pressures, while at the same time seeking to raise awareness of the specific impact UV exposure has on ageing and damaging the skin. This combined approach may be particularly important with young adults where cultural and peer pressures may maintain appearance-driven unhealthy sun exposure and where the perception of being invincible might be more commonplace. Healthcare practitioners can provide advice about the effects of the sun on the appearance of the skin and should consider combining this with more sophisticated information containing images of the effects of photoaging. Where resources allow and risks are high, use of UV photography to show the patient the personal impact may well be useful.

There is now significant evidence in other areas of unhealthy behaviours that people can be helped to bridge the gap between intention to change and taking action by being guided to form advance ‘if-then’ plans’. Such plans are seemingly straightforward statements whereby an opportunity or obstacle to taking action is specified (if) and followed by also naming a resulting desired response (then). For example, van Osch et al. reported finding some improvement in parental application of sunscreen to children using the following simple if-then plan: ‘If …………, then I will apply sunscreen SPF 20+ on my child every 2 hours.’ The use of if-then plans in improving sun protection behaviours needs further research but could be an intervention that might easily be built into sun protection guidance leaflets.

**Conclusion**

The review of effectiveness commissioned by NICE identified several interventions that have changed sun-exposure behaviour. NICE concludes that the existing research into behaviour change interventions is limited by small sample sizes, and lacks longer-term follow-up periods, and generally has been shown to have relatively small effect size. The lack of longitudinal studies is particularly worrying as, given the time it may take to develop skin cancer and the ongoing occurrence of environmental risk, behaviour-change interventions will need to be repeated and reinforced, and we currently lack information as to the frequency at which this needs to occur.

**References**


**Table 4.**

<table>
<thead>
<tr>
<th>Web-based information sources for HCPs and the public.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Web pages</strong></td>
</tr>
<tr>
<td>Sun Safety Q&amp;A</td>
</tr>
<tr>
<td>Sun Smart – Healthcare professional area</td>
</tr>
<tr>
<td>Skin at work: outdoor workers and sun exposure</td>
</tr>
<tr>
<td>Staying safe in the sun: top tips to help</td>
</tr>
<tr>
<td>La Roche-Posay Skin Checker Campaign</td>
</tr>
</tbody>
</table>
SUNLIGHT

- Sunlight is a small part of the electromagnetic spectrum as shown below. It consists of visible and invisible light. The invisible light is ultraviolet (UV) and infrared radiation. The warmth of the sun comes from the infrared radiation.

Sunlight comprises about **5% UVB** and **95% UVA** radiation.

- UVB causes **burning** and UVA causes **ageing**. UVA is the radiation used in sunbeds.
- BOTH UVB and UVA are capable of causing skin cancer.
- UVA can penetrate through clouds, glass and clothing; you may not burn sitting next to a window but you can still experience photoageing and UVA damage.

SUN EXPOSURE: THE BENEFITS

- Many people enjoy spending time in the sun for its warming effects on the body.
- The sun can have a positive effect on psychological **wellbeing**.
- Short periods of exposure to strong sunlight (before burning) produces **vitamin D**.

SUN EXPOSURE: THE RISKS

- Overexposure to sunlight increases the risk of **non-melanoma skin cancers** (basal cell carcinoma and squamous cell carcinoma).
- There are over **102,000** cases of non-melanoma skin cancer each year.
- The most serious form of skin cancer is melanoma and the main environmental risk factor is sun exposure or the use of sunbeds.
- Overexposure to sunlight can also lead to eye problems such as **cataracts**.
- The UV index (see below) measures the danger from UV rays for the average person.

UVA penetrates deeper in the skin and damages the dermis layer leading to photo-ageing signs such as **wrinkles**.

SUN EXPOSURE: REDUCING THE RISKS

- Avoid being out in strong sunlight between **11am and 3pm**.
- Cover up with any clothing that has close-weave fabrics such as **cotton** (e.g., T-shirt material), which gives good protection.
- However, wet clothes offer less sun protection.
- Wear a wide-brimmed hat and sunglasses with a **UV 400** label.
- Use a sunscreen with a **high SPF** of 30 or 50+ and bearing the UVA logo.
- Keep babies and children under 3 years old out of direct sunlight.

**Sources**

2. https://www.nice.org.uk/guidance/NG34/chapter5-Context
**SUNSCREENS**

- The sun protection factor – SPF – of a sunscreen is a measure of how much longer you can stay in the sun before burning.
- For example, if you would normally burn after 10 minutes in the sun without a sunscreen, using an SPF 30 product means you can spend up to 300 minutes (i.e., 30 x longer) in the sun before burning.
- To achieve the SPF of your sunscreen you need to apply generously in the quantities suggested below.
- Re-apply every 2 hours.
- The SPF provides shielding against UVB radiation **not** UVA.
- Broad spectrum sunscreens offer protection against both UVB and UVA. UVA protection is shown by the UVA in a circle logo on the pack. This means the sunscreen meets European regulations.
- Water resistance = 2 swims or baths of 20 minutes
  - Very water resistant = 4 swims or baths of 20 minutes

**TIPS ON USING A SUNSCREEN**

- Always apply the sunscreen to clean, dry skin roughly **15 minutes** before going outdoors.
- Apply from head to toes and behind the ears. To achieve the required SPF you need:

<table>
<thead>
<tr>
<th>Age</th>
<th>Teaspoons</th>
<th>ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 years</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>7 years</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>10 years</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>13 years</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>15 years</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>Adults</td>
<td>6-8</td>
<td>35</td>
</tr>
</tbody>
</table>

- Use a sunscreen **all year round**, not just during the summer. Specially on the face and visible areas like your hands.

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**LA ROCHE-POSAY. COMMITTED TO DERMATOLOGY**

La Roche-Posay has focused on helping to raise awareness on skin cancer prevention. Working with dermatologists all over the world, we are now in a stronger position than ever to help promote early detection.

