Chapter 6
Other beneficial effects of sunscreens

Other potential beneficial effects of sunscreens that are not related to the prevention of skin cancer are prevention of painful sunburns, photodamage and photoageing, UVR-induced provocation of certain cutaneous diseases and photoimmune suppression. Use of sunscreens can prevent skin diseases from progressing acutely after exposure to the sun; these diseases include cutaneous lupus erythematosus (Taylor & Sober, 1996) and reactivation of herpes labialis (Rooney *et al.*, 1991). The other potential benefits of sunscreens are related to the type and duration of exposure to UVR. Prevention of photodamage and photoageing, which are related to cumulative exposure to UVR, in countries where solar irradiance is intense throughout the year requires daily, long-term sun protection (Fig. 40). Prevention of acute flares of cutaneous diseases, which may be related to episodic exposure to UVR, requires anticipatory use of sun protection. As an episode of exposure that will provoke a flare is difficult to predict reliably, people with skin diseases that are highly sensitive to the sun, such as chronic actinic dermatitis, are advised to protect themselves from the sun daily, with measures that include a broad-spectrum sunscreen.

Prevention of non-carcinogenic dermal effects of the sun

The type of protection required to prevent chronic photodamage may differ from that for preventing acute sunburn, as cutaneous injury as defined by histological changes is different from acute sunburn and photodamage. In acute sunburn, the most obvious change is epidermal, the dermal changes being subtle and transient. The changes seen with chronic photodamage are alterations to the epidermis and dermis.

Exposure to sunlight and blistering sunburns may occur more frequently during childhood and adolescence than later in life (Robinson *et al.*, 1997b). Sporadic or incomplete use of sun protection leads to episodic burning. In a study in the USA, although more adults claimed to use sunscreens in 1996 than in 1986, the proportion who reported having a sunburn was higher in 1996 (Robinson *et al.*, 1997a). Use of sunscreens with a high SPF has been associated with longer recreational exposure to the sun (Robinson, 1992; Autier *et al.*, 1999). Other examples of sunburn due to non-compliance with the recommended use of sunscreen include applying sunscreen after exposure has begun or when the first symptoms of sunburn are recognized or expected; failure to reapply sunscreen after swimming; and applying inadequate amounts and missing certain areas of the body, especially the ears, neck, feet and legs.

Figure 40 Repeated exposure to UVR induces pigmentation (bottom right). Application of a sunscreen before each exposure (bottom left) reduces this pigmentation, whereas application of a placebo with no UVR absorbers has no effect (top left).

Another reason that burning might occur even with use of sunscreen is that acclimitization (tanning and hyperplasia) is largely inhibited when sunscreens are initially applied effectively. Therefore, if on a subsequent occasion sunscreen is not applied, the skin remains vulnerable to burning.

Prevention of cumulative effects of exposure to the sun

Excessive exposure to UVR leads to premature ageing of the skin, with excessive wrinkles. Photo-damaged skin may have a rough, leathery appearance. The cumulative effects of the exposure weaken the skin's elasticity and tensile strength, resulting in sagging of the skin of the cheeks, deeper facial wrinkles and skin discolouration later in life (Gilmore, 1989). The skin becomes yellowish, with mottled hyperpigmentation, telengiactasia (Fig. 41) and purpura. The epidermis is thinned and lacks downward protuberance of rete ridges. Basophilic degeneration of the uppermost reticular dermis, which is common in sun-damaged skin, consists of swelling, coarsening and late homogenization of connective tissue in the upper dermis. Fibroblasts produce abnormal elastin and collagens. Matrix metalloproteins are also altered during repeated exposure, leading to photoageing, and this occurs at very low doses of UVR (Fisher *et al.*, 1996).

There is no established action spectrum for photoageing and it is not known whether the action spectrum is different from that for erythema. Repeated sub-erythemagenic doses of UVA induce stratum corneum thickening and epidermal hyperplasia in humans and an increased number of inflammatory cells in the dermis (Lavker *et al.*, 1995a). A persistent dermal infiltrate may lead to connective tissue damage and perhaps even to elastosis (Lavker & Kligman, 1988). This persistent dermal inflammation resulting from repeated sub-erythemagenic doses of UVR may adversely affect dermal cellular components, with fibrosis as one of the end-points. Cytokines released by lymphocytes and mast cells can alter collagen and elastin production and vascular reactivity. Increased release of lysozyme during the initial phases of dermal inflammation (Kajiki *et al.*, 1988) may be related to the increased staining of lysozyme seen in UVA-irradiated elastic fibres. Boyd *et al.* (1995) reported that solar elastosis was at least partly repaired over 2 years in people who used sunscreens with an SPF of 2–9 every day but not in those who applied a placebo. In a study of 10 people irradiated once daily for 28 days with 11 MED of solar-simulated UVR through an SPF-22 sunscreen, epidermal hyperplasia, stratum corneum thickening, inflammation of the dermis and deposition of lysozyme in the dermis occurred more frequently than on an unirradiated site (Lavker *et al.*, 1995b). Another study showed that sunscreens can reduce acute changes associated with exposure to UVR which may be related to photoageing, including deposition of lysozyme and α-1-antitrypsin on dermal collagen fibres, with up-regulation of matrix metalloproteinase-2 (Seité *et al.*, 2000b). Studies in experi-

