

[Primary Care]

Ultraviolet Light and Skin Cancer in Athletes

Shannon C. Harrison, FACD,* and Wilma F. Bergfeld, MD

The incidence of melanoma and nonmelanoma skin cancers is increasing worldwide. Ultraviolet light exposure is the most important risk factor for cutaneous melanoma and nonmelanoma skin cancers. Nonmelanoma skin cancer includes basal cell carcinoma and squamous cell carcinoma. Constitutive skin color and genetic factors, as well as immunological factors, play a role in the development of skin cancer. Ultraviolet light also causes sunburn and photoaging damage to the skin.

Keywords: skin cancer; melanoma; ultraviolet light; athletes

Vitamin D is an essential vitamin for bone health.³³ Vitamin D is obtained from adequate dietary intake or through exposure to the sun or ultraviolet (UV) light.³³ Although exposure to the sun or UV light may help maintain vitamin D levels, exposure may result in a higher risk for developing skin cancers (Table 1). This is especially true of summer and winter outdoor athletes, who are exposed to higher amounts of UV light due to training and competition schedules. Sun protection strategies, including sunscreens and sun protective clothing, may help to reduce this risk for athletes.

EPIDEMIOLOGY OF SKIN CANCER

In the United States, the lifetime risk for an individual to develop a skin cancer is approximately 20%.²⁸ Nonmelanoma skin cancer (NMSC) is the most common type of cancer in the Caucasian population.²² In the United States, approximately 1.3 million cases of NMSC are diagnosed annually.²² Of this number, 75% are cases of basal cell carcinoma (BCC) and the remaining 25% are cases of squamous cell carcinoma (SCC).²² The incidence of NMSC is much higher than that of melanoma, the former being 18 to 20 times more frequent.¹²

In Caucasian populations, cutaneous melanoma is increasing the fastest.¹² The incidence varies around the world, depending on location, but in the United States, the annual rate is approximately 14 per 100 000.¹² In Queensland, Australia, the incidence of melanoma is as high as 40 to 50 per 100 000 per year.¹² As of 2007, the lifetime risk to develop an invasive melanoma is 1 in 63 in the United States.²⁷

Table 1. Common sun-induced skin lesions potentially seen in athletes.

Solar keratosis
Basal cell skin cancer
Bowen disease (squamous cell skin cancer in situ)
Squamous cell skin cancer
Cutaneous melanoma

EPIDEMIOLOGY OF SKIN CANCER IN ATHLETES

Studies conducted within athletic populations have found that certain sports are associated with increased risk for skin cancers. These studies were limited and did not investigate the relationship between the findings and morbidity/mortality.

In a study by Ambras-Rudolph et al,² the risk of cutaneous melanoma and nonmelanoma skin cancer was found to be increased in marathon runners. A study by Dozier et al⁶ found surfing to be associated with a higher incidence of BCC. According to Moehrle,¹⁷ mountain climbers can also demonstrate an increased prevalence of skin cancer and actinic keratoses. Therefore, athletes may be at increased risk of skin cancers because of their outdoor training and competition, although data are lacking.

From the Department of Dermatology, Cleveland Clinic Foundation, Cleveland, Ohio

*Address correspondence to Wilma F. Bergfeld, MD, Cleveland Clinic Foundation, Department of Dermatology/A61, 9500 Euclid Avenue, Cleveland, OH 44195 (e-mail: bergfew@ccf.org).

No potential conflict of interest declared.

DOI: 10.1177/1941738109338923

© 2009 The Author(s)



Figure 1. An ulcerated basal cell skin cancer.

NONMELANOMA SKIN CANCERS

The main types of nonmelanoma skin cancers are basal cell skin cancer (or BCC) and squamous cell skin cancer (or SCC). Basal cell skin cancer is the most common type of NMSC²² and it is more common in men than in women.³ Researchers believe that BCCs originate from cells in the basal layer of the epidermis or from cells in the outer root sheath of the hair follicle.³ UV light is the major risk factor for BCC, but fair skin, tendency to freckle, inability to tan, family history of skin cancer, a suppressed immune system, previous NMSC history, sun bed use, and certain genetic syndromes are also risk factors.³

