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The effects of ultraviolet radiation (UVR) on the human eye and the importance of ocular protection – Part 1

About the author

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Chris Steele graduated from City University in 1988 and was a double prize winner in ocular disease and contact lens examinations. He qualified in July 1989 after his pre-registration year at the Royal East Sussex Hospital, Hastings. He is Consultant Optometrist, Head of Optometry at Sunderland Eye Infirmary (SEI). Over the past 28 years he has continued to develop a wide range of extended roles within his optometry team involving medical retina, cataract, glaucoma, anterior segment, emergency department and paediatric caseloads.

He has authored over 90 publications re: glaucoma, ocular therapeutics, medical retina, specialist medical contact lenses, refractive surgery and clinical risk management and has undertaken numerous presentations both nationally and internationally on these topics. He has authored two books, the first in the Eye Essentials series, Diabetes and the Eye, published by Elsevier in 2007 and Systemic and Ophthalmic Management of Diabetes Mellitus published by JayPee Medical Publishers in 2018.

Chris was a College examiner for pre-registration final exit examinations and postgraduate higher qualifications (diabetes and glaucoma) for many years. Chris was a member of the original NICE Glaucoma Guideline Development Group that produced the NICE glaucoma guidelines (CG85) published in 2009.

In the past 5 years he was a member of the College of Optometrists Medical Retina Development Group that produced the new Medical Retina Higher Qualifications for optometrists. Most recently he was involved in establishing the new Advanced Practice in Ophthalmology MSc Degree Apprenticeship at UCL and Moorfields, London. He continues in his role as a co-editor for Specsavers' CPD.

Outline

The potentially damaging effects of ultraviolet radiation (UVR) on the human eye and the surrounding tissue are wide-ranging. From pterygium and pingueculum, to both intra- and extra- ocular tumours the impact of these conditions can be serious and debilitating. This article, the first of two looks at the different sources of UVR and the conditions that can result from exposure. In part 2 we'll focus on the importance of ocular protection against UKR and look at some of the products and other solutions available to help minimise damage.

Learning objectives

Domain: Clinical practice

Registrants will understand the different potential sources of ultraviolet radiation (UVR), which may cause damage to the human eye (s.5).

Registrants will have up to date knowledge of the potentially damaging effects of UVR on the human eye (s.5).

Registrants will be able to identify a range of different conditions which may occur as a result of the patient's exposure to UVR (s.5).

Introduction

Ultraviolet (UV) light is electromagnetic radiation that is not visible to the human eye. It is high-energy, short wavelength radiation <380–400nm, which falls between X-rays and visible violet light in the electromagnetic spectrum (**Figure 1**). The sun is by far the strongest source of ultraviolet radiation (UVR). We are all exposed to UVR from the sun and an increasing number of people are exposed to artificial sources used in industry (see below), commerce and recreation. Human exposure to UV radiation is increasing due to ozone depletion and global climate change which influences surface radiation levels. An increase in life expectancy and changing lifestyles is leading to more time spent on leisure activities in ultraviolet-intense environments. Data regarding levels of solar UV received by children and teenagers are relatively few, but suggest that around 40–50% of total UV to age 60 year occurs before age 20 years.[1]

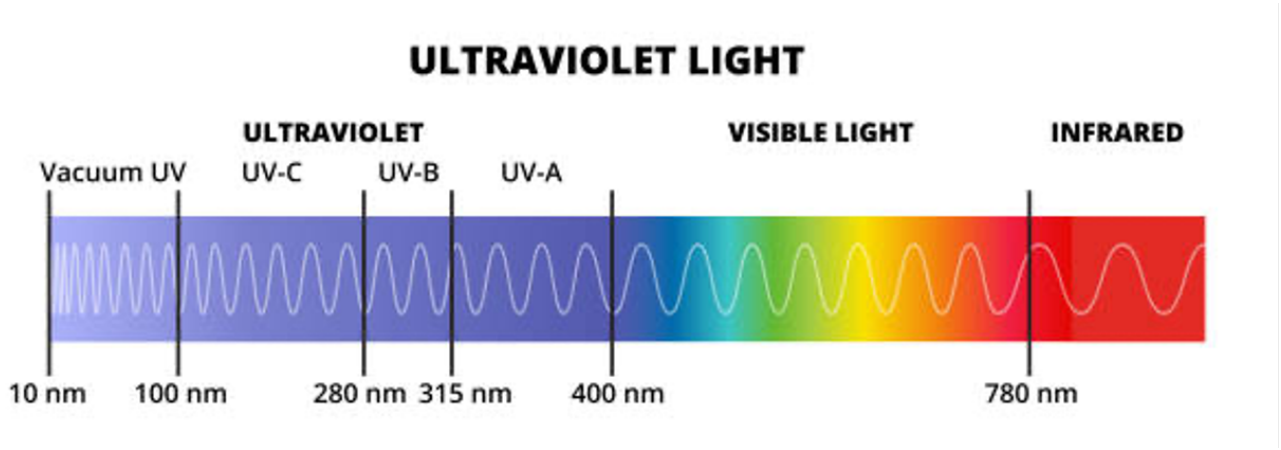

Figure 1: Ultraviolet light

UVR absorption within the eye depends on the tissues considered, the person's age and the wavelength received (UVB 280–315 nm or UVA 315–400 nm).[2] It has been estimated that before the age of 8–10 years, 2–5% of UVR received by the eyes can reach the retina, while over the age of 25 years, this will only be 1–2%.[3]

Some UV radiation is essential to the human body as it stimulates the production of vitamin D. Vitamin D has an important function in increasing calcium and phosphorus absorption from food and plays a crucial role in skeletal development, immune function and blood cell formation.[4] UVR has been used to successfully treat a number of diseases, including rickets, psoriasis, eczema and jaundice.

The interaction of solar radiation with biological tissues is related to two main mechanisms:

- Photochemical – involving ultraviolet wavelengths (see below)
- Thermal – mainly involving infrared radiation

The negative impact of UVR on public health is significant causing sunburn, skin aging and cancer, immunosuppression, and eye damage. Minimisation of exposure to solar UVR is required in order to reduce the risks of these conditions to the public. Optometrists can play an important role in relevant healthcare dissemination with regards to managing the risks of UVR exposure and being fully conversant with the various methods of protection.

The National Institute for Health and Care Excellence (NICE) Guideline 34 (2016) covers how to communicate the risks and benefits of natural sunlight exposure (specifically, the ultraviolet rays UVA and UVB) to help people understand why they may need to modify their behaviour to reduce their risk of skin cancer and vitamin D deficiency (see below).[5]

Aims

Part 1 of this review will discuss the significance of UVR exposure and examine the effects of this on the human eye in particular. Infrared radiation will also be briefly considered for completeness.

Part 2 of this review will discuss the various methods that can be used to protect eyes and skin from UVR over exposure, as well as blue light, in order to disseminate accurate and up to date healthcare information to their patients.

The electromagnetic spectrum

Within the electromagnetic spectrum, the visible light range is from ~380nm to ~780 nm. The international Commission on Illumination (abbreviated CIE for its French name, Commission internationale de l'éclairage) defines 'visible radiation as 'any optical radiation capable of causing a visual sensation directly'.[6]

The infrared light range is from ~700 to 1,200 nm. UVR contains more energy than visible or infrared light and consequently has more potential for biological damage. Visibility of light outside the well-accepted range of about 380–780nm depends upon the brightness (radiance) of the source, but is limited in childhood to approximately 310nm at the short wavelength of the visible spectrum to ~1100nm in the near-infrared (**Figure 2**).