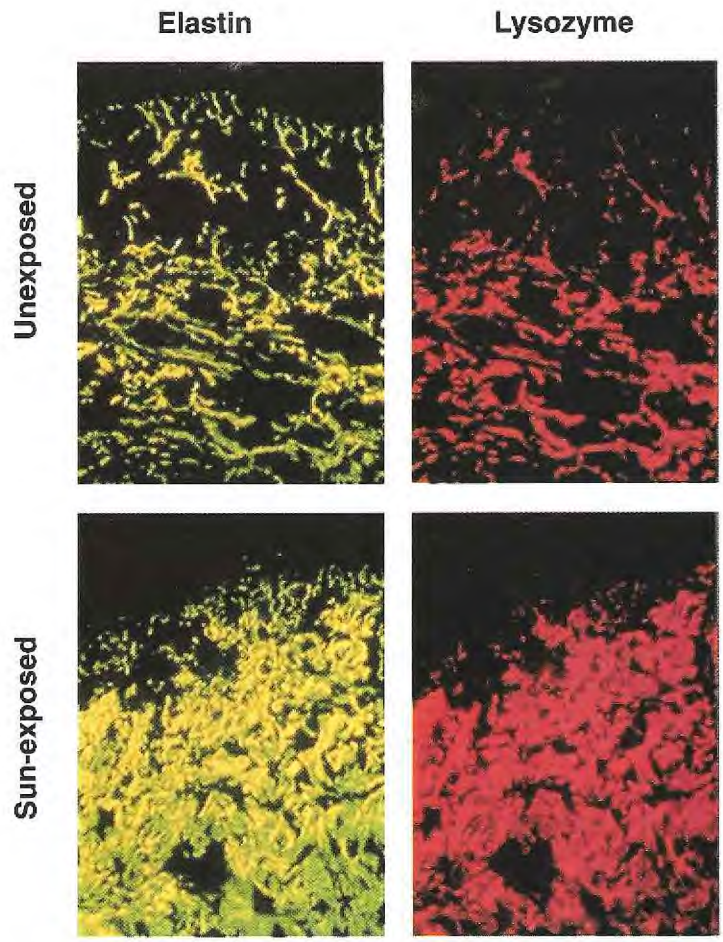

Figure 41 Telangiectasia, a lesion induced in blood vessels by chronic exposure to UVR

mental animals suggest that sunscreens can prevent or allow repair of many of these changes (Kligman *et al.*, 1982, 1983; Kligman, 1989).

Since no single action spectrum defines long-term photodamage, sunscreens that provide protection against a broad spectrum are recommended to prevent such damage (Bergfeld *et al.*, 1997). As it has been estimated that at least 24–48 h are required for skin to recover from a single, sub-threshold exposure, small, repeated doses of UVR within this interval result in cumulative damage (Arbabi *et al.*, 1983). The recovery interval is often cited in recommending daily use of sunscreens by people who spend significant amounts of time outdoors in regions where there is intense sunlight. In areas with less sunlight, daily use of sunscreens may be less important.

Prevention of UVR-induced stimulation of cutaneous diseases

Idiopathic photodermatoses

Idiopathic photodermatoses develop only with exposure to light. Severely affected persons, such as those with chronic actinic dermatitis or hydroa vacciniforme, must use physical measures of protection, and sunscreens play a minor role in the management of these disorders. People who are less affected, such as patients with polymorphic light eruption or juvenile springtime eruption, can have controlled, gradual exposure to the sun which allows the build-up of natural defences (Moyal & Binet, 1997) and may permit nearly normal exposure during summer months.

Photoaggravated dermatoses

Under certain circumstances, diseases of various etiologies can be aggravated by sunlight in patients who on other occasions may react normally. These diseases include lupus erythematosus, lichen planus and herpes simplex. The disease most frequently recognized as requiring careful photoprotection from both UVB and UVA is lupus erythematosus in the discoid, systemic and subacute forms.

Exposure of patients with systemic lupus erythematosus or of mice susceptible to a similar disease results in both systemic and cutaneous manifestations (Sakane *et al.*, 1978; Strickland, 1984). Patients with systemic lupus erythematosus show decreased proliferation of T cells in the autologous mixed leukocyte reaction. This deficiency may be due to the impaired suppressor T-cell function seen in this disease. Hence, in patients with lupus erythematosus, unrestrained autoreactive T-cell and B-cell responses to UVR-induced antigens may lead to cutaneous inflammation, autoantibody production or both. Sunscreens with an SPF of at least 15, providing protection against both UVB and UVA, are required (Drake *et al.*, 1996). The available evidence suggests that regular use of sunscreens reduces morbidity from both cutaneous and systemic lupus erythematosus (Callen *et al.*, 1991; Vila *et al.*, 1999).

Photodermatoses related to congenital abnormalities

Patients with xeroderma pigmentosum, Bloom syndrome, Cockayne syndrome, Rothmund-Thomson syndrome or Smith-Lemli-Optiz syndrome (Anstey *et al.*, 1999) can develop an erythematous, papular, exaggerated sunburn or other symptoms of photosensitivity. Treatment depends on avoidance of UVR by wearing photoprotective clothing, sunglasses and a broad-spectrum sunscreen (Schaefer *et al.*, 2000).