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Technical Report—Ultraviolet Radiation: A Hazard to Children and Adolescents

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KEY WORDS

sun, ultraviolet radiation, children, skin cancer, skin-cancer prevention, melanoma, vitamin D, prevention, sun protection, sunscreen, tanning, artificial tanning

ABBREVIATIONS

UVR—ultraviolet radiation
NMSC—nonmelanoma skin cancer
PABA—para amino benzoic acid
SPF—sun-protection factor
BCC—basal cell carcinoma
SCC—squamous cell carcinoma
IARC—International Agency for Research on Cancer
FDA—Food and Drug Administration
UPF—ultraviolet protection factor
NHANES—National Health and Nutrition Examination Survey
AAP—American Academy of Pediatrics
25(OH)D—25-hydroxyvitamin D

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abstract

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Sunlight sustains life on earth. Sunlight is essential for vitamin D synthesis in the skin. The sun's ultraviolet rays can be hazardous, however, because excessive exposure causes skin cancer and other adverse health effects. Skin cancer is a major public health problem; more than 2 million new cases are diagnosed in the United States each year. Ultraviolet radiation (UVR) causes the 3 major forms of skin cancer: basal cell carcinoma; squamous cell carcinoma; and cutaneous malignant melanoma. Exposure to UVR from sunlight and artificial sources early in life elevates the risk of developing skin cancer. Approximately 25% of sun exposure occurs before 18 years of age. The risk of skin cancer is increased when people overexpose themselves to sun and intentionally expose themselves to artificial sources of UVR. Public awareness of the risk is not optimal, compliance with sun protection is inconsistent, and skin-cancer rates continue to rise in all age groups including the younger population. People continue to sunburn, and teenagers and adults are frequent visitors to tanning parlors. Sun exposure and vitamin D status are intertwined. Adequate vitamin D is needed for bone health in children and adults. In addition, there is accumulating information suggesting a beneficial influence of vitamin D on various health conditions. Cutaneous vitamin D production requires sunlight, and many factors complicate the efficiency of vitamin D production that results from sunlight exposure. Ensuring vitamin D adequacy while promoting sun-protection strategies, therefore, requires renewed attention to evaluating the adequacy of dietary and supplemental vitamin D. Daily intake of 400 IU of vitamin D will prevent vitamin D deficiency rickets in infants. The vitamin D supplementation amounts necessary to support optimal health in older children and adolescents are less clear. This report updates information on the relationship of sun exposure to skin cancer and other adverse health effects, the relationship of exposure to artificial sources of UVR and skin cancer, sun-protection methods, vitamin D, community skin-cancer–prevention efforts, and the pediatrician's role in preventing skin cancer. In addition to pediatricians' efforts, a sustained public health effort is needed to change attitudes and behaviors regarding UVR exposure. *Pediatrics* 2011;127:e791–e817

BACKGROUND

Sunlight sustains life on earth. The sun provides warmth, is needed for photosynthesis, drives biorhythms, and promotes feelings of well-being, and sunlight is essential for vitamin D synthesis in skin.

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The sun emits electromagnetic radiation that ranges from short-wavelength, high-energy x-rays to long-wavelength, lower-energy radio waves. Ultraviolet ("above-violet") radiation (UVR) waves range from 200 to 400 nm. UVR waves are longer than x-rays and shorter than visible light (400–700 nm) and infrared ("below-red" or "heat") radiation (>700 nm). UVR is subdivided into UVC (200–290 nm), UVB (290–320 nm), and UVA (320–400 nm, further subdivided into UVA2 [320–340 nm]) and UVA1[340–400 nm]). UVC rays possess the highest energy but do not penetrate the atmosphere. Thus, middle-wavelength (UVB) and long-wavelength (UVA) UVR, visible light, and infrared radiation have the greatest biological significance.

Solar radiation that reaches the earth's surface constitutes approximately 95% UVA and 5% UVB.[1] Most UVB radiation is absorbed by stratospheric ozone, but ozone absorbs little or no UVA or visible light. The ozone layer does not have uniform thickness; ozone concentration tends to increase toward the poles but is thinning in some areas.[2] Ozone depletion has a significant effect on the amount of UVB that reaches the earth.[2] Chlorofluorocarbons used as aerosol propellants and in refrigeration and air conditioning can destroy ozone.

