Although most members of our society still idolize a suntan, we are all becoming increasingly aware that exposure to the sun can be a negative factor in our lives, causing premature aging, premalignant and malignant skin lesions, and thus earlier death.

In man, sunlight is necessary for vitamin D synthesis, the absence of which causes rickets. Our office does not treat the medical problem of underexposure to sunlight, but we do see the effects of acute and chronic overexposure. These will be discussed and illustrated in the following pages, together with a discussion of their effective management and prevention.

Acute Overexposure: Sunburn
Melanin is the pigment in our skin that acts as a light absorber and thus is the skin’s major natural protection against sun damage. Melanin is produced by melanocytes that lie between basal cells at the bottom of the epidermis. While the number of melanocytes differs little among different races, the activity of these melanocytes varies greatly, both normally and with stimulation by sunlight. It is this increase in production of melanin that causes black skin to be so much darker than that of the light Caucasian. Albinos lack an enzyme necessary for the chemical change to produce the melanin. Albinos do have melanocytes, but they cannot produce true melanin, and thus their skin remains light-colored despite external solar stimulation. Such skin is particularly susceptible to sun damage. Patients suffering from vitiligo, or piebald skin, also lack melanin, but for a different reason. The patches of vitiligo truly lack melanocytes. Without this pigment factory, skin color reverts to a milky white and also loses natural protection against the sun.

Sunburn is a common but uncomfortable experience most frequently experienced in the beginning of summer before acquiring a protective tan. Redheads and blondes are most susceptible, whereas brunets usually tan quickly. Blacks can get a sunburn, but it requires extensive and prolonged exposure.

The onset of erythema and subsequent tenderness varies with skin color and intensity of exposure. Blonds may note ery-
Thema after only 30 minutes, while dark-completed persons note redness within 24 hours. Local heat and burning are the first symptoms. In severe burns, erythema is followed by edema and blister formation (Fig 1). This can be terribly painful and may persist for several days. The subsequent desquamation can be marked (Fig 2). Early treatment with systemic oral prednisone, 30 to 40 mg a day for five to seven days, can prevent much morbidity.

Prostaglandins probably play a role in the sunburn reaction. Antiprostaglandins like aspirin and indomethacin have been used experimentally to reduce such reaction. After the sunburn reaction, pigmentation usually increases and may be marked when compared to sites not exposed to the sun. Such tanning provides natural protection for subsequent exposure to the sun. However, the genetic predisposition of some people makes the melanocytes incapable of such a sun-induced tan. Their feeble response produces only freckling. Such persons run a high risk of developing skin cancers.

Acute Sun-Exposure
Although sunburn is the most common adverse effect of acute exposure to the sun, it is by no means the only one. Many drugs may sensitize a patient so that the amount of sunlight they could formerly enjoy without any reaction causes a photoallergic or phototoxic reaction. As a rule, such light-hypersensitivity states are most frequently manifested in the areas exposed to the sun: the face, the V of the neck, the forearms, and the dorsa of the hands. Symmetrical symptoms are often the clue that the sun is the culprit (Fig 3). Changes in the skin are general and not specific for the inducing wavelength of light. They include erythema, dermatitis, urticaria, vesicles, bullae, and hyperpigmentation and hypopigmentation.

Some antibacterial soaps contain substances like tribromosalicylanilide that can induce photoallergic contact dermatitis. Discovering that there was a recent change in soaps (for example, after increasing exposure to the sun and using soaps from a motel) is helpful in making this diagnosis.

Drug-induced photosensitivity reactions are adverse skin reactions that result from the interaction of light with drugs present in the skin at the time of irradiation. The drug may have reached the skin through the oral, topical, or parenteral route. Blacks have a lower incidence of drug photosensitivity, presumably due to their inherently greater melanin protection. The two major types of drug-induced photosensitivity are the phototoxic and the photoallergic reactions. The distinction can be made on clinical and histologic grounds. (Table 1).