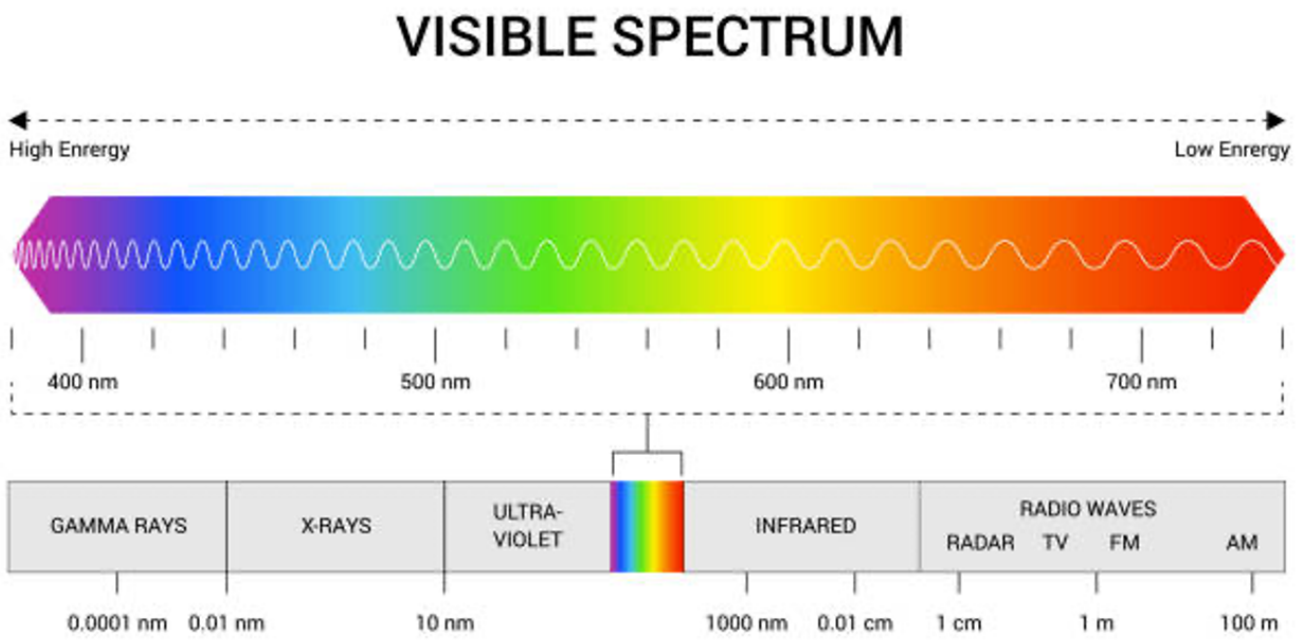

Figure 2: Visible spectrum

The International Commission on Non-Ionizing Radiation Protection (ICNIRP)

The International Commission on Non-Ionizing Radiation Protection (ICNIRP) defines several sub-groups of ultraviolet or invisible radiation classified into:[7,8]

- UVA (315-400 nm) – Accounting for ~95% of the UVR that reaches the Earth's surface. It can penetrate deep into the skin and eyes
- UVB (280-315 nm) - This is very biologically active but cannot penetrate beyond the superficial skin layers. UVB can also penetrate the eyes, but mostly it is absorbed by the cornea and lens before reaching the retina
- UVC (100-280 nm) - Short-wavelength UVC is the most damaging type of UV radiation. UVC is used in industry for various purposes and for disinfection for example. Nearly all germicidal ultraviolet lamps on the market produce UVR at 200 to 280nm, which is most dangerous to the eye

The ICNIRP like, numerous other health organisations, have now defined the standard for UVR protection as up to 400nm.[9]

UV lamps

UVC light technology is a radiation method that makes use of a specific wavelength of ultraviolet light to neutralise micro-organisms. UVC light is germicidal, which means it de-activates the DNA of microorganisms like bacteria, viruses, and other pathogens, disrupting their ability to multiply and cause disease (**Figure 3**). Lamps used in industry are made from high-quality quartz that allows for the most effective transmission of UV light. The tubes are filled with noble gases (such as argon and xenon) and additional chemicals.

When an electrical current is passed through the lamp this excites the elements inside and creates artificial UV light. By using specific construction materials or additional coatings, UV lamps can be adjusted to block or increase the emission of certain wavelengths required for different purposes. The spectral output of a mercury-based (germicidal) UV lamp can also be shifted by adding other metals such as iron, gallium, lead, tin, bismuth, or indium. This is referred to as 'doping'.

Use of UVR emitting tanning devices has been classified as "carcinogenic to humans" (group 1) by the International Agency for Research on Cancer and should therefore be used with extreme caution. One study reported that UV emissions from 100% of low-pressure appliances and from nearly 80% of high-pressure appliances exceeded the irradiance limit of 0.3 Wm set by the European technical standard EN 60335-2-27: "Household and similar electrical appliances-Safety,[10] thus posing a potential ocular hazard without appropriate eye protection.

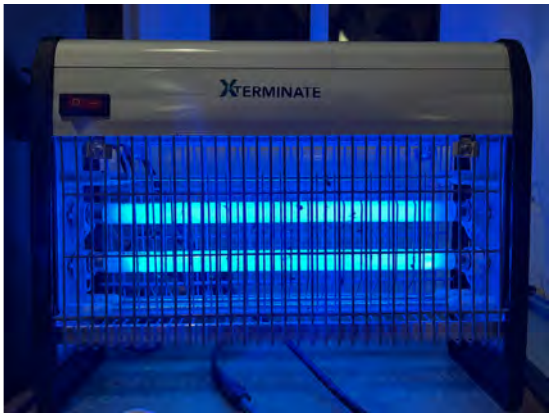

Figure 3: UV germicidal lamp

Sunlight

Figure 4: Sunlight through the atmosphere

As sunlight passes through the atmosphere, all UV-C (unless the ozone layer overhead has been damaged) and approximately 90 % of UVB radiation are absorbed by ozone, water vapour (e.g. cloud cover), oxygen, aerosols and carbon dioxide (**Figure 4**). Therefore, UVR reaching the Earth's surface is largely composed of UVA with a small level of UVB. Factors affecting just how much UVR there is at any given time include:[11]

- **Height of the sun in the sky**
  Mid-latitudes for example are highest during the summer months during the 4-hour period around solar noon. During these times the sun's rays take the most direct path to earth.
- **Latitude**
  UV levels are highest closer to the equator.
- **Altitude**
  With increasing altitude less atmosphere is available to absorb UV radiation. With every 1000 m in altitude, UV levels increase by approximately 10 per cent.
- **Clouds and haze**
  UVR levels are highest under cloudless skies, and cloud cover generally reduces a person's exposure. However, light or thin clouds have little effect and may even enhance UV levels because of scattering.[12]
- **Ozone**
  Ozone absorbs some of the UVR that would otherwise reach the Earth's surface. Ozone levels vary over the year and even across the day.

- **Ground reflection**
  UVR is reflected or scattered to varying extents by different surfaces. For example:
  - Snow ~ 80%
  - Grass and soil ~10%
  - Dry beach sand ~15%
  - Sea foam about ~25%

UVR exposure and the eye

The eye is recessed within the anatomy of the head and shielded well by the brow ridge, the eyebrows and the eyelashes. However, these anatomical adaptations are of limited use in UVR protection under extreme conditions such as sunbed use or strong ground reflection from snow, water and sand. Constriction of the pupil, closure of the eyelids and the squinting reflex minimise the penetration of the sun's rays into the eye. These mechanisms are activated by bright visible light and not by UVR – but on a cloudy day UVR exposure may still be high. Therefore, the effectiveness of these natural defences in protecting against UV damage is limited.

When an eye is exposed to UVR, the proportion absorbed by different ocular structures depends on the wavelength. The shorter wavelengths (<300nm) are the most biologically active, and are almost completely absorbed by the cornea. The longer the wavelength, the higher the proportion that may be transmitted by the cornea to reach the lens, vitreous and retina.

The cornea's absorption properties remain relatively constant throughout life. However, the lens varies depending on age. For example, an infant's lens may transmit light at 300 nm (peak transmittance is at 380 nm), compared with an adult where transmission commences at 400 nm (peak transmittance at 575 nm). The retina and uvea absorb light between 400nm and 1,400 nm including infrared radiation.

Infrared radiation (IR) is also divided into 3 groups:[13]

- IR-A (700-1400 nm) – well transmitted by water in the ocular media and thus able to reach the retina. IR-A is deeply penetrating into biological tissues and thus used in diagnostics and in skin treatments
- IR-B (1400-3000 nm) - these wavelengths do not reach the retina but penetrate as much as a few mm into the skin and ocular tissues
- IR-C (3000-10 000 nm) - These far-infrared wavelengths are absorbed very superficially (<1 mm)[14]

Photokeratitis - (Ultraviolet [UV] burn, Arc eye, Snow Blindness)

Photokeratitis is a condition that occurs following unprotected exposure to acute high-dose or supra-threshold UVR.[15, 16, 17]

Photokeratitis results from exposure to UVB (290 to 320nm) or UVC (100 to 290nm), without appropriate eye protection, which is absorbed by the corneal epithelium. This gives rise to transitory punctate erosions causing varying degrees of discomfort, depending on exposure, from mild irritation to severe pain. The corneal epithelium absorbs most UVR wavelengths because of its high protein and nucleic acid content. Then, oxidative photodegradation and production of reactive oxygen species are responsible for most damage in the course of photokeratitis.[18, 19, 20]

Sources of UV include sunlight (including reflection from snow or water) and various artificial sources such as tanning lamps, therapeutic high intensity UV (for skin conditions or seasonal affective disorder), germicidal UV lamps and other industrial sources of UVB or UVC used in e.g. arc welding, causing welder's flash.[21] Recurring incidences of snow blindness or photokeratitis in skiers emphasise that UVR protective measures must take ground reflection into account.