BCC presents as a painless, slow-growing plaque or lump or a nonhealing sore or ulcer. Clinically, BCCs are pearly whitish-pink plaques, papules, or nodules with surface telangiectasiae.³ BCCs can ulcerate and bleed (Figure 1). Some BCCs may form an ulcer with a pearly rolled edge, whereas other BCCs may contain grayish-blue areas of pigment that can be seen by the naked eye. BCCs typically occur on the sun-exposed skin of the head and neck, but they may present anywhere on the trunk and limbs.³ There are different types of basal cell skin cancer, based on their morphology and histology: superficial, nodular, morpheic (sclerosing), pigmented, cystic, metatypical (basosquamous), and ulcerated.^{3,30} BCCs slowly grow over time and thus destroy the surrounding tissues. BCCs rarely metastasize or spread but are locally invasive.³

Treatment for BCCs depends on several factors, including the site, size, type, and histology of BCC, in addition to patient-based factors, such as age and health. There are surgical and nonsurgical options. Treatment options for BCCs include surgical excision, Mohs micrographic surgery, photodynamic therapy, curettage and cautery, cryotherapy with liquid nitrogen, radiotherapy, and topical immunomodulating therapies.²² Biopsy of the lesion by a physician to confirm histology may be done before surgically excising it or treating it with a destructive or immunomodulatory therapy.²² Referral to a dermatologist may be needed if the health care provider is not comfortable biopsying or treating the lesion.

SCC is the second-most common skin cancer in the United States.⁴ It arises from the keratinocyte cells that make up the



Figure 2. Squamous cell skin cancer of the lip.

epidermis. SCCs present as atypical cells throughout the full thickness of the epidermis and as abnormal cells invading into the underlying tissue (ie, the dermis).^{4,9} SCC can present as a nonhealing sore or lump. SCCs are red or pink scaly plaques, papules, nodules, or ulcers. They occur on body parts chronically exposed to sun, but they have a predilection for the head, ears, and back of hands⁹ (Figure 2). SCCs enlarge faster than BCCs, and some can grow quite rapidly over weeks. SCCs can spread, metastasize, and even cause death.⁹ UV light exposure is the most significant risk factor for developing an SCC. Other risk factors include fair skin, tendency to freckle, inability to tan, multiple sunburns, family history of skin cancer, chronic burns, ulcers or sinuses, arsenic use, a suppressed immune system, the human papilloma wart virus, previous NMSC history, and previous radiation.⁹

Actinic keratoses are considered part of the SCC spectrum³¹ and are focal areas within the epidermis of atypical keratinocyte cells but not full-thickness atypia of the epidermis. Clinically, actinic keratoses are painless, rough, pink-red hyperkeratotic macules and papules on any sun-exposed area of the skin (Figure 3). Some actinic keratoses may progress to SCCs.³¹ Bowen's disease or SCC in situ is full-thickness keratinocyte atypia of the epidermis without dermal invasion.⁵ Bowen's disease typically presents as red, scaly, well-defined plaque on sun-exposed skin.⁵

Surgical excision or Mohs micrographic surgery is the gold standard treatment for SCCs.⁹ Surgical excision is also an option for Bowen's disease at certain sites. Destructive treatments, such as cryotherapy with liquid nitrogen, curettage and cautery, photodynamic therapy, and topical immunomodulating and chemotherapeutic agents, can be used for actinic keratoses and Bowen disease in certain body sites.³¹ The physician and patient guide the decision on which treatment option is appropriate.

CUTANEOUS MELANOMA

Cutaneous melanoma arises from the pigment or melanocyte cells in the skin,⁸ from a preexisting mole, or from normal skin. It can affect a person at any age, and it is the most serious skin