The most common photosensitizing groups of drugs include sulfonamides, sulfonylureas, chlorothiazides, tetracyclines (especially dimethylichlorotetacycline), halogenated salicylan-
TABLE 1
Distinctions Between Phototoxic and Photoallergic Reactions

<table>
<thead>
<tr>
<th></th>
<th>Phototoxic</th>
<th>Photoallergic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>High (theoretically 100%)</td>
<td>Low</td>
</tr>
<tr>
<td>Clinical changes</td>
<td>Mimics a sunburn often with blisters and hyperpigmentation</td>
<td>Varied morphology, little hyperpigmentation</td>
</tr>
<tr>
<td>Reaction possible on first exposure</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Incubation period needed after first exposure</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Chemical alteration of photosensitizer</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Concentration of drug necessary for reaction to occur</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Passive transfer, macrophage inhibition, lymphocyte simulation test</td>
<td>No</td>
<td>All are possible</td>
</tr>
<tr>
<td>Flares at distant, previously involved sites</td>
<td>No</td>
<td>Can occur</td>
</tr>
<tr>
<td>Action spectrum</td>
<td>Usually similar to absorption spectrum</td>
<td>Usually higher wavelength than absorption spectrum</td>
</tr>
<tr>
<td>Recurrence from exposure to ultraviolet light alone</td>
<td>No</td>
<td>May occur in persistent light eruptions</td>
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The porphyrias are a group of genetically determined or acquired disorders characterized by defect in pyrrole metabolism. The heme that transports oxygen as hemoglobin in the human red blood cell is a porphyrin. In porphyria cutanea tarda there is an excessive excretion of uroporphyrin and coproporphyrin in both urine and feces with symptoms occurring in adult life. Porphyria cutanea tarda is the most frequently diagnosed porphyria in the United States. Its incidence seems to be increasing, perhaps due to increased use of oral contraceptives. Sun-exposure in the 400-nm range induces lesions because of the presence of uroporphyrin deposited in the skin. The primary lesion in porphyria cutanea tarda is a vesicle or blister. The more severe the sun-exposure, the more severe the sub-epidermal vesication and bullae formation. Facial hutchitis is also common. Type 1 urinary uroporphyrin levels are usually sharply elevated. Twenty-five percent to 50% of patients with porphyria cutanea tarda have diabetes mellitus. It should always be looked for in any patient with porphy-

...silides (found in topical cosmetic products including antibacterial soaps), phenothiazines, furocoumarins, and coal tar. Since there are no diagnostically abnormal chemical, biologic, or serologic findings associated with drug photosensitivity, the diagnosis depends upon adequate history, keen observation, and sometimes on the use of photopatch testing. Photopatch testing can effectively confirm the induction of the skin reaction by cutaneous contact. The suspected agent is diluted with petrolatum or acetone and is then applied to nonaffects sun-spared skin under a nonocclusive patch. Light of the appropriate wavelength is then given over the area and tests are read at one-, two-, and three-day intervals. Duplicate patches are applied without subsequent light exposure to rule out a simple contact dermatitis.
ria cutanea tarda. Abnormal liver function is also common.

Some drugs known to induce porphyria cutanea tarda include alcohol, barbiturates, sulfonamides, chloroquine, griseofulvin, tolbutamides, chlorpromazine, diethylstilbestrol, and oral birth control pills. Treatment for porphyria cutanea tarda must first include cessation of any drug that might induce it. Phlebotomy (blood-letting) is the next treatment of choice, usually 500 ml at two-week intervals until the 24-hour uroporphyrin levels are markedly reduced. Chloroquine has been used but serious ocular reactions can cause blindness. Protective clothing and broad-spectrum sun-blockers should be used.

