CLINICAL MANAGEMENT extra

Skin Cancer: More than Skin Deep

CME
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This continuing education activity will expire for physicians on December 21, 2010.

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PURPOSE:

To provide the wound care practitioner with an updated overview of the epidemiology, clinical presentation, treatment, and prevention of skin cancer.

TARGET AUDIENCE:

This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES:

After participating in this educational activity, the participant should be better able to:

1. Describe the epidemiology, pathophysiology, and clinical presentation of skin cancer.

2. Discuss the diagnosis, management, and prevention of skin cancer.

ADV SKIN WOUND CARE 2009;22:574-80; quiz 581-2.

Skin cancer is the most common carcinoma in the United States, affecting millions.[1] Statistics show that 1 in 5 Americans and 1 in 3 whites will develop skin cancer in their lifetime; 1 person dies of melanoma almost every hour.[1] It is also one of the most preventable cancers. Protecting the skin from UV light exposure and early detection through increased public awareness and skin screening are paramount to guarding against this disease.

The deadly link between UV exposure and skin cancer is well established.[1–3] The epidemic rate of new skin cancer cases makes it seem nearly endemic. Healthcare providers must increase their knowledge and familiarity with the epidemiology, clinical presentation, and treatment of skin cancer, as well as prevention and education.

EPIDEMIOLOGY

The incidence of skin cancer crosses every socioeconomic group and demographic region, includes every ethnicity, and covers the entire life span. The American Cancer Society (ACS) predicted an excess of 1.1 million new cases of cutaneous malignancy ending in 11,200 deaths in 2008.[1] Actual figures are not available because reporting nonmelanoma skin cancer to the cancer registry is not required. The ACS predicted 62,480 new melanoma cases diagnosed in the United States in 2008, resulting in 8420 deaths.[1] The cost of treating skin cancer in the United States is estimated to be more than $2.9 billion annually.[3]

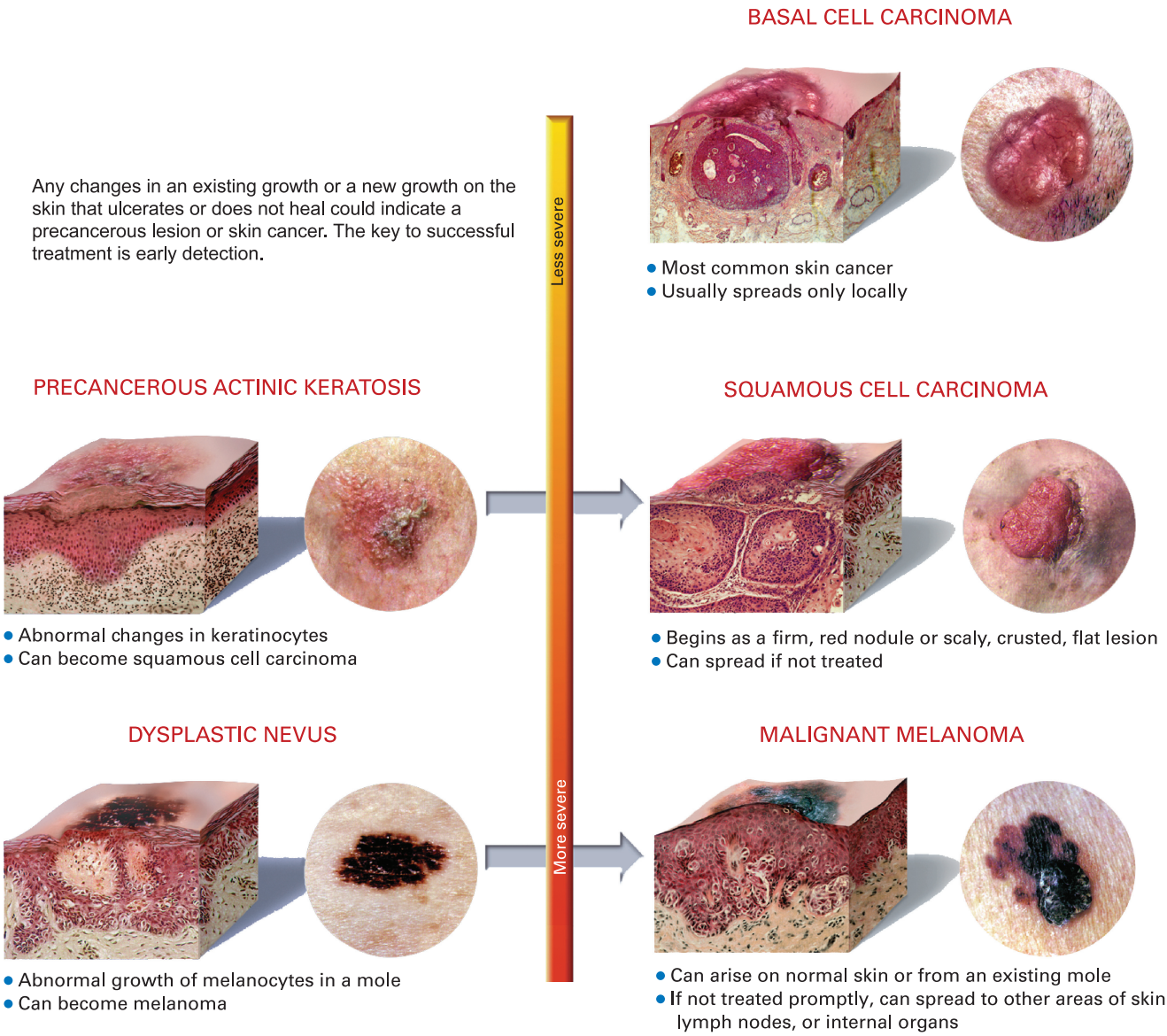
Skin cancer is also a growing global problem. The Netherlands is predicting an 80% increase in the total number of skin cancer patients by the year 2015.[4] Canadian researchers have