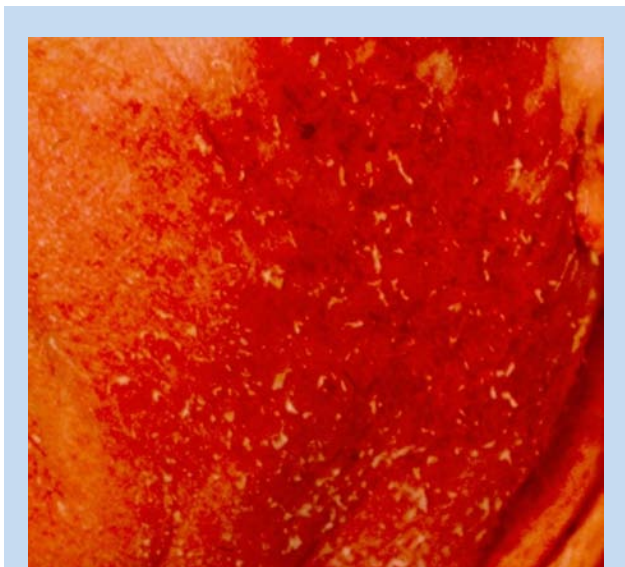


Figure 3. Actinic keratoses of the cheek.

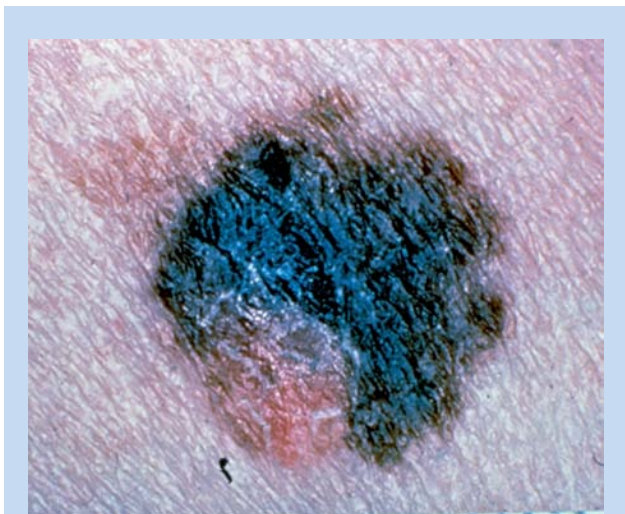


Figure 4. Cutaneous melanoma.

cancer. Melanoma can metastasize and cause death.⁸ Apart from UV exposure, further risk factors for melanoma include red hair, blue eyes, fair skin, inability to tan, family history of melanoma or dysplastic nevus syndrome, previous NMSC, a suppressed immune system, having 50 to 100 moles, and a history of multiple blistering sunburns.⁸

Cutaneous melanoma presents as an enlarging brown-black plaque or nodule on the skin (Figure 4). Rarely, melanoma can be nonpigmented and pink-red in color. Lesions are usually painless but can ulcerate and bleed. There are different types of melanoma, including superficial spreading melanoma, as well as nodular, acral lentiginous (affecting hands, feet, and

nails), and lentigo maligna melanoma.⁸ Cutaneous melanoma can occur at any body site, but it favors the trunk in men and the legs in women.¹²

Suspicious changes in pigmented lesions are denoted by the mnemonic *ABCDE*: A, asymmetry; B, border; C, color; D, diameter; E, evolution.²⁹ Asymmetry can be determined by drawing imaginary lines down and across the middle of the lesion in 2 axes, to look for a difference or asymmetry between the halves, whereas *border* refers to a lesion whose border is uneven or irregular.²⁹ Other concerning features include multiple or uneven colors (ie, darker or even pink-red), a lesion with a 6-mm diameter (or greater), or an evolving lesion.²⁹ Any new pigmented lesion or any changing pigmented lesion should be promptly examined by a physician.

Melanomas are treated with surgical excision.⁸ Typically, the lesion undergoes an excisional biopsy with a small margin of normal skin (1-2 mm) surrounding the lesion of concern and the histology examined by a pathologist. The most important factor of melanoma is the thickness of the tumor, which is read by the pathologist. If the diagnosis of melanoma is confirmed, then a second surgery is performed, with a wider margin of normal skin removed around the lesion, depending on the thickness of the melanoma. For example, if the melanoma is 1-mm thick, 1 cm of tissue would be excised around the excisional biopsy scar.⁸

Once a person has had skin cancer, he or she should be followed at regular intervals by a physician or health care provider for full skin surveillance examinations and examination of the scar site, lymph nodes, liver, and spleen. Strict sun protection and sunscreen are essential.