UVR that passes through the stratosphere (10–50 km above sea level) is scattered by molecules such as oxygen and nitrogen. It then passes through the troposphere (0–10 km above sea level), where it is absorbed and scattered by pollutants, such as soot, and attenuated by clouds. Clouds reduce the intensity of UVR but not to the same extent that infrared intensity is reduced; the sensation of heat is diminished, which results in the potential for overexposure.

The intensity of UVB radiation varies; it has greater intensity in summer than in winter, at midday than in morning or late afternoon, in places closer to the equator, and at higher altitudes. Sand, snow, concrete, and water can reflect up to 85% of sunlight, thus intensifying exposure.[3] Water is not a good photoprotectant, because UVR can penetrate to a depth of 60 cm, which results in a significant exposure. In contrast to the variability of UVB radiation, UVA radiation is relatively constant throughout the day and the year.

UVR can be produced by man-made lamps (eg, sunlamps) and tools (eg, welding tools), but the sun is the primary source of UVR for most people.[4] UVR has been used for decades to treat skin diseases, especially psoriasis.[1]

UVR EFFECTS ON THE SKIN

The skin is the organ most exposed to environmental UVR and to associated sequelae. Exposure to UVR may result in erythema and sunburn, tanning, skin aging, photosensitivity, and carcinogenesis (nonmelanoma skin cancer [NMSC] and cutaneous malignant melanoma).

Erythema and Sunburn

Erythema and sunburn are acute reactions to excessive amounts of UVR. Exposure to solar radiation causes vasodilatation and increases the volume of blood in the dermis, which results in erythema. The minimal erythema (or erythemal) dose (the amount of UVR exposure that will cause minimal erythema or slight pinkness of the skin) depends on factors such as (1) skin type, (2) skin thickness, (3) the amount of melanin in the epidermis, (4) melanin production after sun exposure, and (5) the intensity of the radiation. A classification system of 6 skin types ranging from light to dark (Table 1) takes into account a person's expected sunburn and suntan tendency.[5]

The ability of UVR to produce erythema depends on the radiation wavelength expressed as the erythema "action spectrum" (the rate of a physiologic activity plotted against wavelength of light showing which wavelength of light is most effectively used in a specific chemical reaction). The action spectrum for erythema and sunburn is mainly in the UVB range.[6]

TABLE 1 Classification of Sun-Reactive Skin Types[5]

Skin Type	History of Sunburning or Tanning
I	Always burns easily, never tans
II	Always burns easily, tans minimally
III	Burns moderately, tans gradually and uniformly (light brown)
IV	Burns minimally, always tans well (moderate brown)
V	Rarely burns, tans profusely (dark brown)
VI	Never burns, deeply pigmented (black)

Tanning

Tanning is a protective response to sun exposure.[7] Immediate tanning (or immediate pigment-darkening) results from oxidation of existing melanin after exposure to visible light and UVA. Immediate pigment-darkening becomes visible within several minutes and usually fades within 1 to 2 hours. Delayed tanning occurs when new melanin is formed after UVB exposure. Delayed tanning becomes apparent 2 to 3 days after exposure, peaks at 7 to 10 days, and may persist for weeks or months. According to recent evidence, the tanning response means that DNA damage has occurred in skin.[8]

Skin-Aging (Photoaging)

Chronic unprotected exposure to UVR weakens the skin's elasticity and results in sagging cheeks, deeper facial wrinkles, and skin discoloration. Photoaged skin is characterized by alterations of cellular components and of the extracellular matrix. There is accumulation of disorganized elastin and of fibrillin (its microfibrillar component in the deep dermis) and a severe loss of interstitial collagens, the major

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structural proteins of the dermal connective tissue. These changes result primarily from exposure to UVR-generated reactive oxygen species that deplete and damage the skin's enzymatic and nonenzymatic antioxidant defense systems.[9,10]