demonstrated an overall lifetime risk for diagnosis of a nonmelamoma skin cancer increased 2 to 3 times in the past 4 decades.[5] Australia has the highest incidence of skin cancer in the world.[6] By 2011, it is projected that melanoma will overtake lung cancer as the third highest cancer incidence for Australian men.[7] The rise in global incidence will undoubtedly put a significant strain on every national healthcare system.

PATHOPHYSIOLOGY

The UV radiation in sunlight induces all 3 major forms of skin neoplasm (Figure 1). UV radiation is composed of 2 main types of rays: ultraviolet A (UVA) and ultraviolet B (UVB). UVA rays pass deeper into the skin and UVB rays are more likely to cause sunburn.[8,9] UVB is associated with direct damage to DNA, whereas UVA is associated with indirect damage

Figure 1.
TYPES OF SKIN CANCER



mediated by free radical formation and damage to cellular membranes.[10]

Researchers have suggested an association between UV radiation-induced immune suppression and carcinogenesis.[3] When UV radiation penetrates the skin, much of its energy is absorbed by the DNA of epidermal keratinocytes. Researchers hypothesized that DNA is the photoreceptor in the skin and that UV-induced pyrimidine dimer formation is the initial molecular step that leads to immune suppression.[3] The mechanics of UV-induced damage progressing to skin cancer is detailed and complex. Mutation of the p53 tumor suppressor genes and production of reactive oxygen species are only 2 of the many processes cited in the literature as implicating factors that lead to the development of cancerous cells.[8]

SKIN FINDINGS

Actinic keratoses (AK) are precancerous or precursor lesions to 10% of squamous cell carcinomas (SCCs).[11] Clinical features include single or multiple, dry scaly adherent lesions on habitually sun-exposed skin. Lesions begin as barely perceivable rough spots of skin, more often felt than seen. The early lesions feel like sandpaper. Later lesions become erythematous, scaly plaques that may enlarge to more than a centimeter. Often, these lesions flake off or are exfoliated by normal daily activities such as toweling off after a shower or shaving, only to recur again. Scaly lesions on sun-exposed skin that do not respond to moisturizers, itch, or bleed with minimal provocation need medical attention. The length of time for an AK (Figure 2) to progress to an SCC can be as early as 24.6 months.[8]