- **90%** of skin cancers can be treated if detected in time*.
- Learn more about what to look for when checking your moles and those of your loved ones at: [www.laroche-posay.co.uk/skinchecker](http://www.laroche-posay.co.uk/skinchecker).
- In 2016, La Roche-Posay is introducing My UV Patch. An innovation in wearable technology that can help you learn more about your own sun exposure and improve your behaviour when outside during the day. Get yours for free from: [www.laroche-posay.co.uk/myuvpatch](http://www.laroche-posay.co.uk/myuvpatch).

*Source: [www.euromelanoma.org/intl/node/25](http://www.euromelanoma.org/intl/node/25)

Epidemiological fact sheet
As well as describing vitamin D metabolism, this article appraises the proven and less-likely benefits of vitamin D. To help with this, the evolutionary theory of pigmentation is outlined together with information on how disrupted diurnal rhythms may link to metabolic syndrome and other diseases. The main functions of vitamin D are discussed, including maintenance of calcium and phosphorus levels in the blood, bone mineralisation and neuromuscular transmission, as well as guidance on intake, recommended blood levels and the need for oral supplementation.

Table 1. Harms and benefits of sun exposure.

<table>
<thead>
<tr>
<th>Harm</th>
<th>Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sunburn</td>
<td>1. Warmth</td>
</tr>
<tr>
<td>2. Photocarcinogenesis</td>
<td>2. Vitamin D production</td>
</tr>
<tr>
<td>3. Photoaging</td>
<td>3. Phototherapy</td>
</tr>
<tr>
<td>4. Photodermatoses</td>
<td>4. Immune effects</td>
</tr>
<tr>
<td>5. Aggravation of skin disease</td>
<td>5. Circadian rhythm</td>
</tr>
<tr>
<td>6. Immune effects</td>
<td>6. Mood improvement</td>
</tr>
</tbody>
</table>
of folate degradation, deficiency and foetal abnormality, and possibly that of skin cancer if the pale, interfollicular skin was amelanotic. The acquisition of dark eumelanin would then have conferred an evolutionary advantage over both handicaps.

This evolutionary change to hairless, dark-pigmented skin occurred over several million years, whereas migration out of Africa to higher latitudes has happened over the last few hundred thousand years. It has been suggested, rightly or wrongly, that the change from dark to light skin colour in higher latitudes is predominantly due to the selective advantage of paler skin being able to synthesise more vitamin D.

Diurnal rhythm
Melatonin is a highly conserved molecule that is present in cyanobacteria that appeared on earth around 3 billion years ago. Its primary function is that of an antioxidant (locally produced in the mitochondria of many human tissues including keratinocytes) but during evolution a separate, secondary one developed, that of a circadian rhythm regulator manufactured by the pineal gland. Throughout evolution life has been constrained by the 12-hour light/dark cycle leading to compartmentalisation of biochemical, physiological and/or behavioural processes to specific times of the day. Every cell has its own clock genes but these are predominantly governed by the master clock: in the supra-chiasmatic nucleus (SCN), which in turn is very sensitive to the blue light wavelengths 460-480nm hitting the melanopsin receptors in the retina. Control over other body/cell systems is then mediated through various neural and endocrine signals (figure 1).

While sunlight working through the SCN is the main entrainer (zeitgeber) of peripheral clock systems, sleep disruption, altered meal times, stress and artificial light exposure (light at night) might desynchronise peripheral clock systems to such an extent that disease may follow.

Shift workers are known to have an increased risk of metabolic disease, hypertension, immune dysfunction and cancer. For dermatologists perhaps the most important risk is the circadian rhythm disruption seen from the effects of sleep disruption in individuals with bad atopic eczema.

The frequency of cardiovascular disease, hypertension, diabetes and autoimmune disease increases with latitude, as the further you are from the equator the higher the risk. Because vitamin D levels inversely correlate with latitude there has been an assumption that these lower levels of vitamin D might be responsible for these increased risks. So far intervention trials with vitamin D supplementation have not shown the major benefits expected.

It remains to be seen whether the increased risk is due to the effect of diurnal rhythm change or whether other mechanisms might be responsible, such as Wille's hypertension and metabolic syndrome theories involving nitric oxide. Here UVA exposure has been shown to lower blood pressure by a small amount, mediated through release of the vasodilator nitric oxide from reservoirs in the skin. It is suggested that less UVA exposure at higher latitudes might result in higher blood pressures and therefore a higher risk of cardiovascular events. Also UVA-triggered nitric oxide suppresses obesity and symptoms of metabolic syndrome independently of vitamin D in mice fed a ‘high-fat diet’. As melatonin can, among many other things, influence nitric oxide production, it will be interesting to see in the future whether latitude-dependent disease is predominantly a function of a disrupted circadian rhythm, a direct effect of lack of sunlight or a combination of both, and not due to vitamin D deficiency.

Effects on the immune system
As stated above, autoimmune diseases such as multiple sclerosis, type 1 diabetes and early-onset Crohn’s disease increase in frequency with higher latitudes and it is unlikely that vitamin D has a major contribution to this effect. There is a belief that the effect is due to the lower sun irradiance at these latitudes and that high levels of UVB and UVA at the equator might increase self-tolerance to autoantigens involved in systemic disease through increasing the numbers of regulatory T-cells through a cutaneous mechanism. Phototherapy for immune diseases such as psoriasis and eczema is already known to work and is of recognised benefit in lupus nephritis, ie UVA having a systemic effect on the immune system. It is not yet known if phototherapy might benefit these other autoimmune diseases.

As far as cancer is concerned low vitamin D levels at time of diagnosis are believed to adversely affect individuals with melanoma and colon cancer. But the likelihood of latitude-dependent cancer risk being due to low vitamin D levels is less clear-cut, there being some suggestion from mouse studies with intestinal cancer that benefit is not due to vitamin D alone but that UVB exposure may have an additional beneficial effect.

So some latitude-dependent diseases may correlate with vitamin D deficiency but so far causality has not been demonstrated.

The pro-hormone vitamin D
By definition a vitamin, an essential requirement for human growth and cellular activity, cannot be
synthesised by the body. Vitamin D is not a vitamin but acts like an endocrine hormone whose pre-synthesis by the skin is further modified by the liver and then completed predominantly by the kidney for distribution and action throughout the body (Figure 2).