Photosensitivity

Chemical photosensitivity refers to an adverse cutaneous reaction that results when certain chemicals or drugs are applied topically or taken systemically at the same time that a person is exposed to UVR or visible radiation. Phototoxicity is a form of chemical photosensitivity that does not depend on an immunologic response; the reaction can occur on first exposure to an agent. Most phototoxic agents are activated in the range of 320 to 400 nm (the UVA range). Drugs associated with phototoxic reactions include those commonly used by adolescents, such as nonsteroidal anti-inflammatory agents; tetracyclines and tretinoin; other medications such as phenothiazines, psoralens, sulfonamides, and thiazides; and para amino benzoic acid (PABA) esters.[11] Photoallergy is an acquired altered reactivity of the skin, usually triggered by exposure to UVA, that depends on antigen-antibody or cell-mediated hypersensitivity. Photoallergic reactions involve an immunologic response to a chemical or drug that is altered by UVR. PABA-containing sunscreens, fragrances, sulfonamides, and phenothiazines are associated with photoallergic reactions.[11] The consequences of exposure to a photosensitizing agent can be uncomfortable, serious, or life-threatening. People who take medications or use topical agents known to be sensitizing should do their best to limit sun exposure and avoid UVA from artificial sources. They should wear fully protective clothing and apply sunscreen with a high sun-protection factor (SPF) when some light exposure is inevitable.[12]

Plants that contain furocoumarins may lead to phototoxic reactions or phytophotodermatitis. These commonly encountered plants include anise, diseased celery, dill, fennel, fig, lemon, lime, mustard, parsnip, parsley, and chrysanthemums. Phytophotodermatitis can occur through ingestion of plants or, more commonly, through topical contact.[13]

Up to 80% of patients with lupus erythematosus have photosensitivity. The threshold UVR dose that triggers cutaneous or systemic reactions is much lower than that for sunburn. Many patients are not aware of the association of flares with UVR exposure, because the latency period between exposure and skin eruptions can range from several days to 3 weeks.[14]

Carcinogenesis

Nonmelanoma Skin Cancer

NMSC includes basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). In the US adult population, NMSC is the most common malignant neoplasm, with more than 2 million cases diagnosed each year. Most of these are BCC, SCC occurs less often.[15] The rate of NMSC has been increasing in the United States, but the exact number is not precisely known, because physicians are not required to report NMSC to cancer registries. NMSC is rarely fatal; nevertheless, it is estimated that each year, approximately 2000 people die of NMSC.[15]

In general, NMSC occurs in maximally sun-exposed areas of fair-skinned people. NMSC is uncommon in black people and people with increased natural pigmentation. The head and neck region is the most common site for BCC and SCC; 80% to 90% of cases occur in this area in the general population. NMSC is more common in people older than 50 years, and the incidence in this age group is increasing rapidly.[16–18] People with immune suppression, including organ transplant recipients, also are at higher risk. Genetically based conditions, such as basal cell nevus syndrome, xeroderma pigmentosum (a condition in which there is a genetically determined defect in the repair of DNA damaged by UVR),[19] and albinism, are risk factors for the accelerated development of NMSC. Treatment with UVR for psoriasis also increases risk.[19] NMSC is extremely rare in children in the absence of predisposing conditions.[20]

The incidence of NMSC is increasing in young adults. Researchers examined the gender- and age-specific incidence of BCC and SCC in a young (<40-year-old), primarily white and middle-class population within Olmsted County, Minnesota, by using comprehensive medical records available through the Rochester (MN) Epidemiology Project.[21] Over the period of 1976–2003, the incidence of BCC increased significantly among young women, and the incidence of SCC increased significantly among both men and women.

A trend toward a greater number of BCC cases occurring on the torso in younger patients has been reported.[21–23] This change in location supports the possibility that excessive outdoor tanning, use of tanning booths, or both give rise to BCC. Tanning-bed use has been shown to be a risk factor for NMSC in young women.[24]

Sun exposure is the main environmental cause of NMSC. Cumulative exposure over long periods, which results in photodamage, is considered important in the pathogenesis of SCC.

Melanoma

Melanoma is primarily a disease of the skin. Primary extracutaneous sites include the eye, mucous membranes,

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gastrointestinal tract, genitourinary tract, leptomeninges, and lymph nodes. Ninety-five percent of melanomas occur in the skin.[25] If detected when the tumor is thin and small, cutaneous malignant melanoma has an excellent prognosis. However, metastatic melanoma has no successful treatment options. Prevention and early detection, therefore, are crucial in this disease.