UV LIGHT AND SKIN CANCER

UV exposure is the most significant risk factor for the development of cutaneous melanoma and nonmelanoma skin cancers.²⁷ UV light affects the skin cells at a genetic level, causing characteristic mutations in the cellular DNA.²⁷ The dosing, total dose, and wavelength of UV light are all important factors in the development of skin cancers. Both UVB (290-320 nm) and UVA (320-400 nm) affect the skin. UVB is important in the development of cutaneous melanoma and NMSC, whereas UVA is thought to be an important co-factor in melanoma development and photoaging skin changes.²⁷ UV exposure also causes immune suppression in the skin, which can lower the immune system defenses against detecting and destroying any mutated or sun-damaged cells, thus allowing cancers to develop.¹⁷

Geographic location is important in determining the amount of UV exposure. There is a direct association between latitude and incidence of nonmelanoma skin cancers; for instance, Australia has the highest skin cancer rates.^{20,27}

The association with latitude and melanoma is less clear.²⁷ UV light dosage increases as the distance from the equator decreases.²⁷ Skin cancers develop most frequently at body sites of highest average UV exposure (eg, head), whereas skin cancers are rarely seen in sun-protected sites (eg, buttocks),

thus emphasizing the importance of the total dose of UV exposure in skin cancer development.²⁷

Artificial UV rays (tanning salons) also increase the dose of UV light exposure and the risk of skin cancer.¹⁵ Some sun beds have an UV intensity 10 to 15 times higher than that of the midday sun.¹⁵ Long-term cumulative sun exposure increases the risk of developing SCC.^{12,27} Meanwhile, intermittent and chronic cumulative sun exposure is believed to be a causative factor in BCC development.¹² Intermittent acute sun exposure early in life appears to be associated with increased risk of cutaneous melanoma²⁷; however, a study by Pfahlberg et al²³ demonstrated that having more than 5 sunburns at any time of life doubles the risk of melanoma.

UV Light, Athletes, and Skin Cancer

Some athletes are exposed to increased amounts of UV rays and are at increased risk of sunburn because of their training schedules and conditions. Summer sports are most often conducted during peak UV exposure hours (ie, in the middle of the day) with uniforms that do not provide adequate sun protection.¹⁷ During the Hawaiian Ironman Triathlon World Championships, the average UV exposure of 3 triathletes was approximately 20 standard erythema doses,^{17,18} with 1 dose equaling 100 J/m².¹⁷ A minimal erythema dose is the smallest amount of radiation exposure causing a defined skin erythema or sunburn 24 hours after exposure¹⁷—approximately, 200 to 300 doses of 100 J/m².^{17,18} Cyclists in the Tour de Suisse were exposed to approximately 8 times more than the minimal dose of UV needed to cause sunburn.^{19,26}

In winter sports (eg, skiing, snowboarding), direct radiation occurs from the sun, often at higher altitudes, and from reflection off the snow and ice, to exposed areas such as the face and hands.¹⁷ Rigel et al demonstrated that skiers without sunscreen at 11 000 feet begin to develop sunburn after only 6 minutes of UV exposure.¹²⁶ Outdoor athletes are frequently exposed to high levels of UV light and are at risk for sunburn and the potential future development of skin cancer—especially, athletes with lighter skin types.¹ Furthermore, coaches and trainers may be at increased risk because of similar exposure.

Factors such as sweating and friction during sports may increase the photosensitivity of the skin and, thus, the risk of sunburn.¹⁷ In addition, some medications (eg, acne antibiotics) can increase photosensitivity and the subsequent risk of sunburn.¹

Sun Protection, Athletes, and Skin Cancer

Athletes, coaches, and trainers should be educated about sun protection, including sunscreen, protective clothing, and behavioral factors. Regular daily sunscreen use may decrease the risk of actinic keratoses and SCC.²⁷ MacLennan et al¹⁶ has shown that use of sunscreen in childhood is associated with decreased counts of moles or nevi, suggesting that sun protection in childhood may reduce melanoma risk by reducing the total number of moles on the skin.²⁷