7-dehydrocholesterol (7DHC) is needed for production of lipid bilayers in the cell membranes of keratinocyte and their organelles. Only 10–15% is converted to pre-vitamin D3 and that is age-dependent as the amount of 7DHC in keratinocytes declines with age. The action spectrum for conversion of 7DHC is the same as for erythema and DNA damage. Feedback mechanisms and catabolism to inactive metabolites determine that cholecalciferol achieves plateau levels below the erythema threshold dose; thus excessive UVB exposure cannot lead to vitamin D toxicity.\(^{11}\)

The vast majority of hydroxylation of cholecalciferol takes place first in the liver and then the kidney. With its half-life of 15 days the inactive 25OHD3 is the standard laboratory measure for vitamin D (usually D2 & D3 with units differing between laboratories; nmol/L = 2.5nmol/L) and there is an assumption that, for the most part, it is a true reflection of the active vitamin in all cells. The active 1,25(OH)\(_2\)D3 form binds to the vitamin D receptor present in the nucleus of cells in skin, kidney, brain, heart, prostate, breast, lymphocytes, gut and fat. Many of these cells also contain the 25 and 1α-hydroxylases, and the degrading 24 hydroxylases (all are cytochromes) but T and B cells only contain 1α-hydroxylase.

The main function of vitamin D is the maintenance of calcium and phosphorus levels in the blood to support metabolic functions, bone maintenance of calcium and phosphorus levels in the blood to support metabolic functions, bone mineralisation and neuromuscular transmission.

This is achieved through the interplay between mineralisation and neuromuscular transmission. The main effect of vitamin D as outlined in Figure 3 is a true reflection of the active vitamin in all cells. The active 1,25(OH)\(_2\)D3 form binds to the vitamin D receptor present in the nucleus of cells in skin, kidney, brain, heart, prostate, breast, lymphocytes, gut and fat. Many of these cells also contain the 25 and 1α-hydroxylases, and the degrading 24 hydroxylases (all are cytochromes) but T and B cells only contain 1α-hydroxylase.

Other effects of vitamin D are shown in Figure 4, which also shows sources of origin. At a cellular level vitamin D slows down the keratinocyte cell cycle, is pro-apoptotic, pro-differentiation, anti-angiogenic and regulates the hair cycle. On the immune side it suppresses T-cell proliferation, shifts emphasis from TH1 to TH2, inhibits TH17 development, facilitates Treg development, inhibits dendritic cell maturation, and induces cathelicidin expression in monocytes.

Over 90% of the body’s vitamin D requirement comes from the photochemical conversion in the epidermis with up to 30% of the annual requirement being achieved during a summer holiday. The most important factors affecting vitamin D status are: amount of UVB exposure at a particular time of day, latitude and season; the degree of skin melanisation and the practice of sun avoidance/protection.\(^{11}\) Lesser factors contributing to vitamin D deficiency are: breast feeding especially if prolonged; obesity with sequestration in fat (D3 is fat soluble); fat malabsorption; eg coeliac disease; biliary obstruction; pancreatic disease; Crohn’s disease; cystic fibrosis; gastric bypass; vegetarianism (reliant on biologically less active ergocalciferol); liver disease; renal disease; drugs; eg cholestyramine, Colestipol, anticonvulsants, HAART; genetic factors; vitamin D binding protein–SNPs; absent vitamin D receptors or absent hydroxylases or a combination.

For obvious reasons serum vitamin D levels in the UK peak at the end of summer and are lowest at the end of winter. Individuals with type 6 skin have levels considerably lower than type 1. There is uncertainty whether this is due to melanisation or perhaps behaviour as all skin types produce a similar rise in serum D3 when exposed to a standard dose of UVB.\(^{11}\)

Our experience with xeroderma pigmentosum (XP) patients who employ extreme photoprotection measures is that, if not supplemented, they have shown extremely low levels, though none had rickets. Patients with photodermatoses or previous skin cancers have unpredictable levels so anyone employing photoprotection needs vitamin D blood levels monitored.

The optimal range for the serum level for vitamin D is unknown. Functional studies to determine deficiency have only been done against serum PTH. The vitamin D/PTH correlation curve goes into a long, flat trough making it difficult to determine a valid level for all. Increased fractures and falls in the elderly occur at a level of around 75nmol/L, and some hospital trusts take that as a target minimum. Other trusts use 50nmol/L, based on supplementation studies which suggest no population benefit above that level. The minimum level for childhood is unknown. Functional studies to determine deficiency have only been done against serum PTH. The vitamin D/PTH correlation curve goes into a long, flat trough making it difficult to determine a valid level for all. Increased fractures and falls in the elderly occur at a level of around 75nmol/L, and some hospital trusts take that as a target minimum. Other trusts use 50nmol/L, based on supplementation studies which suggest no population benefit above that level.\(^{11}\) The minimum level for childhood is unknown but might be a little lower according to the age-related graphs of vitamin D against PTH. The deficiency level, where the risk of rickets then becomes high, though still somewhat unpredictable, is arbitrarily set at below 25nmol/L with the range 25-50nmol/L being classed as insufficiency.

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**Figure 2**

Synthesis of vitamin D. 7DHC is converted to pre-vitamin D3 by UVB. At the right temperature this then isomerises into the inactive vitamin D3, cholecalciferol, which is then carried around the circulation attached to its binding protein (DBP). The cholecalciferol may be sequestered (not stored) in fat or hydroxylated first in the liver and then again in the kidney.
Standard advice for a minimal level for an individual would be to follow the guideline of your own trust or region as it would give you medico-legal protection.

If 50 or 75nmol/L are defined minimal levels, there is no evidence to suggest what the optimal range or upper level should be. Toxicity starts at levels of around 300nmol/L, which is difficult to achieve unless taking a much larger dose (supplementation >4000IU daily orally). Some studies have shown that high-dosing schemes increase the risk of fractures in the first three months. The trend for high self-dosing is not based on evidence and seems to be increasing, the consequences of which are uncertain. There is, though, some epidemiological support for there being an increased risk of mortality at levels higher than 125nmol/L. How much lower than the upper level of normal this should be is unknown.