Many authorities have stated that the incidence of cutaneous malignant melanoma (hereafter referred to as "melanoma") has reached epidemic proportions. Possible factors contributing to the increased incidence of melanoma include the decrease in the earth's protective ozone layer, changing patterns of dress that favor more skin exposure, more opportunities for leisure activities in sunny areas, and increased exposure to artificial sources of UVR for tanning purposes.

In the United States, melanoma is the fifth most common cancer in men and the sixth most common in women.[26] The incidence of melanoma is increasing rapidly in the United States.[27] In 1935, the lifetime risk for a person in the United States developing invasive melanoma was 1 in 1500. In 2007, this risk was 1 in 63 for invasive melanomas and 1 in 33 when in situ melanomas were included. Worldwide, melanoma is increasing faster than any other malignancy.[28] Melanoma represents fewer than 5% of all skin cancers but is the cause of almost all skin-cancer deaths. The American Cancer Society predicted that approximately 68 130 new melanoma cases would be diagnosed in 2010, with 8700 deaths.[29]

Melanoma is more likely to occur in males and at older ages but also occurs in teenagers and young adults. It is the second most common cancer of women in their 20s and the third most common cancer of men in their 20s.[30] Melanoma incidence is increasing in young women aged 15 to 39 years.[31] People at highest risk have light skin and eyes and sunburn easily. Risk of developing melanoma is increased at older ages, in people who have already had melanoma, or in people who have had a first-degree relative with melanoma. Melanomas frequently are found in people with xeroderma pigmentosum and related disorders. In a large case-control study from the Netherlands, the risk of developing melanoma was increased in women who had used estrogens (either as oral contraceptives or hormone-replacement therapy) for more than half a year.[32]

Melanoma is rare in children, but it does occur. Studies have documented an increase in the incidence in children and adolescents, even in the absence of predisposing conditions such as xeroderma pigmentosum. From 1973 to 2001, the incidence of melanoma in US children younger than 20 years increased 2.9% annually.[33] An increase in incidence was noted in Sweden during 1973–1992,[34] but incidence then decreased.[35] Ferrari et al[36] reviewed a 25-year experience with 33 Italian children with melanoma who were 14 years or younger at presentation. The children's lesions were not typical of melanoma lesions in adults. Melanoma lesions in adults generally follow the "ABCDEs": they are asymmetric (A), have irregular borders (B), variegated color (C), and diameter (D) larger than 6 mm (the size of a pencil eraser), and change or evolve (E).[37] In the Ferrari et al[36] series, however, many lesions in children were amelanotic (pink, pink-white, or red) and tended to be raised and to have regular borders. The key to diagnosis for these children was the recognition that the melanoma lesions were unlike any other lesions on the child.

EVIDENCE THAT UVR CAUSES SKIN CANCER

In 1992, the International Agency for Research on Cancer (IARC) reviewed the evidence for the carcinogenicity of solar radiation. They concluded that "[t]here is sufficient evidence in humans for the carcinogenicity of solar radiation. Solar radiation causes cutaneous malignant melanoma and nonmelanocytic skin cancer.[1]" Since that time, evidence has strengthened the link between sunlight exposure and skin cancer.

Cellular Studies

UVB is absorbed by and can directly damage DNA, which ultimately leads to the development of skin cancer.[38] The genotoxic effects of solar UVB radiation are mainly mediated by direct absorption in the epidermis of photons by DNA, which results primarily in cyclobutane pyrimidine dimers (formed between adjacent pyrimidine bases located on the same DNA strand) and pyrimidine (6-4) pyrimidone photoproducts.[7] Incorrect repair of these lesions results in the formation of mutations in epidermal cells, which causes the development of cancer.[7,39]

UVA penetrates more deeply into the skin than does UVB, including reaching the basal layer of the epidermis and dermal fibroblasts.[38] UVA causes oxidative damage to DNA that is potentially mutagenic.[7]

Biological Evidence

Biological evidence suggests that sunlight exposure is important in the pathogenesis of melanoma. Results of studies in opossums suggest that portions of the UVA spectrum may play a role in the pathogenesis of melanoma[40] and that portions of the UVA and UVB spectrums promote development of carcinomas in mice.[41] Melanoma can be induced by UVB and UVA radiation in certain fish.[42] Research ethics make it

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impossible to determine directly which wavelengths result in skin cancer in humans.