Figure 2.
ACTINIC KERATOSIS

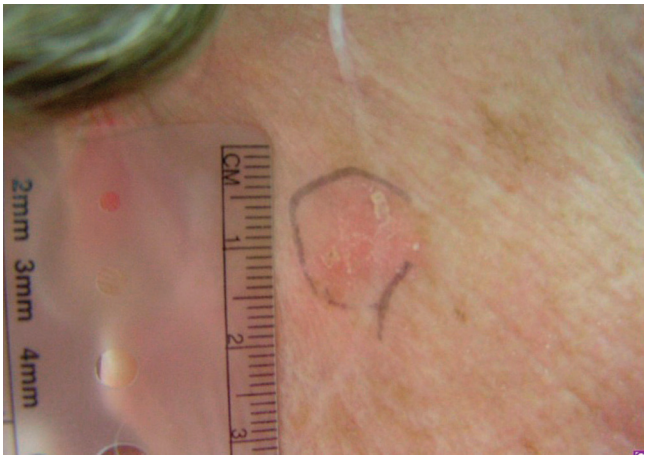


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Figure 3.
BASAL CELL CARCINOMA

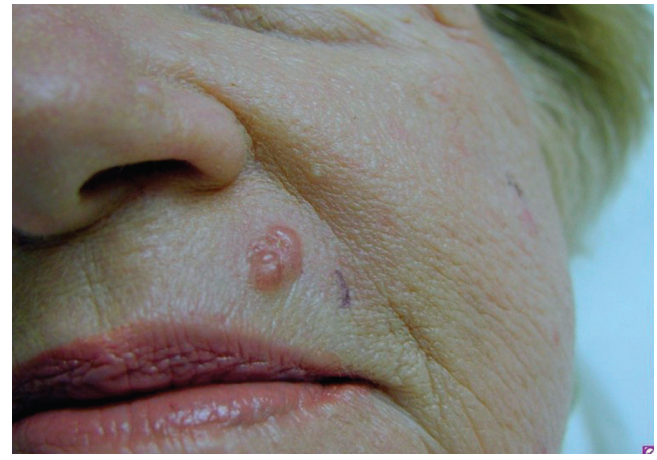


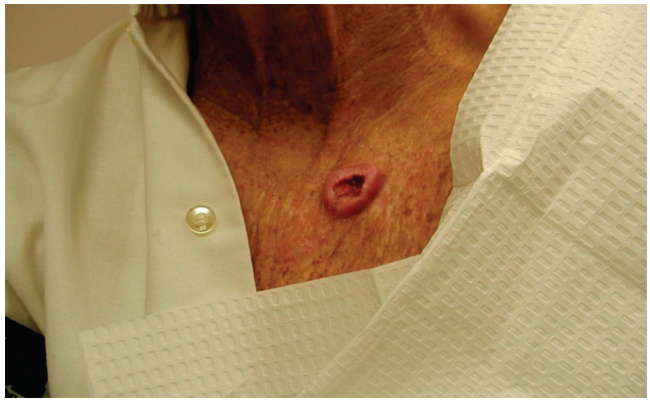
Photo courtesy of Gulf Coast Dermatology.

Basal cell carcinoma (BCC) represents 65% to 75% of all skin cancers and most commonly occur on sun-exposed parts of the face, ears, scalp, shoulders, and back.[12] BCC develop from exposure to both UVA and UVB.[13] DNA mutations secondary to UV radiation is the primary etiology for the development of both BCC and SCC.[5] Specifically, BCCs are believed to arise from basal keratinocyte cells of the epidermis and adnexal structures.[14] Clinical features include pearly translucent flesh-colored papules or nodules with superficial telangiectasias (broken blood vessels). More active lesions may have rolled edges or ulcerated centers.[11]

The course of BCC is unpredictable. BCCs tend not to metastasize but may become locally invasive if left untreated. BCC can also occur at sites of previous trauma (scars), thermal burns, and injury.[14] The incidence for recurrent BCC in patients with prior BCC is 44% during the consecutive 3 years.[14] One study speculated that the risk for a new neoplasm largely depends on the number of prior skin tumors. These findings strongly support the need for careful and frequent follow-up[15] (Figure 3).