Guidelines for intake for populations as a whole vary. The declination of the sun means almost no vitamin D is produced in the skin during the winter months in London between October and April and in Dundee between September and May. The concept of 10 minutes’ daily exposure of face and arms to midday sun during appropriate summer months is a theoretical construct and not reliable and it gives a mixed message, particularly to skin cancer patients. Confirmatory prospective trials have not yet been done.

Studies in Manchester in individuals with white skin suggested daily 10-minute summer exposure would not provide enough to go through the winter months and one suggestion has been that individuals like this would be better off receiving supplementation during the winter. For individuals with dark skin (South Asians in Manchester) it has been suggested that they should receive supplementation throughout the year. These suggestions, though, should not be used as a guideline for communities as a whole as they have not been validated.

In individuals where sun-avoidance and sun-protection behaviour has been recommended, whether it be for a photodermatosis or skin cancer diagnosis, it is preferable to just supplement with oral vitamin D rather than give them the mixed message of avoid the sun but utilise the 10-minute rule at midday to keep levels high. Recommended daily allowances vary from country to country and within country. Dietary recommendations are difficult to achieve as standard doses give a wide variety of results and probably need to be related to BMI in view of vitamin D being sequestered.
rather than stored in fat\textsuperscript{12}. UK guidelines are due to be published this year but the draft outline is recommending that all UK individuals aged 4 and over should receive 400 units (\textit{10\mu g}) supplementation daily so that 97.5\% of the population achieves a serum level $\geq 25\text{nmol}\text{L}^{-1}$\textsuperscript{12}. This careful recommendation should address the overall 1 in 5 deficiency status in the UK\textsuperscript{15} (up to almost 40\% in institutional adults) but recognises the dearth of evidence for optimal levels.

Under controlled conditions, increasing thicknesses of sunscreen application results in diminishing production of vitamin D\textsuperscript{16}. In practice this does not seem to matter as it is thought that, because most people apply sunscreen too thinly, vitamin D production may be adequate in the short term\textsuperscript{17}. Possibly there would not be enough to last through the winter.

Oral supplementation for those at risk (ie anyone employing photoprotection measures) is simple and safe. In the UK margarine is fortified with added vitamin D as well as some cereals, so most people apply sunscreen too thinly, this does not seem to matter as it is thought that, because most people apply sunscreen too thinly, vitamin D production may be adequate in the short term\textsuperscript{17}. Possibly there would not be enough to last through the winter.

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Care needs to be taken with infants and in patients with renal stones, renal failure, sarcoidosis and hypercalcemia. Children and adults with vitamin D deficiency may be prescribed vitamin D supplements orally (oral sprays are convenient but not available on the NHS drug tariff). Vitamin D supplements can also be purchased from health-food shops. For bone demineralisation calcium needs to be given as well. Care must be taken using cod liver oil as supplementation as it also contains vitamin A.

As a result of milk in the UK being fortified with vitamin D during World War II, rickets — a disease that can lead to bone deformities and cause some children to become deformed — was eradicated in the UK.\textsuperscript{21} Episodes of hypercalcaemia in the UK in the 1950s blamed on poor fortified food quality control led to a reversal of that policy, except for margarine and some cereals. The re-emergence of rickets in the UK,\textsuperscript{22} poor bone health in older adults, the burgeoning skin cancer epidemic and consequent sun avoidance/protection advice and the appreciation that many other health outcomes might be affected by the prohormone vitamin D, both at an epidemiological and a cellular level, has led to a huge resurgence of interest in vitamin D.

For dermatologists our role is to make sure that every time we give advice to our skin cancer or photodermatology patients on sun-avoidance and sun-protection measures, we couple that with appropriate advice on long-term vitamin D supplementation. We also need to be aware of the possibility of insufficient/deficiency status in any individual with type 5-6 skin (particularly South Asian), any child undergoing prolonged breast feeding and any older adult restricted to a life indoors.

References
20. Rhodes LE, Webb AR, Fraser HL, et al. Recommended summer sunlight exposure levels can produce sufficient (>$20$ ng ml$^{-1}$) but not the proposed optimal ($>32$ ng ml$^{-1}$) $25$(OH)D levels at UK latitudes. J Invest Dermatol 2010, 130(5): 1411-8
22. Veugelers PJ, Pham TM, Ekwaru JP. Optimal vitamin D supplementation doses that minimize the risk for both low and high serum 25-hydroxyvitamin D concentrations in the general population. Nutrients 2015, 7(12): 10189-208
Photosensitivity can present in a number of different ways and through a variety of mechanisms. Treatment should be tailored to the individual, and based on an understanding of the disease process and patient lifestyle. Common to all conditions is the need for adequate photoprotection directed at the wavelength of light responsible for the condition. Specialist medical and nursing management is often required for successful control of the photodermatoses. In this review we focus on both the medical and nursing management of these often disabling conditions.

Citation: Fityan A, West H. Managing the patient with photodermatoses and sun protection. Dermatological Nursing 15(2suppl): s21-s25

Introduction

The photodermatoses are a group of conditions defined by an abnormal response in the skin following exposure to light. This group encompasses a range of conditions, from those immunologically mediated to (photo)toxicity due to drugs or chemicals, inherited disorders of DNA repair to the aggravation of pre-existing conditions through exposure to light.

Presentation of these disorders varies greatly and diagnosis may be complicated by the absence of a rash at review. Consequently, the patient’s history, the timing and description of the rash are highly relevant.

In some cases further investigation will be required. Routinely available blood tests for antinuclear antibodies should be performed in all patients to exclude lupus erythematosus. Specific investigations for photosensitivity (phototesting) are available in a few centres in the UK.

- Monochromators: artificial light is dispersed with the use of a prism, allowing its constituent wavelengths to be applied to the skin. The reaction provides information on the type of light an individual is sensitive to and helps to guide diagnosis and treatment.
- Solar simulator or broadband Ultraviolet A (UVA) or Ultraviolet B (UVB) source: These are used to provoke the reaction, thereby allowing the character of the rash to be visualised and biopsied if necessary.