During the COVID-19 pandemic caused by SARS-CoV-2, a greatly increased need for disinfection arose due to fear of infection. To meet the need for disinfection, many people purchased and used ultraviolet lamps because of their effectiveness and simplicity.[22] However, improper and unprotected use of (germicidal) ultraviolet lamps has reportedly led to significantly increased photokeratitis.[23, 24, 25]

With photokeratitis, usually there is a delay in onset of symptoms of ~6-12 hours from exposure although this can be as short as 1 hour following high doses of UVR. These may persist for 24 to 48 hours, although mild photophobia and blurring may persist for 7-10 days.[26]

Most cases may result in no more than mild to moderate ocular irritation and a foreign body sensation. In severe cases the following symptoms may be present:

- Pain
- Conjunctival hyperaemia (**Figure 5**)
- Photophobia
- Blepharospasm
- Lacrimation
- Blurring of vision

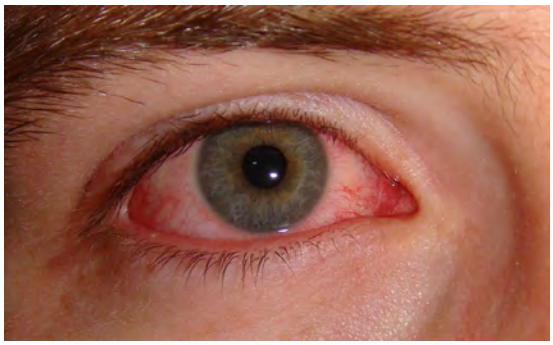

Figure 5: Photokeratitis and associated eye redness (conjunctival hyperaemia)

Signs of photokeratitis include:[27]

- Bilateral (if unilateral, suspect corneal or sub-tarsal foreign body)
- Lid chemosis and redness
- Conjunctival hyperaemia (**Figure 5**)
- Epiphora
- Punctate staining of corneal epithelium with fluorescein (may be coalescent)
- Mild transitory visual loss
- Associated skin burns from UV exposure

Local anaesthetic (benoxinate or proxymetacaine) should only be used if required during the clinical slit lamp biomicroscope examination and never for pain relief.

Patients should be advised on how best to avoid future UVR exposure and managing symptoms with cold compresses and ocular lubricants (preferably unpreserved), with ointment for overnight use, if necessary. In some cases, a next day review may be advisable to ensure any corneal epithelial lesions have healed. If symptoms persist, patients should be advised to return for further help and advice as necessary. Oral analgesics may be used for pain relief e.g. ibuprofen and paracetamol. Also consider prescribing gutt. cyclopentiolate 1% tds to aid pain relief and ciliary spasm, where clinically indicated. The use of antibiotics e.g. gutt. Chloramphenicol 0.5% drops may be considered necessary if there is deemed to be a high risk of infection in cases with e.g. significant corneal epithelial defects.

Climatic droplet keratopathy (CDK)

CDK is an acquired and potentially handicapping cornea degenerative disease that is highly prevalent in certain rural communities around the world. It predominantly affects males > 40 year of age. It has many other names such as e.g. Bietti's band-shaped nodular dystrophy or Labrador keratopathy.

CDK is characterised by corneal haziness and opalescence of varying severity leading to globular deposits. If severe enough, these may cause disruption of Bowman's membrane and elevation and thinning of the corneal epithelium. The exact aetiology and pathogenesis of CDK is still uncertain, but multi-factorial.[28] Excessive exposure to UVR from solar irradiation is considered the main causal factor.[29]

Pterygium and pingueculum

Pterygium is a fleshy bulbar conjunctival fibrovascular growth that crosses the limbus and extends onto the peripheral cornea, sometimes leading to significant visual complications (**Figure 6**).

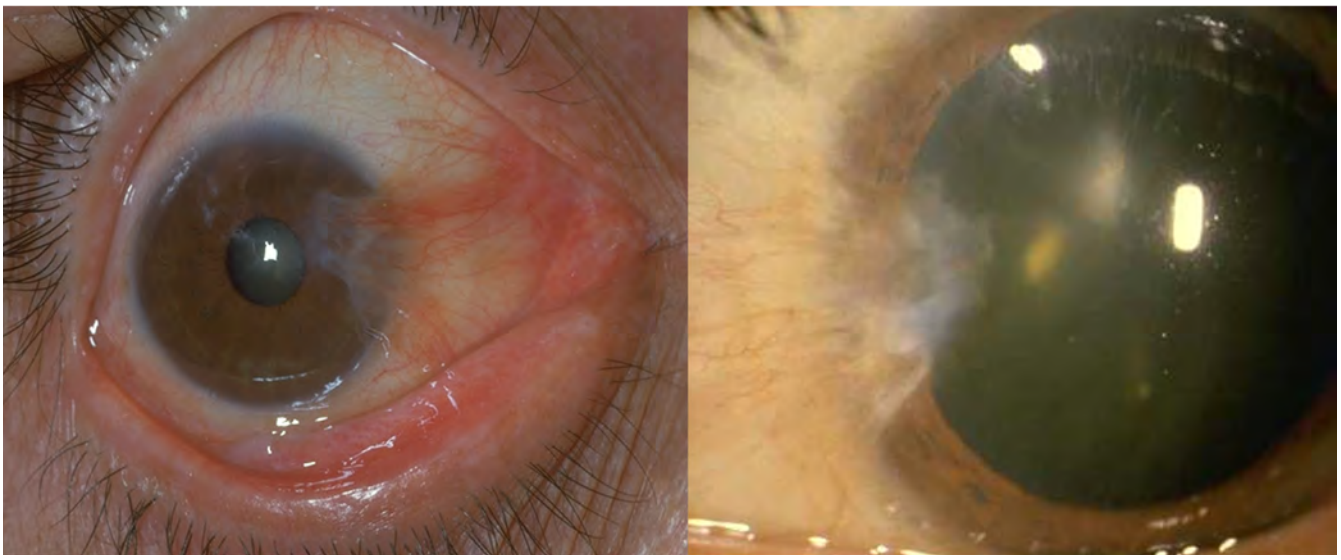

Figure 6: Pterygium

A pterygium is usually bilateral and predominantly found on the nasal bulbar conjunctiva. Pterygia may impair vision, limit eye movements and can cause eye irritation, foreign body sensation, and dryness. In some susceptible patients, the pterygium can grow over the entire corneal surface.[30]

Pterygia are twice as common in men as in women with a prevalence reported between 1.2% and ~40%.[31, 32] In one recent large meta-analysis mostly from Asia, prevalence of pterygium was 10.2%. Pterygium is uncommon in people less than 20 years of age and those who wear spectacles, as these help to protect the eye from UVR exposure, dust and wind. A greater prevalence of pterygium is associated with increasing geographical latitude, age and increasing levels of outdoor activity.[33] The pathogenesis of pterygium is mainly related to exposure to UVR.[30]

UVR-induced changes in corneal epithelial stem cells are thought to cause the corneal invasion by the pterygium, leading to destruction of Bowman membrane and elastosis.[34]

UVR exposure also induces activation of epidermal growth factor receptors resulting in downstream signalling via the mitogen-activated protein kinase pathways leading to expression of pro-inflammatory cytokines and matrix metalloproteinases (MMPs) from fibroblasts within pterygium cells.[35] This in turn leads to pterygium progression.[36]

Pterygium is an active, invasive, inflammatory process following UVR exposure, a key feature of which is focal limbal failure. The pathogenesis consists of two stages:[37]

1. Initial and progressive disruption of the limbal corneal-conjunctival epithelial barrier with scarring, thickening and distortion of the bulbar conjunctiva. Small grey corneal opacities may be seen near the limbus.
2. Progressive active “conjunctivalisation” of the cornea by tissue characterised by extensive cellular proliferation, inflammation, connective tissue remodelling, and angiogenesis as outline above. Bowman’s membrane and superficial stroma lamellae are destroyed in the process. Epithelial iron deposits (Stocker’s line) ahead of an advancing pterygium may be observed on slit lamp biomicroscope examination and relatively rich surface vascularisation. Flattening of cornea in horizontal meridian is common leading to possible induced (often irregular) astigmatism.