Xeroderma pigmentosum is a rare disease characterized by extreme sensitivity to sunlight in the 290 nm to 320 nm range. Patients lack an enzyme for repair of sun-induced DNA damage. Thus, premalignant and malignant changes caused only by prolonged exposure to sunlight in normal persons are seen in these patients when they are children or teenagers. Without severe restrictions to protect their skin from virtually all sunlight, they die of skin cancers by their early twenties.

Patients with systemic lupus erythematosus also suffer from sunlight sensitivity. Skin lesions of systemic lupus erythematosus are often initiated by ultraviolet light. Even fatal exacerbations of the disease have been noted after overexposure to sunlight of the sunburn (erythrogenic) spectrum (290 nm to 320 nm). Sunlight can also initiate viral disease like herpes simplex (Fig 4).

Chronic Changes of the Skin

The sun sends a spectrum of radiation to earth that varies from very short gamma radiation to long wavelength radio waves. The ozone layer 500 miles above the earth’s surface effectively shields most shorter wavelength light from the earth’s surface. Photobiologists and dermatologists have divided the ultraviolet light that does reach us into three basic groups: ultraviolet A, 400 nm to 315 nm; ultraviolet B, 315 nm to 280 nm; and ultraviolet C, less than 280 nm. From now on we shall be talking primarily about ultraviolet B. It is radiation at this wavelength that is primarily responsible for the aging and malignant changes, as well as the sunburn reaction.

What transforms the even-textured, smooth-surfaced skin of the infant to the blotchy, irregularly pigmented skin of the sun-damaged person? Sunlight! Sun-exposure is the answer far more often than age itself, since many octogenarians who have effectively shielded their skin from harmful solar radiation have fairer skin than the sun-worshipping 30-year-old. A single exposure to erythrogenic ultraviolet light has been shown to inhibit DNA, RNA, and protein synthesis. Thymidine dimers have been isolated and lyosomal rupture may occur. The sun-worshipper or outdoorsman is often quite unaware that any of these changes are occurring within the body. Yet simply avoiding acute sunburn is not enough to prevent the aging changes that most of us fear.

Commonly seen changes of chronic sun-exposure include the tiny red vessels of telangiectasia, skin laxity and solar elastosis due to loss of elasticity from degeneration or faulty production of elastic and collagen fibers in the upper dermis, wrinkling and leather-like changes, and the brownish flat “sun spots” of solar lentigo. All these skin signs are signals that the patient has had too much sun, but these are just the forerunners to the more severe and debilitating premalignant and malignant lesions.

Premalignant Changes in the Skin

The skin provides a unique background upon which we can closely observe the gradual formation of malignant growths. Although the above signs of sun damage form the fertile ground for malignant growths, the solar keratosis is the premalignant growth that is associated with (and may become) a true skin malignancy.
The solar keratosis is clearly born of the sun, and is therefore the most common on the face, hands, ears, and forearms. These lesions are dry, scaly, and often have a red base. A patient may pick off the superficial scale, only to have it recur. Some solar keratosis lesions may bleed. Understandably, they are most prevalent in fair-skinned, blue- or green-eyed individuals who have spent a lifetime out in the sun. Sailors, farmers, and tennis and golf enthusiasts run a great risk. Such premalignant growths should be treated, and not to do so is flirting with subsequent malignant transformation.

Treatment methods vary. Cryosurgery with topical liquid nitrogen is clean and effective, and, moreover, it does not usually leave the scarring that can occur from electrodessication and curettage. This -196°C solution is applied to the affected area. A blister may form subsequently, followed by crust and desquamation, leaving new, slightly erythematous skin in its place. With appropriate sun protection, scarring is minimal. When patients have widespread solar keratoses, topical 5-fluorouracil is most effective. Topical application twice a day for four to six weeks initially causes premalignant areas to become red, crusty, and tender. Topical corticosteroids and oral antarctic agents like hydroxyzine (Atarax) may lessen patient discomfort. This safe topical regimen does not leave scars, but occasional postinflammatory hyperpigmentation and hypopigmentation may take months to resolve. The photographs of patients taken before and after such therapy are often dramatic (Fig 5). 5-fluorouracil does not clinically alter or inflame normal skin.