In this review we discuss a general approach to managing photosensitive individuals from both a medical and nursing perspective, with a focus on more specific treatment options for some commoner photodermatoses.

General principles of photoprotection

It is essential that each individual has a good understanding of both the role that light plays in their condition and of the different photoprotective methods available. Knowledge of the specific wavelength of light a patient is sensitive to will help to guide the choice of photoprotection. When unknown, advice should be based on the expected sensitive wavelength for their condition.

The impact that photoprotective measures may have on an individual cannot be underestimated. Many of the more severe photodermatoses require fundamental alterations to lifestyle and routine, which for some patients is too onerous and ultimately unsustainable. Support, tailored advice and, when necessary, reinforcement may be required.

Vitamin D production in the skin occurs in response to UV exposure and is essential for bone health (among other health benefits). Consequently individuals requiring rigorous photoprotection are at risk of vitamin D deficiency. Vitamin D levels should therefore be checked in photosensitive patients and supplementation commenced if they are found to be deficient.

Advice on photoprotection for healthy and at-risk individuals is available through recent published NICE guidelines. While this is not aimed at photosensitive individuals, it does provide some useful general advice to patients and practitioners.

Avoiding exposure when ultraviolet light is at its peak

Ultraviolet levels are at their highest between 11 am and 3 pm in the UK. It is therefore advisable to seek shade, or limit outdoor activities during this period. This is particularly important during the spring and summer months. Smart-phone applications providing daily updates on UV levels may help an individual to tailor their daily activities.
Clothing

Clothing can provide a relatively constant and reliable method of photoprotection. The level of photoprotection afforded by clothing can be measured using the Ultraviolet Protection Factor (UPF). A simple method of assessing this is to hold a single layer up to the light in order to visualise how much penetrates the fabric. This can be useful when demonstrating the different levels of protection provided by different materials. Effective protection can be attained through the use of thick fabrics, a tight weave or multiple layers. Factors such as the wetting or stretching of fabric may increase the transmission of light. Specifically designed high-UPF clothing is available to purchase.

Window glass

Glass blocks the transmission of UVB light while allowing the transmission of UVA and visible light. Clear window film can be purchased and applied to windows at home or on the side windows of cars to prevent UVA transmission. Use may be limited by cost.

Sunscreen

Sunscreens are an important adjunct to the above lifestyle modifications. Depending on the formulation, they can provide protection against UVB, UVA or a combination of the two. A small number of formulations also provide protection against low wavelengths of visible light.

The protection afforded by a sunscreen is highly user-dependent and will vary according to the thickness and evenness of spread. The Sun Protection Factor (SPF) is the measure of UVB protection that a sunscreen provides and represents the factor by which a sunscreen lengthens the time taken to develop erythema following exposure to UVB, e.g. if a person were expected to burn within 20 minutes in the sun, the appropriate application of an SPF6 sunscreen would extend the time taken to burn by a factor of six (6 x 20 minutes = 120mins or 2 hours). The SPF of a sunscreen is based on the application of 2mg/cm². Studies have consistently shown that both healthy and photosensitive individuals apply much lower quantities (roughly 25-50% of that suggested)13. Subsequently, the protection provided by sunscreen is often significantly lower than that listed on the product.

Finding a product that an individual feels is cosmetically acceptable, non-irritant and does not contain compounds that he/she is known to be allergic to (particularly in the case of chronic actinic dermatitis), is therefore essential. Only once a satisfactory sunscreen has been identified is one likely to use it at the necessary frequency and thickness.

Polymorphic light eruption (PLE)

This is the most common of all photodermatoses in the UK, affecting roughly 15% of the population. The condition presents with numerous exposed-site pruritic erythematous papules, vesicles or plaques (Figure 1). These occur a few hours to days following exposure to UV and clear within 2-7 days without scarring. Most patients will only flare on holidays abroad, while the more sensitive individuals will also flare in the UK.

Enquiring about features linked to hardening may provide clues to the diagnosis. The phenomenon of hardening is the process by which repeated UV exposure increases the amount of UV exposure required to induce a rash. Because of this, exposed sites such as the face and dorsal hands, are less commonly affected, and patients may gradually improve as the summer progresses.

Management

PLE is predominantly provoked by UVA light, but UVB may be relevant in some. The management of PLE can be divided into preventative therapies and therapies for the management of flares.

Prevention

Striking a balance between regular low-level exposure/hardening without provoking PLE and prevention of overexposure/flaring with photoprotection can be effective in mild cases.

Phototherapy

Photoensensitisation utilises the hardening phenomenon to prevent PLE in patients that flare in the UK as well as abroad. Given that, PLE is predominantly a UVA-induced condition, narrowband UVB (TL0) is preferable in most. A low, increasing dose of TL0 is used 2-3 times per week for between 4-6 weeks and given in early spring. Treatment to commonly exposed sites only and/or topical corticosteroids prescribed regularly on the days of phototherapy may reduce the incidence of flares. If severe, flaring may require several days of systemic corticosteroids to settle12. For those individuals sensitive to UVB, oral or bath PUVA can be employed in a similar manner.

Patients should be encouraged to maintain regular gentle exposure to sunlight following a course, in order to prevent loss of benefit, which would otherwise occur over 4-6 weeks.

Hydroxychloroquine

The antimalarial hydroxychloroquine at a dose of 200mg twice a day during the spring and summer has been shown to provide moderate protection against PLE. The mechanism of action may occur in part through the disruption of antigen presentation to T cells.

Omega-3 fish oils

At high doses, these have been demonstrated to be effective at increasing the threshold of PLE provocation by UVA14.

Management of flares

For hot holiday flares, a short five-day course of the oral corticosteroid, prednisolone, at the onset of the eruption is often sufficient to settle a PLE flare. Doses of roughly 0.3mg/kg/day are often used. Frequent use is avoided due to numerous undesirable side-effects, which include reduced bone density, hypertension and altered glucose metabolism. Repeated flares should be managed with alternative therapies.

