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Review

Acute effects of UVR on human eyes and skin

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Abstract

Solar UVR (~295–400 nm) has acute clinical effects on the eyes and the skin. The only effect on the eye is inflammation of the cornea (photokeratitis), which is caused by UVB (and non-solar UVC) and resolves without long-term consequences within 48 h. The effects on the skin are more extensive and include sunburn (inflammation), tanning and immunosuppression for which UVB is mainly responsible. Tanning is modestly photoprotective against further acute UVR damage. Skin colour is also transiently changed by UVA-dependent immediate pigment darkening, the function of which is unknown. Skin type determines sensitivity to the acute and chronic effects of UVR on the skin. Some of the photochemical events that initiate acute effects are also related to skin cancer. Solar UVB is also responsible for the synthesis of vitamin D.

Keywords: Skin; Eye; Ultraviolet radiation; Sunburn

Contents

1. Introduction

The human body is exposed to terrestrial sunlight that contains UVB (~295–315 nm), UVA (315–400 nm), visible (400–800 nm) and infrared (IR) (800 nm–1 mm) radiation. UVB wavelengths <295 nm and UVC

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(100–280 nm) radiation are totally filtered by the stratospheric ozone layer. The ratio of UVB to UVA depends on the solar zenith angle that depends on latitude, season and time of day. The absolute and relative quantity of UVB is greatest when the sun is high in the sky. However, the sun is primarily a UVA source with a maximal terrestrial UVB content of about 5%.

Visible radiation is detected by the eye and used for sight. IR is detected by the skin's sensory system but the body has no specialized UVR sensory system. The skin is the body's main interface with the environment and is frequently exposed to UVR with either intentional or unintentional exposure to the sun. The eye is also exposed to UVR. This paper focuses on the acute effects of solar UVR on the eye and the skin, but from a health point of view the chronic effects are more important. In this context it should be remembered that chronic effects on the eye, such as cataracts, and the skin, such as cancer, are the consequence of decades of multiple acute effects that are most often sub-clinical.

2. Eye

The eye is a complex multi-layered organ that receives visible radiation on its retina. The intermediate layers attenuate UVR to different degrees and thereby protect the retina from UV photodamage. The outermost cornea absorbs UVC and a substantial amount of UVB, which is further attenuated by the lens and the vitreous humor in front of the retina. UVA is less well attenuated by the cornea but is attenuated by the internal structures so it does not reach the retina (Sliney, 2001; Roberts, 2001; Johnson, 2004).

The only acute clinical effect of UVR on the eye is photokeratitis that is also known as snow blindness or welder's flash (Sliney, 2001; Roberts, 2001; Johnson, 2004). This is a painful transient inflammatory condition caused by UVC and UVB-induced damage to the corneal epithelium. Typically it appears 6–12 h after exposure and resolves, without long-term consequences, within 48 h. In some ways it can be regarded as sunburn of the eye.

3. Skin

The skin comprises two main layers: (i) the outer cellular epidermis and (ii) the inner largely extracellular dermis. The main epidermal cell type is the keratinocyte, a major function of which is to differentiate till death to create the outermost cornified protective stratum corneum. Keratinocytes are regularly renewed by cell division in the epidermal basal layer. Dendritic pigment producing melanocytes and immunocompetent dendritic Langerhans cells (LC) are also present in the epidermis. The dermal connective tissue is mostly collagen synthesized by fibroblasts. The dermis also contains the skin's vascular supply.

4. Chromophores that initiate the skin's response to UVR

The skin contains a range of chromophores (Young, 1997) that initiate photochemical and photobiological events. The most important is likely to be nuclear DNA that undergoes base structural change on absorption of UVR. A wide range of photolesions are formed but the most common are *di*-pyrimidine lesions such as cyclobutane pyrimidine dimers (CPD) and pyrimidine-(6–4)-pyrimidones widely known as 6–4 photoproducts (6–4). CPDs and 6–4 are readily detected in human epidermis and dermis immediately after exposure with solar simulation radiation (SSR), UVB and UVA (Chadwick et al., 1995; Young et al., 1998). Wavelength dependence studies (action spectroscopy) for CPD in human skin in vivo show that UVB is 3–4 orders of magnitude more effective that UVA (Young et al., 1998). Epidermal CPD and (6–4) are repaired by a process called excision repair (Bykov et al., 1999; Young et al., 1996) which is dependent on a complex of repair enzymes. Failure to repair *di*-pyrimidine lesions, as is the case in patients with the rare genetic disorder xeroderma pigmentosum (XP), results in multiple skin cancers at an early age.

Trans-urocanic acid (UCA), a deamination product of histidine, is an important chromophore found in high concentrations in the stratum corneum. *Trans*-UCA undergoes a photoisomerization to *cis*-UCA in the presence of UVR. There is a considerable body of evidence that *cis*- but not *trans*-UCA has immunoregulatory properties (Norval and El Ghorr, 2002). UVB is much more effective than UVA in the trans to cis photoisomerization in human skin in vivo (McLoone et al., 2005).