Squamous cell carcinomas represent 30% to 65% of all cutaneous malignancies.[12] SCCs are most attributable to UVB exposure.[13] Whereas BCCs appear associated more with intense short-term exposure, SCCs seem to be associated with cumulative exposure over time.[15] SCCs develop from epidermal squamous cells (keratinocytes). The spectrum of severity ranges from low-grade intraepidermal carcinoma (Bowen disease) to invasive SCC with the potential to metastasize.[11] Human papillomavirus (HPV) types 6, 11, 16, and 18 are among the most common HPV types associated with genital warts. Types 16 and 18 are high-risk viruses with oncogenic potential, which suggests that papillomavirus infection is a cause of anal cancer.[14]

Figure 4.
SQUAMOUS CELL CARCINOMA



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Renal-transplant recipients receiving immune-suppressing antirejection medication have a 253-fold increased risk of SCC.[14]

Clinical features include crusted papules and plaques that may become indurated, nodular, or ulcerated. SCC (Figure 4) may arise in chronic wounds, scars, and leg ulcers.[8,11] Recurrent SCC development within 3 years is 18%, a 10-fold higher incidence compared with initial SCC diagnosis in the general population.[15] Malignant melanoma (MM) represents the most serious of all cutaneous malignancies. Approximately 65% to 90% are caused by UV exposure, predominantly UVA.[9,13] Roughly 10% of all melanoma cases are strictly hereditary.[2]

Melanomas originate from melanocytes, which are melanin-producing cells contained within the basal layer of the epidermis. UV exposure appears to be the greatest inducer of melanoma through many mechanisms, including suppression of the immune system of the skin, induction of melanocyte cell division, free radical production, and damage of melanocyte DNA.

The ABCD rule outlines the clinical presentation and warning signals of the most common type of melanoma. ''A'' is for asymmetry (one-half of the mole does not match the other half); ''B'' is for border irregularity (the edges are ragged, notched, or blurred); ''C'' is for color (the pigmentation is not uniform, with variable degrees of tan, brown, or black); ''D'' is for diameter greater than 6 mm (about the size of a pencil eraser). Some clinicians now include ''E'' regarding evolution, elevation, or enlargement of a lesion.[1,11] Table 1 illustrates these guidelines.

A favorable prognosis of MM (Figure 5) is entirely attributable to early detection. Prognosis is directly related to the size and the depth of the tumor invasion of the skin (Breslow thickness).[11,14] Even with a rising morbidity rate, there has been an encouraging improvement in the overall survival rate.[11] The 5- and 10-year relative survival rates for persons with melanoma are 91% and 89%, respectively. About 80% of melanomas are diagnosed at a localized stage yielding a 5-year survival rate of 99%. The 5-year survival rates for regional and metastatic stage diseases are 65% and 15%, respectively.[1]

Table 1.
GUIDELINES FOR EVALUATING PIGMENTED LESIONS[1]

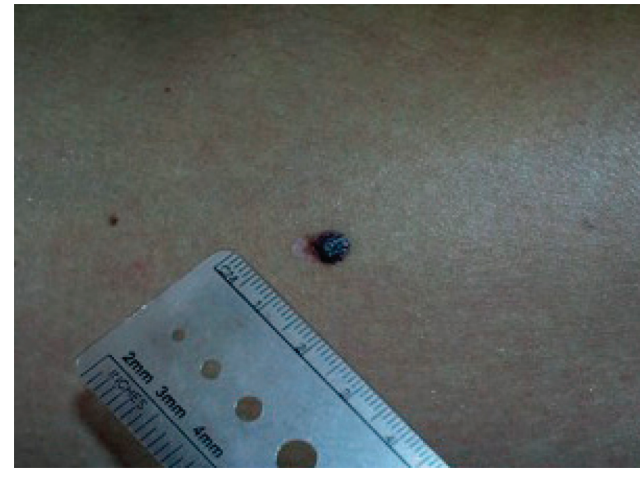
A = Asymmetry: one-half is different than the other half
B = Border: border is blurred, notched, or irregular
C = Color variation: pigment is not uniform
D = Diameter: size is >6 mm
E = Evolution of a lesion or new onset of a lesion
Additional red flags: color spreads into surrounding skin, loss of pigment, itching, tenderness, and bleeding without provocation.

DIAGNOSIS

The standard for diagnosis is a biopsy by shave, punch, or excision. This simple procedure is a skill all primary care providers can master and saves valuable time when working up a patient for skin cancer. Biopsy is indicated in all skin lesions that are suspected of being neoplasms. Treatment is contingent upon histopathology.