Initially a pterygium is usually asymptomatic, however, dry eye manifestations may be present, such as burning, itching, and tearing. As the lesion grows toward the optical zone, visual acuity becomes compromised, and surgical treatment may be indicated. Because of recurrences and repeated surgeries, the growth of the lesion may become more aggressive and cause irregular astigmatism.

Surgery is an effective treatment for pterygia causing significant symptoms and is aimed at not only removing the lesion, but also preventing recurrences, which are quite common. With simple excision techniques, involving excising the pterygium and leaving bare sclera, the risk of recurrence has been reported to be ~80%.

Other surgical options with reduced recurrence include:[29]

- Modified bare sclera techniques with subsequent transposition of the conjunctival flap.
- Conjunctival auto-transplantation where conjunctival tissue from another part of the person's eye along with limbal tissue is resected in one piece and used to cover the area from which the pterygium was excised.
- Amniotic membrane transplantation where a piece of donor amniotic membrane is fixed to the remaining limbus and bare sclera area after the pterygium has been excised. It is fixed either with sutures or tissue adhesive.
- Peripheral lamellar keratoplasty (in cases of significant ingrowth).

In some cases, the use of anti-metabolites such as mitomycin C, or 5-fluoruracil are indicated which help reduce post operative scarring.

Ocular lubricants (preferably preservative free) for symptomatic relief (drops for use during the day and ointment at night) can be very useful for some patients. For acutely inflamed pterygia, a short course of a 'non-penetrating' topical steroid (e.g. fluorometholone, loteprednol) or a topical non-steroidal anti-imflammatory drug (NSAID) used off-license may be prescribed by IP qualified optmetrists.

General advice regarding UVR protection is important. Referral to an ophthalmologist is appropriate in the following scenarios:[38]

- Threatens visual axis
- Causing significant (often irregular) astigmatism
- Associated chronic inflammation
- Cosmetically unacceptable

Pingueculae

A pingueculum is a small fibro-fatty degenerative bilateral lesion usually situated horizontally and limbally in the bulbar conjunctiva within the palpebral aperture (**Figure 7**). Its development is also related to UVR exposure. However, the association between pingueculae and UVR appears to be weaker than for pterygia.[39] Other features include:

- Degeneration of conjunctival stromal collagen fibres
- Hyalinisation of sub-epithelial collagen
- Elastotic degeneration
- Thinning of overlying epithelium
- Occasional calcification.

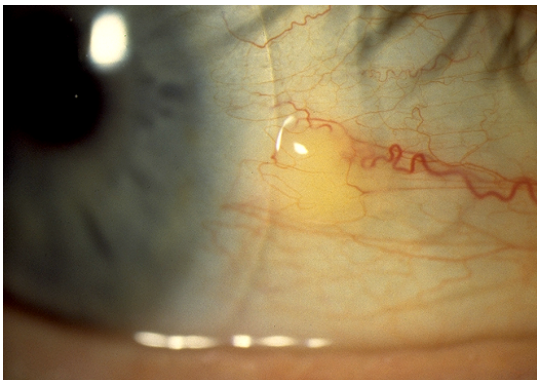

Figure 7: Pingueculum in a soft contact lens wearer

On slit lamp biomicroscopic examination pingueculae appear to be elevated and less transparent than normal conjunctiva, often whitish or yellow in colour, with a fat-like appearance. Calcification is sometimes present. They may present with slight surrounding conjunctival hyperaemia leading to pingueculitis, causing mild ocular irritation. This in turn can lead to a dellen in the adjacent cornea. Associated dry eyes are common, so always check for a decreased tear break up time (TBUT) and tear prism height.

Pingueculae are easily distinguished from pterygia as they do not cross the limbus and do not involve the cornea. Also, pingueculae do not progress to become pterygia as they are two distinct entities.

As with pterygia ocular lubricants for symptomatic relief can be useful. Where pingueculitis is confirmed, consider prescribing a short course of a 'non-penetrating' topical steroid (e.g. fluorometholone or loteprednol) or a topical non-steroidal anti-inflammatory drug ((off-licence use)[40] (if Independent Prescriber (IP) qualified).

Use of anterior segment optical coherence tomography (OCT) has recently been reported as a method of quantifying differences in pingueculitis measurements before and after treatment. In one recent study, anterior segment OCT demonstrated significant reduction in the thickness and cross-sectional area of the pinguecula following steroidal treatment outlined above.[41]

Surgical excision is very rarely necessary for pingueculae although recent case reports have described effective cosmetic removal of pingueculae by argon laser photocoagulation and by surgical excision with free conjunctival auto-grafting.[42, 43]

Skin and eyelids

Children are at very low risk of UV-related skin damage compared with adults, but the most common UV-related skin diseases occurring in adults may be observed in the first two decades of life as well, namely photo-aging (seen as premature mild skin wrinkling in teenagers) and specific pigmentary signs of UV exposure such as freckling and development of melanocytic naevi (moles) (**Figure 8**).

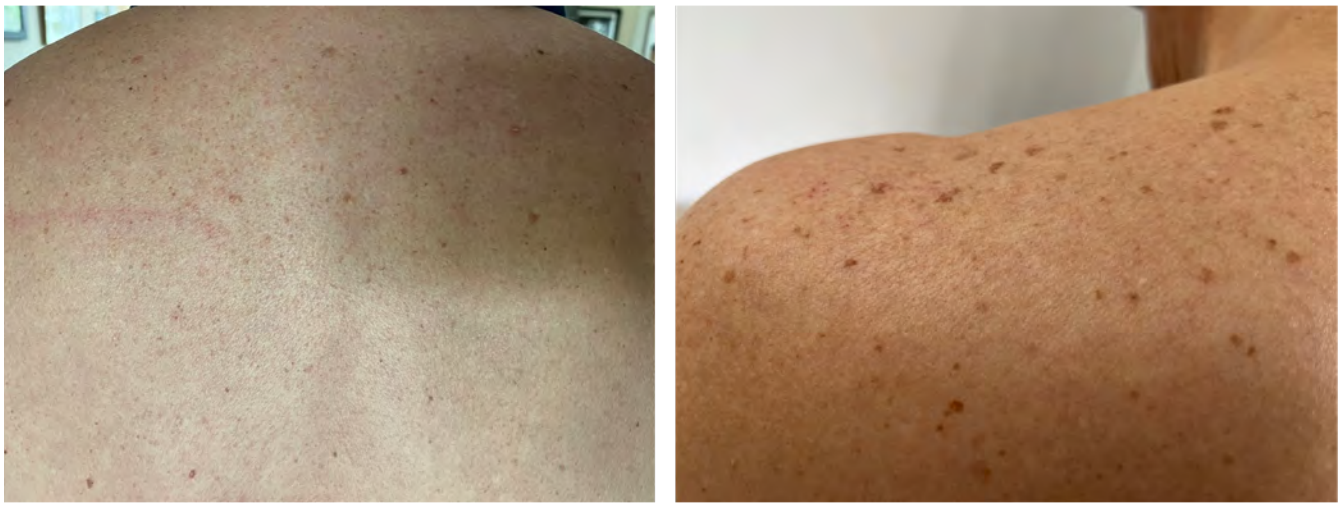

Figure 8: Sun damage causing freckles and moles (naevi)

Severe UVR exposure is related to an increased incidence of benign keratinocytic skin tumours, actinic keratoses and malignant tumours including basal cell carcinoma (BCC), squamous cell carcinoma (SCC) of the skin and cutaneous malignant melanoma (CMM). The incidence of these malignancies, especially CMM, has continued to increase in Caucasians in the last 50 years.[44] Among skin cancers, only melanoma occurs to any measurable incidence in childhood.

Human skin

The three main layers are the (**Figure 9**):

- Epidermis,
- Dermis
- Hypodermis (also called sub-cutaneous layer)

These have different specific components of epithelial, mesothelial, and neural origin. Longer UV wavelengths penetrate deeper. The outer most layer of the epidermis (stratum corneum) provides an optical barrier to UVC, whereas up to 25–50% of UVA can reach melanocytes in the dermis. In general, visible wavelengths and IR, particularly IR-A, penetrate deeper into the skin, even reaching the hypodermis.