Solar urticaria

Solar urticaria (SU) is a rare photodermatosis that presents with pruritus, well-defined erythema, whealing and flaring following exposure to light (Figure 2). The condition occurs due to the development of a specific IgE targeted at a light-altered molecule known as a chromophore, present in the skin or blood of a sensitive individual. IgE-mediated mast cell degranulation results in the release of histamine among other mediators9. Provocation occurs within minutes of exposure to light and typically resolves within a few hours. Sensitivity to UVB, UVA, visible light and rarely infrared is recognised. Most commonly, sensitivity is to UVA and visible wavelengths15.

Figure 1

Polymorphic light eruption. Erythematous papules and plaques following exposure to sunlight.

Figure 2

Solar urticaria. Immediate wheals on monochromator testing to UVB and UVA within minutes of exposure.

Management

Targeted photoprotection

Particular attention needs to be paid to visible light sensitivity. Reflectant sunscreens containing zinc...
Chronic actinic dermatitis
Chronic actinic dermatitis (CAD) is a persistent eczematous eruption of exposed sites brought about by sensitivity to UV (predominantly UVB) and sometimes visible light (Figure 3). It commonly, but not exclusively, affects older males with a history of chronic sun exposure, but can develop in younger individuals with atopic eczema. Patients present with chronic, lichenified confluent eczema of exposed sites, with a clear cut-off between affected and covered sites.

Roughly three-quarters of patients will also have evidence of allergic contact dermatitis. Therefore, patch testing and photopatch testing (exposure of potential allergens to UVA during testing) should be performed in all.

Systemic therapies
Systemic therapies used for other forms of eczema are often used with success in CAD. The effectiveness of very few has been proven in clinical trials. However, a small study assessing the role of azathioprine in the management of CAD did demonstrate a marked and sustained improvement in 64% of patients. Other treatments commonly used, but without the support of controlled trials, include methotrexate, mycophenolate mofetil, and ciclosporin, with short courses of oral prednisolone for flares.

Photoaggravated disorders
This title encompasses a range of conditions that occur in the absence of light, but that in affected individuals are made worse following exposure to it. Atopic eczema, seborrhoeic dermatitis, herpes simplex and rosacea are common examples. Management is targeted at the underlying condition, with the addition of good photoprotection. Clearing the underlying condition, where possible, will allow the individual to enjoy the sun without flaring.

Actinic prurigo and hydroa vacciniforme
The photodermatoses discussed above are those most commonly seen within specialist photodermatology units. Unfortunately, it is beyond the scope of this article to cover the management of all photodermatoses. However, some rarer conditions encountered are summarised in Table 1.

Managing the photodermatoses and photo-aggravated disorders: a nursing perspective
The phototherapy nurse plays a valuable and diverse role in helping with the management of patients with photodermatoses so it is important that dermatologists and phototherapy nurses establish close working relationships.

As photodermatoses are long-term conditions that may require outpatient treatments on a repeated yearly basis, nurses are able to develop professional relationships with patients and their families. Depending on the treatment protocol patients may need treatment 2 or 3 times a week (sometimes daily) for 4-6 weeks or longer. The needs of patients will differ depending on their age and whether they are newly diagnosed or have been living with the condition for years.

Atopic eczema, seborrhoeic dermatitis, herpes simplex and rosacea are examples of conditions that may be photo-aggravated. Management is targeted...
Photoprotection measures required may demand major lifestyle adjustments and patients may require help in managing restrictions on their work, hobbies, travel and holidays, etc. Nurses need to recognise that some patients may struggle more than others, especially those who spend a lot of time outdoors. Patients may experience body image problems and be embarrassed by their red, bumpy, excoriated, thickened, pigmented, dry and scaly skin. These problems can potentially lead to relationship difficulties, anxiety and depression. Patients can need physical, social and psychological support.

**Psychological support**

The photoprotection measures required may demand major lifestyle adjustments and patients may require help in managing restrictions on their work, hobbies, travel and holidays, etc. Nurses need to recognise that some patients may struggle more than others, especially those who spend a lot of time outdoors. Patients may experience body image problems and be embarrassed by their red, bumpy, excoriated, thickened, pigmented, dry and scaly skin. These problems can potentially lead to relationship difficulties, anxiety and depression. Patients can need physical, social and psychological support.

**Phototherapy for photodermatoses**

Some phototherapy nurses have become skilled in performing specialist tests such as phototesting, photoprovocation, patch testing and photopatch testing. These are often required before photodesensitisation or phototherapy, which may be used when first-line photoprotection measures are not sufficient. Skills in ensuring fully informed consent are essential if nurses are involved with consenting patients. Protocols for both preventative and suppressive desensitisation treatments can be obtained from the National Managed Clinical Network for Phototherapy in Scotland (www.photonet.scot.nhs.uk) and the St John’s Phototherapy Guidelines (www.phototherapysupport.net).

For those conditions effectively managed by desensitisation, early spring is often the best time to treat. However, it may be sensible to discuss timings with patients, given that for some patients this may not be ideal if they have a special event or sunny holiday planned for later in the year.

If patients have extensive disease it may not be possible to perform the required MED/MPD testing for desensitisation and in these patients skin typing starting dosages may be used, which should be cautiously low. In these cases, it is advisable to first treat small areas such as the arm only to assess response before treating the rest of the body.

Desensitisation can commonly provoke the rash of the underlying photodermatosis, therefore deciding whether to treat the whole body or sun-exposed sites only may need to be considered. If this happens, patients should be reassured that treatment may not have to be discontinued. Nurses should undertake very careful skin assessments and be vigilant for signs of disease flare and treatment side-effects: Responses to treatment vary from person to person, however, as does the treatment required, and nurses need to be skilled in managing individual patient needs. In the event of a flare, desensitisation may need to be withheld until the flare settles and ultraviolet light doses adjusted to suit individuals. It is wise to adopt a cautious approach and standard protocols may be too high. If very low doses are required then it is important to warm the ultraviolet light tubes for a few minutes just before the patient’s treatment. In order to prevent treatment flares, potent topical steroids can be applied to affected areas immediately after ultraviolet light or oral steroids can be prescribed on treatment days. Following photodesensitisation patients should be educated about the need to continue with gentle, regular sun exposure if possible.

