PHOTOPROTECTION

Since photosensitivity of the skin results from exposure to sunlight, it follows that absolute avoidance of sunlight will eliminate these disorders. However, contemporary lifestyles make this approach impractical for most individuals. Thus better approaches to photoprotection have been sought.

Natural photoprotection is provided by structural proteins in the epidermis, particularly keratins and melanin. The amount of melanin and its distribution in cells are genetically regulated, and individuals of darker complexion (skin types IV–VI) are at decreased risk for the development of acute sunburn and cutaneous malignancy.

Other forms of photoprotection include clothing and sunscreens. Clothing constructed of tightly woven sun-protective fabrics, irrespective of color, affords substantial protection. Wide-brimmed hats, long sleeves, and trousers all reduce direct exposure. Sunscreens are now considered over-the-counter drugs, and a monograph from the U.S. Food and Drug Administration (FDA) has recognized category I ingredients as safe and effective. Those ingredients are listed in Table 75-5. Sunscreens are rated for their photoprotective effect by their sun protection factor (SPF). The SPF is simply a ratio of the time required to produce sunburn erythema with and without sunscreen application. The SPF of most sunscreens reflects protection from UV-B but not from UV-A. The FDA monograph stipulates that sunscreens must be rated on a scale ranging from minimal (SPF ffi2 and <12) to moderate (SPF ffi12 and <30) to high (SPF ffi30, labeled as 30+).

Broad-spectrum sunscreens contain both UV-B-absorbing and UV-A-absorbing chemicals, the latter including avobenzone and ecamsule (terephthalylidene dicamphor sulfonic acid). These chemicals absorb UVR and transfer the absorbed energy to surrounding cells. In contrast, physical UV blockers (zinc oxide and titanium dioxide) scatter or reflect UVR.

In addition to light absorption, a critical determinant of the sustained photoprotective effect of sunscreens is their water resistance. The FDA monograph has defined strict testing criteria for sunscreens that claim to possess a high degree of water resistance.

Some degree of photoprotection can be achieved by limiting the time of sun exposure during the day. Since a large part of an individual's total lifetime sun exposure may occur by age 18, it is important to educate parents and young children about the hazards of sunlight. Simply eliminating exposure at midday will substantially reduce lifetime UVR exposure.

TABLE 75-5 FDA CATEGORY I MONOGRAPHED SUNSCREEN INGREDIENTS[a]

Ingredients	Maximum Concentration, %
p-Aminobenzoic acid (PABA)	15
Avobenzone	3
Cinoxate	3
Dioxybenzone (benzophenone-8)	3
Ecamsule	15
Homosalate	15
Methyl anthranilate	5
Octocrylene	10
Octyl methoxycinnamate	7.5
Octyl salicylate	5
Oxybenzone (benzophenone-3)	6
Padimate O (octyl dimethyl PABA)	8
Phenylbenzimidazole sulfonic acid	4
Sulisobenzone (benzophenone-4)	10
Titanium dioxide	25
Trolamine salicylate	12
Zinc oxide	25

[a]FDA, U.S. Food and Drug Administration.

PHOTOTHERAPY AND PHOTOCHEMOTHERAPY

UVR can be used therapeutically. The administration of UV-B alone or in combination with topically applied agents can induce remissions of many dermatologic diseases, including psoriasis and atopic dermatitis. In particular, narrow-band UV-B treatments (with fluorescent bulbs emitting radiation at ~311 nm) have enhanced efficacy over that obtained with broad-band UV-B in the treatment of psoriasis.

Photochemotherapy in which topically applied or systemically administered psoralens are combined with UV-A (PUVA) is effective in treating psoriasis and the early stages of cutaneous T cell lymphoma and vitiligo. Psoralens are tricyclic furocoumarins that, when intercalated into DNA and exposed to UV-A, form adducts with pyrimidine bases and eventually form DNA cross-links. These structural changes are thought to decrease DNA synthesis and to be related to the amelioration of psoriasis. Why PUVA photochemotherapy is effective in cutaneous T cell lymphoma is only partially understood, but it has been shown to induce apoptosis of atypical T lymphocyte populations in the skin. Consequently, direct treatment of circulating atypical lymphocytes by extracorporeal photochemotherapy (photopheresis) has been used in Sézary syndrome as well as in other severe systemic diseases with circulating atypical lymphocytes, such as graft-versus-host disease.

In addition to its effects on DNA, PUVA photochemotherapy stimulates epidermal thickening and melanin synthesis; the latter property, together with its anti-inflammatory effects, provides the rationale for use of PUVA in the depigmenting disease vitiligo. Oral 8-methoxypsoralen and UV-A appear to be most effective in this regard, but as many as 100 treatments extending over 12–18 months may be required for satisfactory repigmentation.

Not surprisingly, the major side effects of long-term UV-B phototherapy and PUVA photochemotherapy mimic those seen in individuals with chronic sun exposure and include skin dryness, actinic keratoses, and an increased risk of skin cancer. Despite these risks, the therapeutic index of these modalities continues to be excellent. It is important to choose the most appropriate phototherapeutic approach for a specific dermatologic disease. For example, narrow-band UV-B has been reported in several studies to be as effective as PUVA photochemotherapy in the treatment of psoriasis but to pose a lower risk of skin cancer development than PUVA.