In epidermal cells, solar UVR is absorbed from various chromophores of the cytosol and of cell membranes, including DNA and RNA. The resultant DNA damage may induce skin cell divisions, contributing to the thickening of the epidermis.

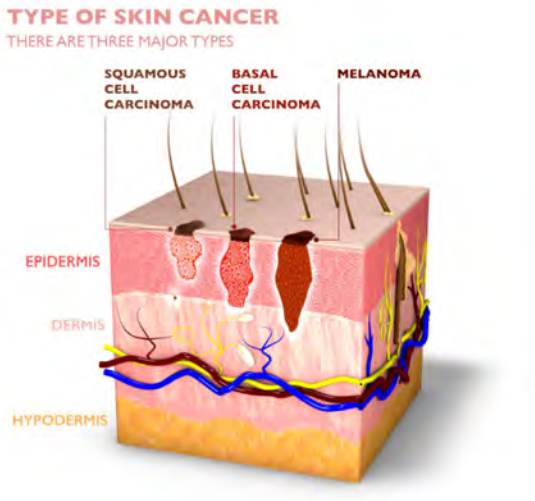

Figure 9: Types of skin cancer

DNA damage may result both from direct absorption of UVR and from oxidation due to the action of reactive oxygen species (ROS). The association between sun exposure and melanoma is complex, and appears to differ according to the site of the tumour. A recent study supports the dual pathway hypothesis, where melanoma on sites that are less frequently exposed to the sun occurs in people with many naevi (moles), whereas melanomas on the head and neck are associated with cumulative sun exposure.[45] The superficial layers of the eye are exposed to UVR and incur damage through the same pathways of DNA damage and production of ROS as is seen in skin elsewhere.

UV A is responsible for the immediate tanning effect. It also contributes to skin ageing and wrinkling (**Figure 10**). Recent studies strongly suggest that it also enhances the development of skin cancers.

UV B is responsible for delayed tanning and burning. In addition to these short-term effects, it enhances skin ageing (**Figure 10**) and significantly promotes the development of skin cancer.

Exposure to UVR on the skin results in a clearly demonstrable mutagenic effect. The p53 suppressor gene, which is frequently mutated in skin cancers, is believed to be an early target of the UVR-induced skin cancer.[46]. Harmful, toxic effects of UVR on the skin include:

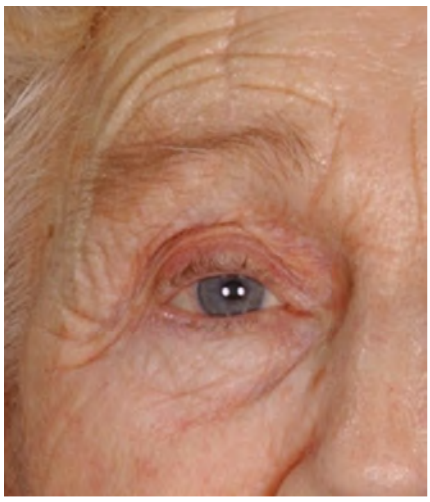

Figure 10: Skin aging and wrinkle formation

- Direct cellular damage
- Alterations in immunologic function
- DNA damage leading to gene mutations
- Immunosuppression
- Oxidative stress
- Inflammatory responses

All these factors play a significant role in photo-aging of the skin and development of skin cancer.[47]

Managing suspected skin cancers

The following advice is according to NICE (2021) and can be summarised as follows:[48]

- Refer people using a suspected cancer pathway referral (for an appointment within 2 weeks) for melanoma if they have a suspicious pigmented skin lesion with a weighted 7-point checklist score of 3 or more.

Weighted 7-point checklist:

- Major features of the lesions (scoring 2 points each):
  - Change in size
  - Irregular shape
  - Irregular colour
- Minor features of the lesions (scoring 1 point each):
  - Largest diameter 7 mm or more
  - Inflammation
  - Oozing
  - Change in sensation

Intraocular melanomas

Intraocular melanoma is the most common type of cancer that develops within the eye, but it is rare compared to cutaneous melanoma (CMM). Intraocular melanomas predominantly occur on the uvea and conjunctiva, but uveal are considerably more common than conjunctival melanomas (**Figure 11**). Exposure to sunlight, light pigmentation of the eye and skin, and living at high latitudes are often reported as risk factors for both types of intraocular melanoma.

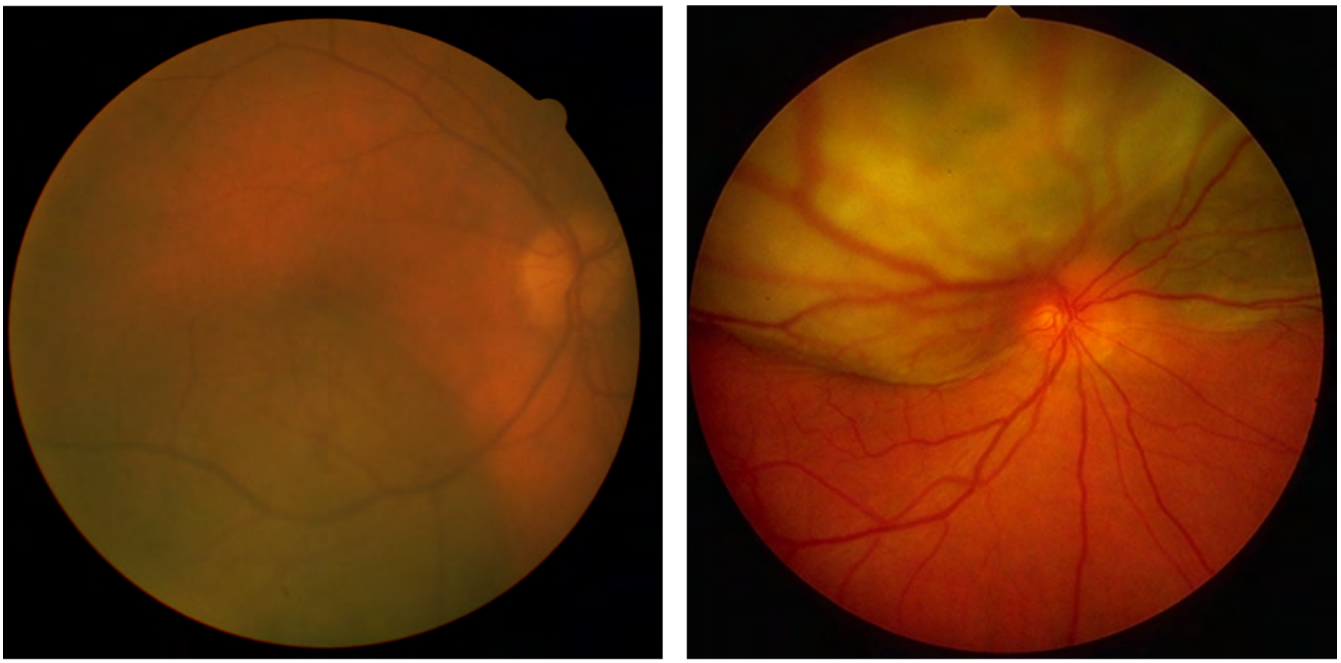

Figure 11: Choroidal melanomas

Recent studies have investigated epidemiological and genetic evidence regarding the role of UVR in the aetiology of intraocular melanoma, with more convincing evidence for conjunctival vs. uveal melanoma.[49] Recent genetic studies provide further evidence of similarity of intraocular to CMM and, thus, a possible causal role of exposure to UVR. One recent study comparing the genetic changes in uveal melanomas with those in cutaneous melanomas revealed many shared mutations, including UV signature mutations, suggesting that some uveal melanomas may be UV dependent.[50]

Basal cell carcinomas (BCC)

BCCs are the most common skin cancers worldwide in fair-skinned adult populations over 50 years of age (**Figure 12**). BCCs are a heterogeneous group of tumours, with histopathological and clinical characteristics ranging from superficial lesions to very extensive and destructive tumours (**Figure 13**). The incidence is increasing throughout the world. UVR exposure is the major carcinogenic factor.[51] Solar UVR exposure, particularly UVB, is the most significant environmental risk factor for the occurrence and progress of BCC.[52] Activation of the Hedgehog signalling pathway characterises the majority of cases. In general, BCCs are slow-growing and rarely metastasise.[53] Nevertheless, they are locally invasive and can be destructive. They are frequently multiple and recurrent on sun-exposed skin.