The process used to determine if the cancer has spread within the skin or to other parts of the body is called staging. For nonmelanoma skin cancers in Stage 0, the abnormal cells are confined to the epidermis, and it is said to be in situ. A carcinoma measuring less than 2 cm or greater than 2 cm is considered Stages I and II, respectively. In Stage III, it involves

Figure 5.
MALIGNANT MELANOMA



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the structures below the skin such as muscle, bone, or cartilage or nearby lymph nodes and in Stage IV has metastasized.[14]

Both the Clark and Breslow systems are commonly used in the staging of MM. The Clark stage, ranging from I to IV, is based on the tissue level of invasion. The absolute depth may differ, depending on the region of the body involved.[14]

Technological advances in diagnostic equipment may soon give rise to new noninvasive tools for early skin cancer detection.[16] High-frequency ultrasound and devices that use infrared light are being evaluated for clinical application.

MANAGEMENT

The treatment modality decision is multifaceted. Ideally, patients should be managed by a dermatology specialist. Cryosurgical destruction with liquid nitrogen, topical application of chemotherapy medications, photodynamic therapy, curettage with electrodessication, and surgical excision are all useful with successful outcomes for nonmelanoma cancers. Mohs surgery involves removing tumors in repetitious stages, processing the tissue in ''slices,'' and determining microscopically exactly where the tumor margins meet the healthy skin. This surgical method is greater than 99% accurate and preserves a maximal amount of healthy skin, resulting in a smaller surgical scar. Radiation therapy is an alternative only when disfigurement may be a problem with surgical excision or when a patient is a poor surgical candidate.[11,17]

Melanoma management is contingent upon lesion thickness, depth, and invasion of lymph nodes or distant organs. Lymphoscintigraphy is a contrast-medium study method of determining lymphatic involvement, which might require lymph node dissection. Surgical excision generally requires wide margins. Multiple medical disciplines may be necessary to fully manage patients including dermatologists, surgeons, and oncologists.[14]

RISK FACTORS

Researchers suggested that more than half of a person's lifetime sun damage occurs before adulthood.[18] Childhood is the most important time for developing nevi (moles), an important risk factor for skin cancer. There is some evidence that sun exposure in childhood heightens the risk of melanoma by increasing the number of nevi. One blistering sunburn in childhood more than doubles the chance for developing melanoma later in life.[1]

It is estimated that less than one-third of children and adolescents comply with recommended sun protection methods.[18] Among adults in the United States, the most commonly reported skin cancer risk behaviors include infrequent application of sunscreen and not wearing sun-protective clothing. Other risks involve youth, residing in the Midwest, male, non-Hispanic white, less education, smoking, fair skin, and sun sensitivity.[19] One study suggested that increased time spent outdoors participating in sports may have a direct correlation to development of skin cancer due to greater UV exposure, inadequate sun protection efforts, and exercise-induced immunosuppression (Table 2).[20]

PREVENTION

The best prevention is strict avoidance of all UV exposure, which is not realistic. Behaviors that reduce skin cancer risk include limiting or minimizing exposure to the sun during midday hours, wearing protective clothing, and using sunscreen.

Not all sunscreens are created equal. The US Food and Drug Administration (FDA) considers sunscreen products to be over-the-counter drugs; therefore, no standard testing is established. The US sunscreen industry has been waiting 30 years for suggestions regarding product labeling. Most companies use terms such as ''UVA/UVB protection'' or ''broad-spectrum protection'' based on the presence of certain ingredients that offer protection from UVA and UVB. The sun protection factor (SPF) is a laboratory measure of the effectiveness of sunscreen from UVB. The SPF reflects the amount of time a person can spend in the sunlight before receiving a sunburn relative to a person in the sunlight without sunscreen. This is an imperfect measure because, to date, there is no way to quantify how effective a product is in shielding UVA. In 2007, the FDA published a new rating system for labeling products with specific regard to UVA protection. This 4-star method consists of 4 progressive categories, denoted as low (1 star) to high (4 stars).[10]

Active ingredients should be considered when choosing a sunscreen. Before sun exposure, remind patients to select a product that contains the highest allowable percentage of zinc oxide (25%) and titanium dioxide (25%). Both do not undergo significant chemical change or photodegradation with exposure to UV light. Avobenzone (3%) is the only truly effective UVA absorber available and offers the greatest photostability.[10]

Recently, there has been increased concern regarding synthetic compounds and carcinogenic effect. Further study

Table 2.
RISK FACTORS FOR MM[20]