Recognising the signs and symptoms of photodermatoses is a skill that all phototherapy nurses should be trained in, to maintain the benefit of treatment.

**General dermatology nursing support**

Nurses who have knowledge, skills and experience in managing eczema patients will be extremely valuable in helping to manage symptoms such as severe itching, burning, stinging, pricking, urtica, redness, papules, swelling, blisters, scaling, peeling and dryness that can occur in photodermatoses. The education, demonstration, support and advice that nurses can provide for treatments including emollients, topical steroids, topical immunomodulators and occlusive bandaging is essential, as is the support they can offer patients who require systemic treatments.

From diagnostic tests to photoprotection, topical and systemic treatments and photodesensitisation, the nurse plays a key role in helping patients with photodermatoses. Individual treatments, education and support will help to minimise the detrimental effects photodermatoses can have on the quality of life of patients and their families.

**Conclusion**

The photodermatoses are a complex group of disorders, requiring specialist investigation and management. Nursing staff working in all fields of dermatology need to have an understanding of these conditions and their management, particularly given that nurses working in phototherapy may be the first to recognise that a patient has a photosensitive condition. Good nursing care and nurse-directed

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**Table 1.**

Characteristics and management of actinic prurigo and hydror vacciniforme.

<table>
<thead>
<tr>
<th>Occurring in association with</th>
<th>Actinic prurigo</th>
<th>Hydroa vacciniforme</th>
</tr>
</thead>
<tbody>
<tr>
<td>HLA type - DRB1*0407</td>
<td>Persistent high circulating Epstein Barr Virus (EBV) viral load</td>
<td></td>
</tr>
<tr>
<td>Typical onset</td>
<td>Childhood</td>
<td>Childhood</td>
</tr>
<tr>
<td>Clinical</td>
<td>Exposed site, persistent, pruritic, erythematous and excoriated papules</td>
<td>Exposed-site inflammatory papules/vesicles, becoming crusted</td>
</tr>
<tr>
<td></td>
<td>Scarring</td>
<td>Pox-like (vacciniform) scars</td>
</tr>
<tr>
<td>Non-exposed sites</td>
<td>Occasionally</td>
<td>Rarely</td>
</tr>
<tr>
<td>Management (in addition to photoprotection)</td>
<td>Topical/systemic corticosteroids</td>
<td>Variable response to anti-viral therapy in the literature, with no evidence of benefit in a UK cohort (unpublished)</td>
</tr>
<tr>
<td></td>
<td>TLo1 to desensitise if clear/treat if active</td>
<td>Phototherapy/hydroxychloroquine have been used with limited benefit</td>
</tr>
<tr>
<td></td>
<td>Thalidomide</td>
<td>Thalidomide</td>
</tr>
</tbody>
</table>

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patient education is essential to the successful management of these patients.

References


XP in brief

XP is a rare genetic DNA repair disorder with around 100 people being diagnosed in the UK. The normal DNA nucleotide excision repair pathway is defective in those with XP meaning when the skin is exposed to UVR it is unable to repair the damage caused. This can present clinically with the incidences of skin cancers in this population being greatly increased—a reported 2,000 fold increase in melanomas and 10,000 fold increase in non-melanomas. Since there is no cure for XP, absolute and meticulous UVR protection is currently the only way of slowing down the rate of these potentially life-limiting skin cancers. There are eight different XP complementation groups with clinical symptoms that can vary greatly between them so any advice needs to be tailored to that individual, but in general the big difference between advice about UVR protection for those with XP and that of the general population is that protection is needed during daylight from UVR at all times, in all weathers, whatever their skin type.

Summary of XP symptoms

- Lentigines (freckles), often occurring around the age of 2 years old, on sun-exposed sites
- History of skin cancers, often early onset, SCC, BCC, melanomas
- History of severe sunburn with rapid onset and prolonged healing time of 65%
- Neurological symptoms in 24%...
- Ophthalamic involvement in 40%

Patients can have some or all of the above.

Nursing role

One aim of the national, highly specialised XP service funded by NHS England and based at Guy’s and St Thomas’ NHS Foundation Trust (www.gtttnhsxu.uk) is to raise awareness of this condition with the hope that patients can be diagnosed early so measures could be taken to help the patient protect from UVR before lasting damage occurs.
The two XP clinical nurse specialists work alongside the multidisciplinary team and are instrumental in teaching and advising the individual patient with XP about what methods they could take and adapt to their lifestyle to enable them to protect from UVR to the best of their ability. These measures include those listed below. It is also important to note that those patients that heed advice and fully protect in this way will be deficient in Vitamin D and so will need supplementation.

**Extreme photoprotection measures for patients with XP**

- **Sun behaviour and lifestyle**
  - Sun protection factor (SPF) 50+ sunscreen with a high UVA rating to be applied every day all year round
  - If going outside then apply high factor sunscreen 50+ with a high UVA rating 2-3 hourly to all sun-exposed sites
  - Lip balm with a high SPF
  - Limit unnecessary outdoor activities
  - Choose indoor hobbies/jobs

- **When outside (Figure 1)**
  - Visor with UV protective film
  - Long-sleeved/legged clothing, dense-weave fabric
  - Gloves/socks to cover extremities
  - Hat, not forgetting back of neck and ears
  - Wraparound sunglasses/thick glasses to protect eyes
  - Long hairstyles can help with protection

- **When inside**
  - UVA can penetrate glass, so windows to be checked for UVR and clear protective UV window film can be applied if necessary. This is for the house as well as the car
  - Lightbulbs can emit UVR; these can be tested with a UV meter and covered or changed.