In the United States, sunscreens are regulated by the Food and Drug Administration as over-the-counter drugs.¹¹ Sunscreens are rated by a sun protection factor (SPF), which is a measure of UVB protection. SPF is the ratio of the minimal erythema dose of skin with sunscreen over the minimal erythema dose of unprotected skin.¹³ The maximum SPF is obtained only when the concentration of sunscreen is 2 mg/cm² (30 mL) for the entire skin surface.¹³ SPF of 55 and 75 are now available. The FDA has suggested that products with SPF 2-14 indicate low UVB sunburn protection; SPF 15-29, medium protection; SPF 30-50, high protection; and SPF 50+, the highest protection.¹¹ There is no SPF rating for UVA protection. However, the FDA has suggested a 4-star UVA protection rating (ie, low, medium, high, and highest).¹¹ Sunscreens should be generously applied and then reapplied every 2 to 4 hours or after sweating, water exposure, swimming, or towel drying.¹¹

Sunscreen ingredients can be classified as *physical* or *chemical* blockers, although the FDA has replaced these terms with *inorganic* and *organic*, respectively.¹¹ Inorganic sunscreens reflect, scatter, or absorb UV radiation and are typically thick and opaque.¹¹ They are broad-spectrum and protect against UVB, UVA, and visible light radiation.¹¹ Zinc oxide and titanium dioxide are inorganic UV filters, now available in micronized or microfine forms, which are cosmetically more acceptable.¹¹ In the UVA range protection, microfine zinc oxide is more effective than titanium dioxide.¹¹ Organic UVB filters (padimate O and octinoxate) are active against UVB radiation only. PABA, a common ingredient in sunscreens, is less commonly used now because of its potential for photoallergy.¹¹ Organic UVA filters (oxybenzone, dioxybenzone, and avobenzone) are active against UVA radiation only.¹¹ Helioplex (avobenzone and oxybenzone) is a new patented sunscreen technology that stabilizes sunscreen ingredients (see <http://www.neutrogena.com>) and provides broad-spectrum coverage (SPF 55). Anthelios SX (Mexoryl SX, 3%; octocrylene, 10%; avobenzone, 2%; and titanium dioxide, 5%; see <http://www.laroche-posay.us>) has FDA approval and provides broad-spectrum UV coverage (SPF 40).³² Athletes exposed to UV radiation should be encouraged to use a broad-spectrum water-resistant sunscreen or a very water-resistant sunscreen of at least SPF 30-50. A high-SPF lip balm is also recommended. Sweating, friction, and water immersion will decrease the effectiveness of sunscreens if not properly reapplied. Alcohol-based sunscreen sprays, gels, and lotions are not as heavy or greasy as creams and are better tolerated by athletes.¹ Hamant and Adams³⁰ surveyed collegiate athletes and found that 85% reported no sunscreen use in the previous week and only 6% used sunscreen at least 3 days of the week during the study. According to Price et al,²⁴ 68% of New Zealand snowboarders were unaware of any educational sun protection messages for winter sports, and at least 48% have experienced a sunburn while snowboarding or skiing (Figure 5).

The behavioral factors of sun protection include seeking shade and avoiding UV or sun exposure during the hottest part of the day, when UV radiation is highest (10 AM to

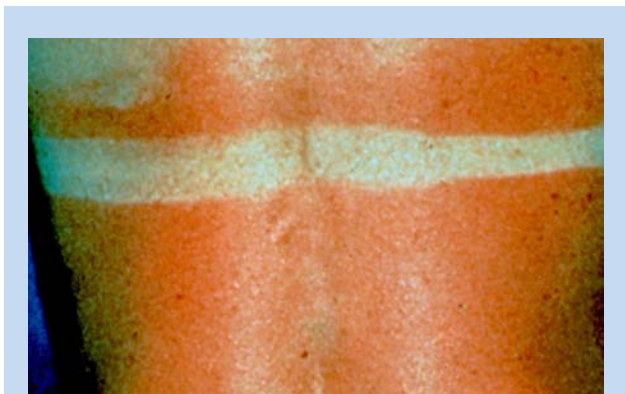


Figure 5. Sunburn on the back of a young woman.