Although there are many types of skin cancers, solar radiation has been found to be a major factor in pathogenesis of the three most common skin malignancies: basal cell carcinoma, squamous cell carcinoma, and malignant melanoma. Skin cancer is the most common human malignancy and can be deadly. One percent of all cancer deaths in the United States are from cancer of the skin.

**Basal Cell Carcinoma**

The basal cell carcinoma is the most common type of skin cancer in the United States. Sunlight is a primary etiologic factor in its development, as shown by the fourfold greater incidence of basal cell carcinomas in the South and Southwest than in the North. It is most commonly found on sun-exposed areas of the face, ears,
neck, and legs, and is usually round or oval with raised reddish borders of a pearly hue. It is often covered with superficial telangiectasia and has central erosion or ulceration. This accounts for the common history of intermittent bleeding.

Some basal cell carcinomas are pigmented and may reach huge proportions if unchecked (Fig 6). They can enlarge slowly but relentlessly, often following the path of blood vessels and nerves, eroding underlying muscle and even bone. Metastasis via the bloodstream or lymphatics is rare, occurring in about 0.1% of cases. Thus, death is usually due to local extension of the primary growth.

Squamous Cell Carcinoma

Squamous cell carcinoma is the second most common skin cancer in the United States. It arises from the epidermal prickle or squamous cells. Again, sun-exposure is a primary etiologic factor. The disease is at least twice as common in the southern as in the northern United States. Squamous cell carcinomas can also occur in other types of traumatized skin, such as postthermal burn, discoid lupus erythematosus, stasis dermatitis, chronic radiodermatitis, or almost any long-standing chronic irritation of the skin. In American blacks, antecedent trauma is more of a factor than sunlight because of inherent pigmenary protection.

Squamous cell carcinomas commonly arise from existing actinic or solar keratoses. Such malignant degeneration may be slow or rapid. The squamous cell carcinoma has a variable clinical picture, and may underlie a cutaneous horn or appear as a small, firm, red papule. Larger lesions often have a red raised border with central horn formation (Fig 7). The differential diagnosis between such a squamous cell carcinoma and a keratoacanthoma is often impossible to make definitively on either clinical or histologic grounds. In such cases one should interpret the lesion as a squamous cell carcinoma, here the more aggressive tumor. The keratoacanthoma, although sometimes resolving spontaneously, has also been known to cause extensive local destruction and resist conservative treatment. Squamous cell carcinomas grow relentlessly if unchecked. The rate of metastasis appears to be related to the size of the primary lesion. Metastasis is less common when the squamous cell carcinoma arises from a premalignant solar keratosis.

Malignant Melanoma

Malignant melanomas are probably the best known and most feared of all skin cancers, and rightfully so. Although
commonly mistaken for benign pigmented nevi (Fig 8), they can quickly grow deep and metastasize early. Malignant melanomas are tumors derived from melanocytes of the epidermis or mucous membranes, or from the conjunctiva or choroid. The incidence of malignant melanoma in North America is between 2 and 5 per 100,000, but seems to be increasing throughout the world at a rate which cannot be explained only by better or earlier diagnosis. Among the numerous factors proposed in the etiology of malignant melanoma, sunlight and ultraviolet radiation appear to be directly related to its incidence. Trauma and nevi that existed before malignant transformation are additional factors. The typical clinical picture of the malignant melanoma is irregularity in size, shape, color, and contour. Most lesions are some shade of black, brown, or grey, but nonpigmented tumors can occur (Fig 9).

The prognosis depends upon the size and shape of the lesion (larger lesions with raised surfaces have a worse prognosis), the histologic type (the deeper the penetration into the underlying skin, the worse the prognosis), the level of invasion, the anatomic site (face, head, and neck is better than the trunk), sex and age (women under 50 have a higher survival rate), and lymph node involvement (negative is better than positive).