**Children diagnosed with XP**

We often hear from parents that, on the one hand they finally have a diagnosis, but on the other how terrifying it is to hear exactly what is the group that has the most skin cancers dependent on the precise molecular defect. Total UVR protection, as described above, can be difficult for a child to understand. There is limited information available for this patient group on such extreme photoprotection. It is not just the child and family that need to understand the risk of UVR with a diagnosis of XP; schools that have pupils with this condition also need to produce patient age and cognitive information, visit www.xpsupportgroup.org.uk and www.teddingtontrust.com

**Adults diagnosed with XP**

As adults, they are free to decide what advice to follow and how far they wish to take their photoprotection depending on their lifestyle, family life and occupations. Informing employers can be a daunting prospect and the fear of losing your job has been an inhibitor for some patients. This can also make extreme photoprotection in the workplace almost impossible.

The social isolation of sun avoidance and the restricting impact it can have on family life can inevitably predispose these patients to depression and poor quality of mental health. Sunburn reactions are also a defining factor for extreme photoprotection. Those with types of XP that cause severe and prolonged sunburn reactions have excellent photoprotection out of necessity to avoid the painful consequences. Subsequently they have marked less skin cancers. Those who have normal sunburn reactions tend to be diagnosed later in life when the damage has been done and habits, health beliefs and opinions have formed. For these patients adapting to a life with extreme photoprotection is very difficult and consequently they are the group that has the most skin cancers and the least optimum photoprotection habits.

**General tips**

- A selection of high-SPF and UVA-rated sunscreens are available on prescription to those with a photodermatosis.
- UVR-protective window film can often be funded by a disability facilities grant obtained from the patient’s local council.
- Patients at present have to make their own UVR protective face visor. Work is currently in process to address this issue.

**Conclusion**

Extreme photoprotection could significantly prolong life expectancy for patients diagnosed with XP; so there is an important role in promoting extreme UVR protection for this patient group. It is not without its challenges but can be helped by working alongside the patient and treating them as an individual. It is recognised at present how difficult it can be for some to achieve absolute protection and still live a productive, active life. More help is also needed to produce patient age and cognitive appropriate information for this patient group. Additionally, work is still ongoing regarding visors. For further information, visit www.xpsupportgroup.org.uk and www.teddingtontrust.com
Continuing Professional Development

Sunscreen supplement learning outcomes

On completion of reading and reflecting on this supplement, you should:

- Develop a new body of knowledge on the science behind sunscreen technology and recent advances
- Be more aware of the importance of changing sun exposure behaviour; and techniques to achieve this, including campaigns and innovative wearable technology
- Have updated knowledge on the benefits and risks in sun exposure; including how to achieve vitamin D status while protecting adequately
- Understand adequate sun protection is essential to preventing UV damage and part of the management of photo-aggravated skin conditions and photodermatoses
- Put into practice that sun protection is for all year round, not just the summer; the ideal sunscreen should be effective for UVB and UVA protection, water resistant and cosmetically effective

Revalidation requirements for CPD

Nurses as part of revalidation must undertake 35 hours of continuing professional development (CPD) relevant to the scope of their practice, over the three years prior to revalidation date. CPD activities may include both individual and participatory learning (mandatory training such as health and safety is not included) — the 35 hours of CPD should include at least 20 hours of participatory learning.

The NMC does not prescribe any particular type of CPD; it is for the nurse to decide which activities will be the most useful for professional development. Individual learning involves reading and reviewing evidence-based practice or completing online learning modules, with no interaction with other healthcare professionals (HCPs). Participatory learning is a learning activity undertaken with one or more professionals where you interact with other people.

The learning activity may be a conference, training course, a virtual environment (online discussion/professional Twitter discussion feed) or a structured face-to-face discussion group. The professionals you engage with through participatory learning do not have to be HCPs.

Using the sunscreen supplement for CPD

The sunscreen supplement is a CPD resource for both individual and participatory learning. For example, individual learning may involve the nurse reading and reflecting on the articles, and taking learning points to improve practice in caring for people with photodermatoses and photo-aggravated conditions and in public health education in sun protection. The sunscreen supplement can also be used for participatory learning, for example a nurse may get together with colleagues and work through discussion points, which can be used to improve practice in the work setting. Creating a public information display on sun protection in the dermatology department is one example of a peer-review activity. In this case the total hours for the activity should be recorded stating how many were participatory.

Recording and reflecting on the sunscreen supplement for CPD

The NMC states that an accurate record must be maintained of CPD activities undertaken and include:

- the CPD method
- a description of the topic and how it related to your practice
- the dates on which the activity was undertaken
- the number of hours (including the number of participatory hours)
- the identification of the part of the Code most relevant to the activity
- evidence that you undertook the CPD activity

Evidence to retain learning from the sunscreen supplement, for proof of learning, reflective discussion and for revalidation confirmation is suggested by the NMC examples in Table 1.

Reflective accounts

As well as recording evidence of CPD, a written reflective account form should be completed. You must explain what you learnt from the CPD activity, feedback, event or experience, how you changed or improved your practice as a result, and how this is relevant to the Code. A template for the written reflective account form can be found on the NMC website; and with CPD evidence this should be stored in a revalidation portfolio (hard copy or e-portfolio).

Table 1. CPD activity and evidence to retain, adapted from NMC.

<table>
<thead>
<tr>
<th>CPD Activity</th>
<th>Suggested evidence to retain</th>
<th>Individual participatory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading and reviewing the supplement</td>
<td>Copy of sunscreen supplement</td>
<td>Individual</td>
</tr>
<tr>
<td>Enquiry-based research — for articles presenting research studies and new evidence</td>
<td>Copy of sunscreen supplement and review notes, including practice-based outcomes</td>
<td>Individual</td>
</tr>
<tr>
<td>Peer-review activities</td>
<td>Evidence of peer review, including notes and outcomes on putting learning from supplement into practice</td>
<td>Participatory</td>
</tr>
</tbody>
</table>

Reference


This CPD guide for the sunscreen supplement was compiled by Julie Van Onselen, Editor, Dermatological Nursing; and fully adheres to the NMC Revalidation guide. For more support on using this supplement for CPD, please contact dneditor@bdng.org.uk
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