3 PM).^{17,25,26} Ideally, training and competition should be held before or after these hours, in times of low UV exposure.¹⁷ Clothing can aid in sun protection. A photodetector can measure the UV protection factor (UPF) of clothing to determine the amount of UV radiation transmitted through fabric.¹³ A UPF of 40 to 50 has excellent UV protection, transmitting less than 2.6% of UV radiation, whereas a UPF of 25 to 39 has good UV protection.¹³ Normal summer clothing has a UPF of 10 or more.¹³ Thickly woven textiles that do not transmit light through the fabric are most protective.¹³ One hundred percent cotton or rayon fabrics are the worst UV absorbers, whereas 100% polyester is the best.¹³

Shrinkage increases the UPF because the fabric weave becomes tighter.¹³ Light-colored clothes scatter light, whereas dark-colored clothes absorb UV radiation.¹³ Most athletes train and compete in short-sleeve or sleeveless tops and shorts in summer, which does not provide adequate sun protection. Newer clothing lines with UV protection are available in long sleeves with cool and breathable fabrics. UV absorbers in laundry-washing detergents are also available and so reduce UV radiation penetration through the fabric.²⁵ Caps or hats should be used to protect the face, scalp, and neck from the sun. Sunglasses should be worn at all practicable times by athletes in summer and winter sports. Australia has mandatory standards for sunglasses: Only 5% of the luminous transmittance is allowed to be UVB radiation.²⁵ In the United States, compliance is voluntary, thus necessitating careful selection of polarized sunglasses with adequate UV protection.²⁵

CONCLUSION

A vitamin D deficiency may be associated with certain cancer types, cardiovascular disease, and some autoimmune diseases.^{7,21,33} Although studies are limited, vitamin D deficiency may affect an athlete's performance.³³ Some authors recommend limited sun exposure to assist in maintaining normal vitamin D levels; however, at present, the current position of the American Academy of Dermatologists is that adequate vitamin D levels are maintained through dietary

intake and/or vitamin D supplementation rather than through sun exposure (see <http://www.aad.org>).

Summer and winter athletes may be at increased risk for melanoma and nonmelanoma skin cancer because of their increased UV radiation exposure. Use of sunscreen, sun avoidance behaviors, and sun protective clothing are important measures to reduce UV exposure. Education for summer and winter athletes on sun protection is needed. Family, coaches, and trainers should encourage appropriate sun protection for athletes.

ACKNOWLEDGMENT

Dr. S. Harrison was funded by the F. C. Florance Bequest through the Australian College of Dermatologists in 2008.

REFERENCES

- Adams BB. Dermatologic disorders of the athlete. *Sports Med.* 2002;32(5):309-321.
- Ambras-Rudolph CM, Hofmann-Wellenhof R, Richtig E, Muller-Furstner M, Soyer P, Kerl H. Malignant melanoma in Marathon runners. *Arch Dermatol.* 2006;142:1471-1474.
- Buljan M, Bulat V, Situm M, Mihic Lugovic L, Stanic-Duktaj S. Variations in clinical presentation of basal cell carcinoma. *Acta Clin Croat.* 2008;47:25-30.
- Cassarino DS, DeRienzo DP, Barr RJ. Part 1, cutaneous squamous cell carcinoma: a comprehensive clinicopathologic classification. *J Cutan Patbol.* 2006;33:191-206.
- Cassarino DS, DeRienzo DP, Barr RJ. Part 2, cutaneous squamous cell carcinoma: a comprehensive clinicopathologic classification. *J Cutan Patbol.* 2006;33:261-279.
- Dozier S, Wagner RFJ, Black SA, et al. Beachfront screening for skin cancer in Texas Gulf Coast surfers. *South Med J.* 1997;90:55-58.
- Gorham ED, Garland CF, Garland FC, et al. Optimal vitamin D status for colorectal cancer prevention: a quantitative meta-analysis. *Am J Prev Med.* 2007;32:210-216.
- Habif TP. Nevi and malignant melanoma. In: *Clinical Dermatology.* 4th ed. Edinburgh, UK: Mosby Inc; 2004:773-813.
- Habif TP. Premalignant and malignant non-melanoma skin tumors. In: *Clinical Dermatology.* 4th ed. Edinburgh, UK: Mosby Inc; 2004:724-772.
- Hamant ES, Adams BB. Sunscreen use among collegiate athletes. *J Am Acad Dermatol.* 2005;53(2):237-241.
- Hexsal CL, Bangert SD, Hebert AA, Lim HW. Current sunscreen issues: 2007 Food and Drug Administration sunscreen labeling recommendations and combination sunscreen/insect repellent products. *J Am Acad Dermatol.* 2008;59:316-323.
- Leiter U, Garbe C. Epidemiology of melanoma and non-melanoma skin cancer: the role of sunlight. *Adv Exp Med Biol.* 2008;264:89-103.
- Lim HW. Photoprotection and sun protective agents. In Wolff K, Goldsmith LA, Katz SI, Gilchrest B, Paller AS, Leffell DJ, eds. *Fitzpatrick's Dermatology in General Medicine.* 7th ed. New York, NY: McGraw-Hill; 2007: 2137-2142.
- International Agency for Research on Cancer Working Group on Artificial Light and Skin Cancer. The association of the use of sunbeds with cutaneous malignant melanoma and other skin cancers: a systematic review. *Int J Cancer.* 2007;120:116-122.
- MacLennan R, Kelly JW, Rivers JK, Harrison SL. The Eastern Australian childhood nevus study: site differences in density and size of melanocytic nevi in relation to latitude and phenotype. *J Am Acad Dermatol.* 2003;48:367-375.
- Moehrle M. Outdoor sports and skin cancer. *Clin Dermatol.* 2008;26:12-15.
- Moehrle M. Ultraviolet exposure in the Ironman triathlon. *Med Sci Sports Exerc.* 2001;33:1385-1386.
- Moehrle M, Heinrich L, Schmidt A, et al. Extreme UV exposure of professional cyclists. *Dermatology.* 2000;201:44-45.
- Muir C, Waterhouse J, Mack T, Powell J, Whelan S. *Cancer Incidence in Five Continents.* Vol 5. Lyon, France: International Agency for Research on Cancer; 1987.
- Munger KL, Levin LI, Hollis BW, Howard NS, Ascherio A. Serum 25-hydroxyvitamin D levels and risk of multiple sclerosis. *JAMA.* 2006;296(23):2832-2838.