Melanoma has been induced in human newborn foreskins grafted onto immunologically tolerant animals exposed to UVR.[43] Melanomas and NMSC are often found in people with xeroderma pigmentosum and related disorders.[44]

Epidemiologic Evidence

Latitude or Estimated Ambient Solar UVR

The rates of BCC and SCC increase with increasing ambient solar UVR. There is a direct relationship between the incidence of NMSC and latitude; higher rates are found closer to the equator (where the amount of sunlight is greater).[28] The relationship of melanoma with latitude is not as clear as that for NMSC.[28]

Race and Pigmentation

BCC and SCC occur primarily in white people.[15] Incidence and mortality rates of melanoma are highest in white people (Table 2).[27] There is, in general, an inverse relationship between skin-cancer incidence and the skin pigmentation of people in various countries in the world. Superficial epidermal melanin decreases the transmission of UVR, which may protect the deeper basal layer melanocytes and several layers of keratinocytes from sunlight-induced changes that lead to their malignant transformation.[7]

Melanin, a dark pigment produced by melanocytes, accounts for most of the variation in human skin appearance. Melanin that is genetically determined is termed "constitutive" melanin pigmentation. When this basic pigmentation is increased by exposure to UVR, it is termed "inducible" or "facultative" melanin pigmentation. Melanin is thought to have evolved as an optical and chemical photoprotective filter that functions as a natural "sunscreen" to regulate UVR penetration into skin. In early human evolution, the more highly melanized skins of people indigenous to the tropics afforded better protection against the deleterious effects of UVR. A dark epidermis protected sweat glands from UVR-induced injury and ensured the integrity of somatic thermoregulation. Highly melanized skin also protected against UVR-induced photolysis of folate, a metabolite essential for normal development of the embryonic neural tube.[45] As people migrated outside the tropics to northern areas, a lighter skin color was needed as an adaptation to promote maintenance of UVR-induced synthesis of vitamin D_3 in areas of lower UVR exposure.[45] As the pace of human migrations quickened in recent centuries, however, populations have found themselves in UVR-irradiation patterns to which they are poorly adapted. Cultural practices, such as sunbathing and covering up for religious reasons, exacerbate or mitigate the mismatch in degree of melanin protection to UVR exposures.[45]

TABLE 2 Melanoma Incidence and Mortality Rates According to Race/Ethnicity[27]

Race/Ethnicity	Men, Rate per 100 000 Men		Women, Rate per 100 000 Women	
	Incidence	Mortality	Incidence	Mortality
White	28.9	4.4	18.7	2.0
Black	1.1	0.5	1.0	0.4
Asian/Pacific Islander	1.6	0.5	1.3	0.3
American Indian/Alaska Native	3.9	1.6	2.8	0.9
Hispanic	4.6	0.9	4.7	0.6

History of Sun Exposure

The pattern of sun exposure is important in the etiology of BCC, SCC, and melanoma skin cancers. Personal sun exposure is usually characterized by (1) total sun exposure, (2) occupational exposure (which signifies a more chronic exposure), and (3) nonoccupational or recreational exposure (which signifies intermittent exposure).[46] SCC is significantly associated with estimated total sun exposure and with occupational exposure. Chronic exposure to UVB is now considered the main environmental cause of SCC. SCC seems to be most straightforwardly related to the total sun exposure: these tumors occur on skin areas that are most regularly exposed (face, neck, and hands), and the risk rises with the lifelong accumulated dose of UVR.[47] BCC and melanoma are significantly associated with intermittent sun exposure (ie, sunburning or "brutal" exposure), whereas SCC does not show this relationship. Melanoma is more strongly associated with intermittent sun exposures than is BCC.[46]

Childhood Sun Exposure

Childhood and adolescence are often considered to contain "critical periods of vulnerability" when people are especially susceptible to effects of toxic exposures. Approximately 25% of lifetime sun exposure occurs before 18 years of age.[48] Sun exposure and blistering sunburns during youth may be more intense than later in life because of youths' behavior. Exposure may result in alteration of melanocyte DNA and an increase in the risk of malignant degeneration in nevi as children age.