Treatment of Skin Cancers
The treatment of skin cancers should be tailored to the patient, the type of cancer, its size, and anatomic location. Small basal cell and squamous cell carcinomas can be removed by cold steel excision surgery or by electrodessication and curettage. The latter provides an excellent result if the size and shape are correct (Fig 10). Both techniques have a 95% cure rate for primary lesions. Recurrences or unusual sites may indicate alternative forms of treatment including irradiation, chemosurgery, or even immunotherapy.

The first and most effective treatment for primary malignant melanoma is surgical excision with wide margins extending to the deep fascia. The site is then grafted. Prophylactic lymph node dissection is not usually performed unless there is deep invasion.

Preventive Therapy
No one wants wrinkles, crow's feet, brown spots, dilated blood vessels, discolorations, loose skin, premalignant or malignant skin lesions. How can we help patients avoid these problems? The answer is protection from sunlight. I often ask female patients to compare the skin of their upper chest and neck to the skin of their sun-spared breasts. The difference is dramatic and reveals that “aging” skin is more closely related to solar damage than to chronologic age. Sunlight damage is like other radiation damage, in that doses are cumulative and the effects incremental. You cannot reverse skin-aging once it occurs. Cream moisturizers and emollients when applied to previously hydrated skin provide temporary relief, but the real answer is avoidance of sunlight.

Two thirds of the most injurious ultraviolet light occurs between 10 AM and 2 PM. Remember that heat and visible sunlight have little to do with this type of radiation. Thus, skin-protective agents are necessary on overcast days as well as sunny days.

Baby oil and other so-called suntan lotions provide no real
protection. Many popular items are valueless as sunscreens. The two basic types of skin-protective agents are chemical sunscreens and physical blockers.

The physical blockers or "sun shades" provide a barrier to sunlight penetration by deflecting and scattering light. Their major advantage is that they protect over a wide range of ultraviolet light. Their disadvantage is chiefly cosmetic, since their opacity makes them visible and often chalky or mask-like. Some women, however, like this aspect of the sun shades, and use them as a foundation under their makeup. Physical blockers include zinc oxide paste, talc, titanium dioxide, kaolin, ferric oxide (which also provides a skin tint), red veterinary petrolatum, and bentonite.

The chemical sunscreens absorb light of a particular wavelength. Although they are usually more acceptable cosmetically, they have several disadvantages: They may cause sensitization or primary irritation, and most screen out limited wavelengths, necessitating precise matching of the sunscreen to the patient. For example, the benzophenones absorb most effectively at the shorter wavelengths used in germicidal radiation in many operating rooms and at longer wavelengths nearer to the visible spectrum, thus protecting patients with porphyria more effectively.

Para-aminobenzoic acid (PABA) is one of the best chemical photoprotective agents within the erythemogenic spectrum. A concentration of 5% para-aminobenzoic acid is effective. Vehicles vary from 55% alcohol (PreSun) to 5% alcohol with a para-aminobenzoic acid ester (Eclipse) to a 20% aloe emollient cream (Pabatection). Although para-aminobenzoic acid can stain clothes a light yellowish-brown, it can usually be washed out. Other effective chemical agents include digalloyl trioleate (as in SunStick), the cinnamates, the anthranilates, the pyrones, and the salicylates. Some clinically effective sunscreens combine physical and chemical blockers with actions that may be complementary or synergistic (Solar Cream with para-aminobenzoic acid and titanium dioxide). Others combine chemical sunscreens with different absorptive qualities (Maxafil with cinnamate and anthranilates).

Patients must remember that hats and clothing are not sufficient protection. Sunscreens should be reapplied if exposure to sunlight is prolonged, and especially after sweating or swimming.

Old age cannot be avoided, but old-looking skin can be. Making the patient more aware of his or her skin and how to protect it should be the job of every physician. Your patient will thank you for it!