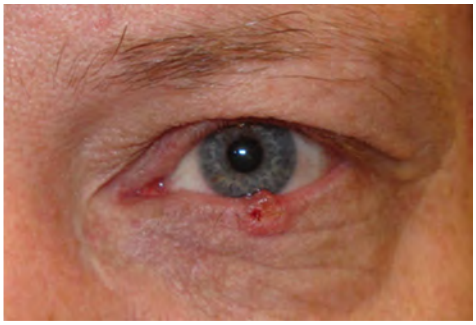

Figure 12: Basal cell carcinoma (BCC)

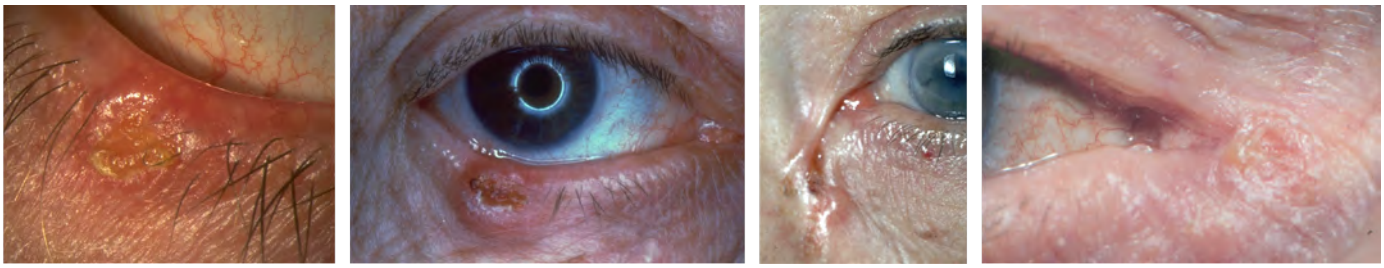

Figure 13: Basal Cell Carcinomas

BCC accounts for approximately 90% of all eyelid tumours. They are generally found on the lower eyelid (50–65%), followed by medial canthus (25–30%), upper eyelid (15%) and lateral canthus (5%).[54]

Early identification can be made clinically, aided by dermoscopy, in addition to newer imaging technologies such as reflectance confocal microscopy. While typical cases are diagnosed based on clinical findings, the clinico-pathological manifestations are varied. Consequently, skin biopsy is essential to confirm the diagnosis and evaluate the risk of recurrence.

BCC most commonly demonstrates an indolent course responsive to local destruction or surgical removal. Mohs micrographic surgery is the most effective treatment, especially for high-risk tumours. Low-risk tumours may be amendable to non-surgical treatment including topical and destructive therapies such as electro-desiccation and curettage, topical application of imiquimod or fluorouracil or photodynamic therapy. Radiation therapy can be used in patients not amendable to surgery. Advanced and metastatic BCC can be treated with Hedgehog pathway inhibitors and other systemic agents with varying responses.[55]

A relatively recent study from one oculo-plastics tertiary referral centre reported malignant transformation of lesions presenting in the periocular skin under patients' eye spectacle nose pads. Periocular malignancies of the inferior medial canthal area, where the nose pad of eye spectacle places pressure, can be easily missed or mis-diagnosed. clinicians should therefore maintain a high degree of vigilance in this regard during routine eye examinations. Furthermore, traumatic chronic pressure in the infero-medial canthal region from long-term eye spectacle nose pad use, may induce poor lymphatic regeneration leading to an immune system deficiency that predisposes this skin to a malignant transformation (**Figure 14**).[56]

There is strong evidence linking eyelid malignancies to UVB exposure and with the incidence of BCC and SCC increasing latitude where UVR exposure is greatest at the equator. The evidence of UVB as a carcinogen is strong for SCC where cumulative sun exposure is the major causative factor in the development of SCC. However, the relationship between UVR and BCC is more complicated. It is now thought that BCC formation may depend more on the severity of UVR exposure at a young age rather than cumulative exposure over a period of time.

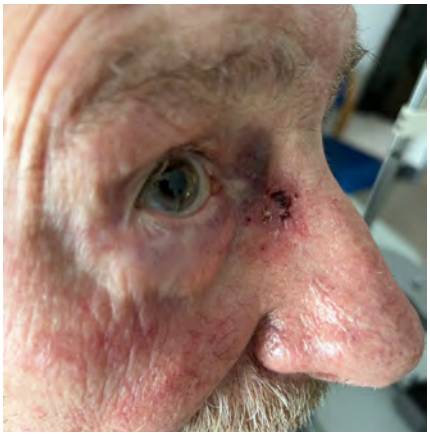

Figure 14: Potential squamous cell carcinoma present for ~12 months. This had been obscured by spectacle nose pad. This patient had already had two SCCs surgically removed from his scalp and from behind his left ear.

Intraocular melanomas

Consider routine referral for people if they have a skin lesion that raises the suspicion of a BCC. Typical features include:

- An ulcer with a raised rolled edge
- Prominent fine blood vessels around a lesion; or
- A nodule on the skin [particularly pearly or waxy nodules].

Only consider a suspected cancer pathway referral (for an appointment within 2 weeks) for people with a skin lesion that raises the suspicion of a BCC if there is particular concern that a delay may have a significant impact, because of factors such as lesion site or size.

Cutaneous squamous cell carcinoma

Cutaneous squamous cell carcinoma (cSCC) is the second most common non-melanoma skin cancer (NMSC).[57] As sun exposure is a major risk factor for cSCC, they arise commonly from the head and neck (**Figure 15**), commonly the ear, cheek, lip, and scalp.[58] SCC accounts for approximately 9% of all periocular cutaneous tumours (**Figures 16** and **17**).[49]

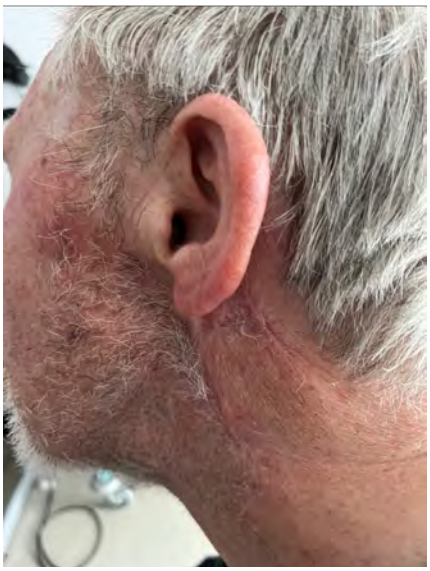

Figure 15: Site of a surgical successful excision of a confirmed squamous cell carcinoma (SCC) behind the left ear.

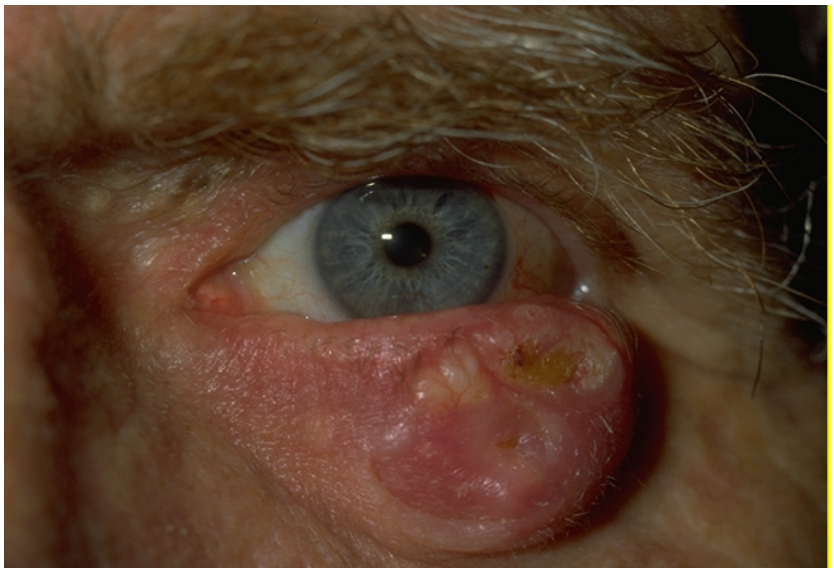

Figure 16:Squamous Cell Carcinoma of lower lid

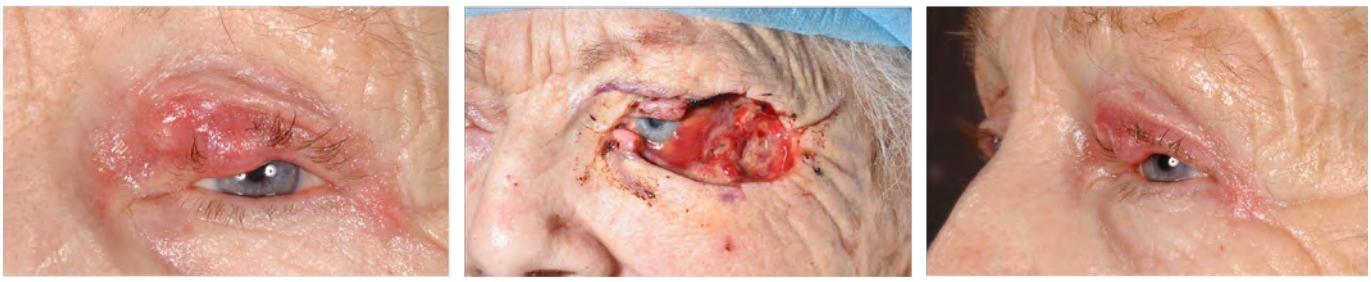

Figure 17: This patient has had an upper lid SCC, which recurred after previous Mohs' excision of an outer canthal BCC. The secondary SCC was also removed as shown, which then required extensive skin grafting.