22. Neville JA, Welch E, Leffell DJ. Management of non-melanoma skin cancer in 2007. *Nat Clin Pract Oncol*. 2007;4(8):462-469.
23. Pfahlberg A, Kolmel KF, Gefeller O. Timing of excessive ultraviolet radiation and melanoma: epidemiology does not support the existence of a critical period of high susceptibility to solar ultraviolet radiation-induced melanoma. *Br J Dermatol*. 2001;144:471-475.
24. Price J, Ness A, Leary S, Kennedy C. Sun-safety behaviors of skiers and snowboarders on the South Island of New Zealand. *J Cosmet Dermatol*. 2006;5:39-47.
25. Rabe JH, Mamelak AJ, McElgunn PJS, Morison WL, Sauder DN. Photoaging: mechanisms and repair. *J Am Acad Dermatol*. 2006;55:1-19.
26. Rigel DS, Rigel EG, Rigel AC. Effects of altitude and latitude on ambient UVB radiation. *J Am Acad Dermatol*. 1999;40:114-116.
27. Rigel DS. Cutaneous ultraviolet exposure and its relationship to the development of skin cancer. *J Am Acad Dermatol*. 2008;58:S129-S132.
28. Rigel DS, Friedman RJ, Kopf AW. Lifetime risk of development of skin cancer in the US population: current estimate is now 1 in 5. *J Am Acad Dermatol*. 1996;35:1012-1013.
29. Rigel DS, Friedman RJ, Kopf AW, Polsky D. ABCDE-394 an evolving concept in the early detection of melanoma. *Arch Dermatol*. 2005;141:1032-1034.
30. Roewert-Huber J, Lange-Asschenfeldt B, Stockfleth E, Keri H. Epidemiology and aetiology of basal cell carcinoma. *Br J Dermatol*. 2007;157(suppl 2):47-51.
31. Schwartz RA, Bridges TM, Butani AK, Ehrlich A. Actinic keratoses: an occupational and environmental disorder. *J Eur Acad Dermatol Venereol*. 2008;22:606-615.
32. US Food and Drug Administration. The FDA approves new over-the-counter sunscreen. *FDA Consum*. 2006;40(5):4.
33. Willis KS, Peterson NJ, Larson-Meyer DE. Should we be concerned about the vitamin D status of athletes? *Int J Sport Nutr Exerc Metab*. 2008;18:204-224.

For reprints and permission queries, please visit SAGE's Web site at <http://www.sagepub.com/journalsPermissions.nav>.