- Fair skin, light eyes
- Many freckles and >50 moles on the skin
- Severe, blistering sunburns as a child or adult
- Family history of melanoma
- Previous melanoma
- Noncancerous, unusual-looking moles (dysplastic nevi)
- Exposure to UV radiation from tanning salons and tanning beds
- A weakened immune system

is needed. Some researchers suggested that as UV filter ingredients absorb into deeper layers of the skin, superficial layers are left vulnerable. Sunscreen makers are developing products that stay on the surface, with minimal absorption and possibly contain antioxidants that can stabilize free radicals.[10]

Investigators suggested that eating green leafy vegetables may help prevent subsequent SCCs among patients with SCC history. Consumption of unmodified dairy products (ie, whole milk, cheese, and yogurt) may increase the risk of SCC in at-risk patients.[21] One recent small study suggested drinking regular tea and an inverse association with skin carcinogenesis.[22] Similar effects are suspected of other botanical agents including *Ginkgo biloba*, vitamins E and C, carotenoids, and selenium.[23] A promising study demonstrated strong evidence to support a role for omega-3 fatty acids (FAs) in the prevention of nonmelanoma skin cancer. Omega-3 FAs have been shown to modulate a number of cytokines and prostaglandins that mediate inflammatory and immune responses, factors implicated in the development of skin cancers in UV-irradiated skin.[24]

EDUCATION

Numerous national awareness campaigns are currently in place to address this obvious crisis. Research findings suggested greater need for cooperation between media and advocacy groups for increased public awareness.[25] Since its inception in 1985, the American Academy of Dermatology National Melanoma/Skin Cancer Screening Program has strived to enhance early detection of cutaneous MM by providing nationwide skin cancer education campaigns in combination with free skin cancer screenings. A recent study showed increased likelihood of early MM detection by clinicians through focused visual inspection. Clinicians can be taught to have a heightened awareness to patients with significantly higher risk factors: history of melanoma, male, age older than 50 years, a changing mole, and no established relationship with a dermatologist.[26]

A key determinant of skin cancer in adulthood is the exposure to UV as a child. Sun protection messages should be linked with other health promotion messages targeting children.[27] The authors of 1 study suggested that children between the ages of 5 and 9 years are more receptive to intervention and change than older children and therefore more apt to have a positive attitude regarding sun exposure and behavior.[6] Children should be taught the correct use of sunscreen. Sunscreen should be applied to all exposed skin at least 20 minutes before going into the sun, even if it is cloudy outside, and needs to be reapplied every 2 to 3 hours or more frequently if swimming or exercising. Use at least 1 oz per application, roughly equivalent to the volume of a shot glass. Everyone needs to wear a hat and sunglasses with 99% to 100% UVA absorption. Patients should be instructed to avoid exposure between the hours of 10 AM and 4 PM when the sun is the strongest, wear sun-protective clothing, and seek shade whenever possible (Table 3).

Table 3.
SUN PROTECTION STRATEGIES[18]

- Choose a sunscreen that contains at least 2 of these ingredients:
  - Titanium dioxide
  - Zinc oxide
  - Avobenzone
- Choose a sunscreen with an SPF 30 or higher
- Apply sunscreen liberally at least 20 min before going out, even on overcast days
- Reapply sunscreen every 2–3 h or sooner if swimming or sweating
- Wear sun-protective clothing and UVA-blocking sunglasses
- Avoid heavy exposure between 10 am and 4 pm
- Seek shade whenever possible

Children and teenagers need to know that there is no such thing as a safe tan. Teenagers and women are considerably more likely to visit tanning salons, some logging more than 20 hours of exposure per year.[28] Tanning operations are poorly regulated, and existing regulations and recommendations are frequently ignored. Ninety-five percent of tanning salon customers exceed the recommended limits for UV exposure, and one-third of tanners begin tanning at the maximum dose recommended for maintenance.[28]

PROMOTING PREVENTION

Skin cancer is a worldwide epidemic. Millions of new cases are diagnosed each year in the United States alone. Primary prevention can be strengthened through augmenting the prevalence of sun-safe behaviors, awareness, education, and attitudes. Secondary prevention entails early detection of skin cancer when it can be most readily cured. Although specialists in dermatology provide effective screening for skin cancer, they encounter a smaller segment of the population than do primary care providers. Clinicians are in an optimal position to provide skin cancer prevention, screening, and detection services. ●

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