SCC referral guidance

Consider a suspected cancer pathway referral (for an appointment within 2 weeks) for people with a skin lesion that raises the suspicion of squamous cell carcinoma.

Cataract

Cataract is the leading cause of blindness worldwide, and one of its main risk factors is solar UVR exposure.[59] Every year some 16 million people in the world suffer from blindness due to cataract. The World Health Organisation (WHO) has suggested that up to 20 per cent of cataracts may be caused by over-exposure to UVR and are therefore avoidable.[60]

The human lens absorbs near UV and far infrared light (<400 nm and >800 nm, respectively). It is known that UVR induces cataract, with a damage threshold at 350 nm.[61, 62] The cornea absorbs wavelengths below 295 nm, but allows longer wavelengths to reach the iris and lens. In adults, the lens of the eye absorbs all wavelengths below 370 nm, and greater than 98% of wavelengths between 370 nm and 400 nm, with higher absorbance in the posterior part of the lens.[52] In young children, the lens may transmit a greater proportion of shorter UV wavelengths (also they have larger pupils compared with adults allowing more light into the eye), enabling these to reach and potentially damage the retina.

Numerous epidemiologic studies have found that the risk factors for age-related cataract formation include:

- Age
- Sex
- Race
- Myopia

Modifiable risk factors include

- Smoking
- alcohol intake
- socio-economic status
- vitamin and protein deficiency
- Long term UVR exposure - now thought to be the most significant modifiable risk factor

After exposure to sunlight or artificial sources, biological damage may occur photochemically or thermally.[63, 64] Photochemical effects, typical of UVR and of shorter wavelengths of visible radiation as violet-blue, are essentially related to the absorption of photons by specific molecules in target tissues, including DNA, called chromophores. The effects related to the photochemical mechanism depend on the total dose, such as a result of the product between the duration of the exposure and the intensity of the radiation. Accordingly, high short-term exposure and less intense but more prolonged exposure can induce similar effects.[65]

There are three main sub-types of cataract:

- Nuclear
- Cortical
- Posterior subcapsular cataract

Epidemiological studies suggest a dose-dependent association between short wavelength UVR and cortical cataract.[66, 67] The lens nucleus is particularly susceptible to UV-A-induced stress leading to changes in the lens fluorescence, increased yellowing and loss of pyridine nucleotides by modulating gene expression and apoptotic stimuli in the lens epithelial cells. It has also been shown that in vivo exposure to sub-threshold doses of UVB can induce apoptosis in the lens epithelial cells but not in lens fibre cells.[68]

Repeated daily exposures to short wavelength UVR generate photochemically induced damage in the lens, and that short delay onset cataract after UVR exposure is photochemically induced. Daily high-intensity short wavelength IRR exposure of individuals, is associated with a higher prevalence of age-related cataract. One of the main determinants of individual long-term solar radiation exposure is outdoor work.[69, 70]

According to the WHO, the upper Population Attributable Fraction of cortical cataract due to solar UVR is 25%.[71] Furthermore, an increasing body of scientific data supports the role of solar UVR in inducing nuclear (**Figure 18**) and posterior subcapsular cataracts.[56] Taken as a whole, these data indicate that a reduction in excessive long-term solar UVR exposure could prevent a significant number of visual impairments and blindness worldwide, and a related reduction in treatment costs.

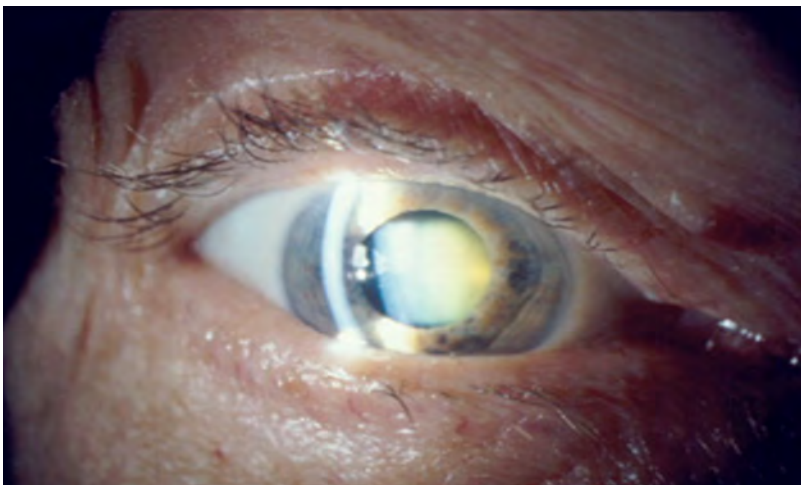

Figure 18: Cataract

Premature presbyopia

Little is known about the effects of UVR on presbyopia. Heat-induced denaturation of proteins in the crystalline lens is linked to reduced ability to focus and to cataract formation.[72] A high incidence of presbyopia occurs at younger ages in countries with high levels of UV.[73]

Glaucoma

Glaucoma is a leading cause of blindness worldwide. The most common type of glaucoma is primary open-angle glaucoma (POAG).[74] Oxidative UVA and UVB damage in the DNA of POAG patients has been described in the literature.[75] A significant correlation between oxidative DNA damage and raised IOP has also been suggested in patients with glaucoma, presumably caused by induced changes in the trabecular meshwork (**Figure 19**).[76]

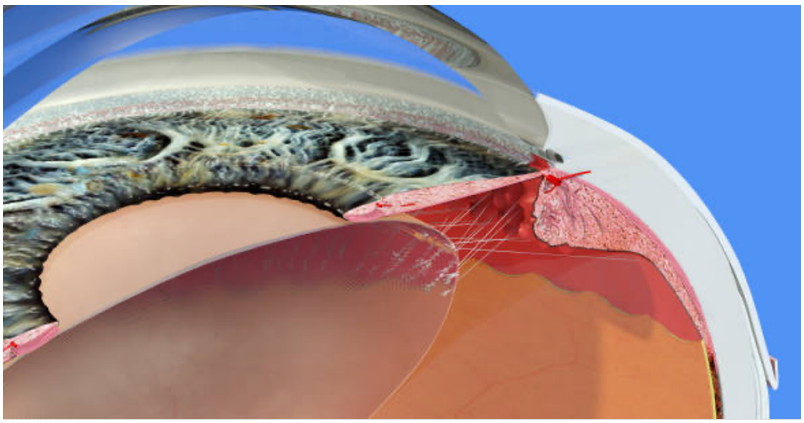

Figure 19: Glaucoma - UV effects on the trabecular meshwork

Reactive oxygen species (ROS) are natural by-products of cellular oxidative metabolism and play an important role in the modulation of cell survival, cell death, differentiation, cell signalling and inflammation-related factor production. One explanation for this is a genetic pre-disposition making them susceptible to ROS induced damage because of a more frequent deletion of specific genes pivotal for the antioxidant defence (AOX) mechanism. Oxidative stress (OS) has also been linked to POAG by increasing flow resistance in the aqueous humour of the eye in the presence of high levels of hydrogen peroxide. These reports suggest that OS is a critical factor involved in retinal ganglion cell (RGC) apoptosis in patients with POAG. While, all processes involved in RGC loss in glaucoma remain unclear, the contribution of OS is without doubt and therefore chronic exposure of the eye to UVR in daily life should be kept to a minimum.[77]

Age-related macular degeneration (AMD)

AMD is a leading cause of blindness in the western world. The causes of AMD are still not fully understood. Numerous studies have identified many risk factors for AMD. Risk factors that have been identified, include:

- Aging
- Gender
- Genetics
- Alcohol consumption
- Smoking
- Diet
- Cardiovascular function.

Several studies have investigated the possible associations between sunlight exposure and AMD; however, the results of those studies are unclear and inconsistent (**Figure 20**).

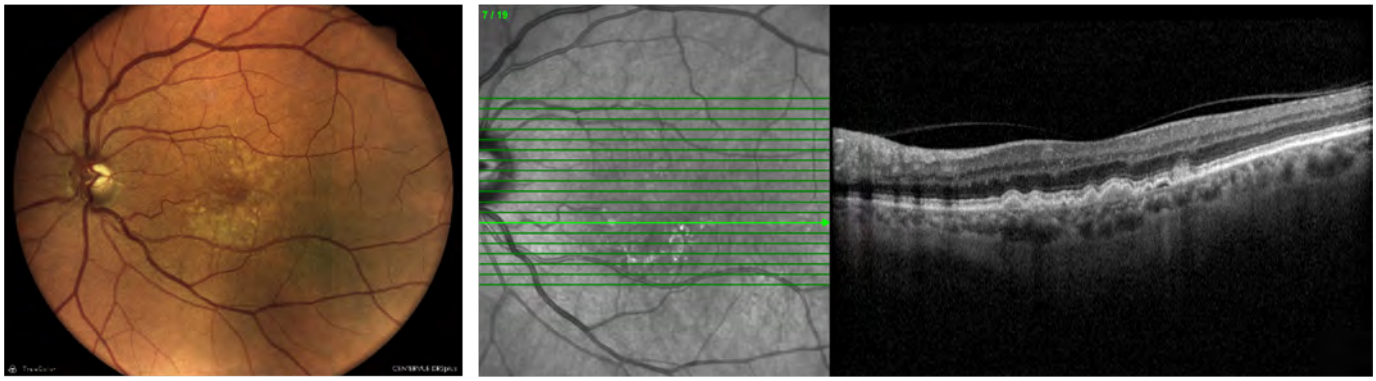

Figure 20: Dry Age related macular degeneration – links to UV exposure remain unclear

Many in-vitro and in-vivo studies have been focused on the association of sunlight and retinal pigment epithelium (RPE) cells.[78] Several studies have unequivocally demonstrated that either UV or blue light results in damage to RPE cells.[79, 80, 81, 82, 83, 84] Conversely, epidemiological evidence of the association between sunlight exposure and AMD is mixed, with one recent meta-analysis showing no relationship.[85] The possible reason is that most UVR is absorbed and blocked by the cornea and the lens and only very small amounts of UVR can reach the retina.[62] Also, the retina possesses inherent protection against damage via antioxidant enzymes such as:

- Superoxidase dismutase (SOD)
- Enzymes such as catalase and glutathione peroxidase
- Macular pigments such as melanin
- Haemoglobin
- Flavoproteins which absorb light.

Furthermore, photoreceptor cells can shed potentially damaged outer segment discs.[86]

While retinal damage due to increased UV radiation exposure can potentially still occur up to the age of 20, in adulthood, exposure of the retina to UV radiation can no longer be assumed, due to decreasing transmission properties of the natural lens. The natural lens, modern intraocular lenses, and wearing of sunglasses and other lenses with appropriate filter function, particularly in childhood and adolescence, provide a significant reduction in UVR retinal exposure (see Part 2).

Damage to the eye from drug-induced phototoxicity

A number of drugs absorb in the UV range and have phototoxic side effects affecting various structures in the eye.[87] For example, fluoroquinolone antibiotics such as ciprofloxacin and norfloxacin (used to treat ocular infections), in the presence of UVA radiation, may cause damage to epithelial cells and proteins of the lens. Exposure of the eye to UVA radiation while using these compounds may accelerate the development of cataract.[88] Use of ophthalmic formulations containing ketoconazole, diclofenac, or sulphacetamide were found to be toxic or irritating in the presence of UV-A radiation.[89]

UV radiation and reactivation of viruses

The association of intense exposure to UVR with subsequent reactivation of Herpes simplex virus 1 (HSV), causing cold sores of the lip, is well described.[42] The presence of IgM class antibodies to HSV reflects recent viral activity, either primary or recurrent infection (**Figure 21**). In a recent study from Sweden, the odds for anti-HSV IgM positivity were nearly twofold higher in summer than winter consistent with UV-induced reactivation of HSV, with or without the manifestation of cold sores.[90]

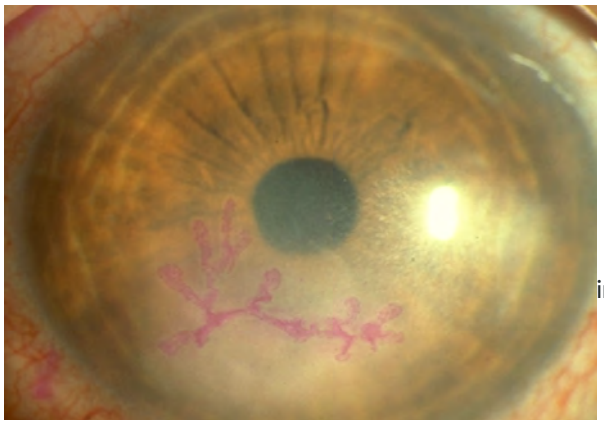

Figure 21: Herpes simplex virus can recur following excessive UV exposure

Summary

The first part of this two-part review has discussed the impact of UVR over exposure on the human eye and the evidence in the literature regarding the impact this may have on various ocular structures. Recognising and appropriately managing a range of skin conditions caused by UVR over exposure has also been discussed with reference to latest NICE guidance and College of Optometrists clinical management guidelines (CMG). In Part 2, the final part of this series, various methods of UVR protection will be discussed.

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How to explain that a piece of CPD benefits your practice and patients

Below are examples of situations where linking CPD to your practice may be less obvious. We've shown how you might link a learning objective to your own practice and patient care where the GOC might feel it is slightly out of core scope of practice:

For a DO completing a contact lens session

This CPD has the learning objective *Practitioners will have a greater understanding of the features, benefits and contraindications of toric contact lenses*. As a dispensing optician I am frequently asked by patients about their options for vision correction, and a common question is how to manage the problem of spectacles steaming up during mask wear which may be required throughout the working day. In order to help them I need to explain the options not only for spectacles but also for dual wear of spectacles and contact lenses, and know what products may suit their prescription. It is therefore important I am up to date with toric contact lenses and the patients who are and are not suitable for this form of vision correction so I can answer patient queries accurately and provide appropriate advice and care.

For an optom completing a therapeutics session

This CPD has the learning objective *Practitioners will have a greater understanding of the therapeutic management of red eye conditions*. As an optometrist, the more understanding of therapeutic options I have the better to enable me to give patients I decide to refer rather than to manage in practice information and reassurance about what may be considered in the next steps of their care pathway, to enhance their satisfaction with their care. Also as part of my CPD plan I am working towards taking on part-time work in a hospital clinic and therefore enhancing my understanding of therapeutic treatments of ocular conditions will support my development in preparation for this new role.

For a CLO competing an optometry session

This CPD has the learning objective *Practitioners will have a greater understanding of good record keeping and referral decision-making*. The cases involved investigation and diagnosis of anterior eye conditions and best practice in documentation of the results. I am part of a multidisciplinary team which is involved in a MECS eyecare scheme so I carry out under supervision diagnostic tests and complete records relating to collecting baseline data and investigation/ management of MECS patients. This session broadened my understanding of best practice in care and documentation relating to anterior eye conditions which I could come across in my MECS work.

How to log your CPD points with the GOC

In the CPD scheme the provider does NOT notify the GOC of your points.

To claim your CPD points you must enter the details on the GOC site MyGOC before the end of each calendar year.

You will be asked to provide the C-reference (see the front of this article), which will populate some of the details of this CPD unit. You will also be asked for evidence of completion. The evidence of completion you will need is the certificate which is automatically saved to your iLearn account when you complete the CPD.

More information on how to plan, access and record the CPD and download the certificate is available by clicking on the following link: CPD Information

Please send us your feedback

We would be very pleased for feedback on this or any other CPD we provide. Please give us feedback by clicking on the link provided in the confirmation email after you have submitted the quiz.