Sunlight exposure during childhood and adolescence is generally considered to confer increased risk of melanoma compared with exposure at older ages. This issue was reviewed in an analysis of epidemiologic studies categorized into 2 groups.[49] The first

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group contained 20 ecologic studies (ie, studies in which the unit of observation is the population or community) relating the risk of melanoma to places of residence. These studies were conducted on the basis of the fact that ambient solar radiation increases with proximity to the equator and included studies of migrants to locations with markedly different levels of sunlight. The second group consisted of case-control studies in which measures of sun exposure between people with melanoma and those without were compared.

In the first group, most studies revealed that people who migrated from "low" to "high" areas of ambient solar radiation had decreasing melanoma risk with arrival at older ages, whereas those who arrived in childhood (younger than 10 years) or adolescence (younger than 15 years) had similar risks as people who were native-born. The 1 study that investigated age-specific "high-to-low" migration demonstrated higher risk in people born in a sunny area or having had more than 1 year living in a sunny area before 10 years of age.[49] The results of most studies of the age of migration, therefore, supported the "critical-period" hypothesis.

Ten case-control studies that examined melanoma risks associated with personal sun exposure during 2 or more age periods were evaluated in the second group. Findings of these studies differed widely without consistent associations with childhood sun exposure. Three studies reported significantly increased risks of melanoma associated specifically with episodes of sunburns during childhood, whereas 1 Swedish study found no effect of childhood sunburn but reported significantly higher risks associated with adulthood sunburns. The remaining 5 studies reported similar risks of melanoma regardless of whether sunburn occurred during childhood or adulthood. The summary odds ratios associated with sunburn during childhood and adulthood were 1.8 (95% confidence interval: 1.6–2.2) and 1.5 (95% confidence interval: 1.3–1.8), respectively, although there was significant heterogeneity among the studies for the estimates of childhood sunburn. The authors underscored the lack of reliability of recalling personal sun exposure as a reason for the inconsistencies between the migrant and case-control studies and considered the evidence from the migrant studies to be of higher quality.[49] In a large multicenter case-control study, the authors concluded that excessive UVR exposure later in life may be as important a risk for melanoma as UVR exposure earlier in life.[50] There was a similar upward gradient of melanoma risk related to sunburns during childhood (defined as age $\leq$ 15 years) and adulthood (defined as age $>$ 15 years). More than 5 sunburns doubled the melanoma risk irrespective of whether those sunburns occurred in childhood or adulthood.[50]

There is biological plausibility to support the heightened susceptibility of young melanocytes. Peak melanocytic activity occurs in early life as demonstrated by the steady acquisition of nevi during childhood and adolescence. Freckling is also prominent at these ages; freckles in children often appear abruptly after high-dose sun exposure and are thought to represent clones of mutated melanocytes. The presence of freckles is associated with an increased risk of melanoma.[7] Young melanocytes may be especially vulnerable to the adverse effects of solar radiation. Sunlight may have both early and late effects on the development of melanoma (akin to cancer "initiation," "promotion," and "progression"[51]), and the biological effectiveness of sunlight in initiating melanoma is greatest during the period of peak melanocytic activity. Populations exposed to high sunlight levels in childhood will have more people with more initiated melanocytes than populations of those who experienced lower sunlight levels. This "melanoma potential" is retained when people move to a different environment.[49]

Nevi

Acute sun exposure is implicated in the development of nevi (moles) in children. The number of nevi increases with age[52]; nevi occur with more frequency on sun-exposed areas, and the number of nevi on exposed areas increases with the total cumulative sun exposure during childhood and adolescence.[53] Children with light skin who tend to burn rather than tan have more nevi at all ages, and children who have more severe sunburns have more nevi.[52]

There is a relationship between the number and type of melanocytic nevi and the development of melanoma. The presence of congenital melanocytic nevi (CMN) (pigment cell malformations formed during gestation and visible at or shortly after birth) increases melanoma risk. In a review of 14 studies—case series with adequate follow-up periods—investigators found an overall risk of melanoma arising in CMN of 0.7%, which was lower than expected. Melanoma risk strongly depended on the size of the CMN and was highest in nevi designated as garment nevi (defined as nevi situated on the trunk that measure $>$40 cm in largest diameter or expected to reach this size in adulthood). The mean age at melanoma diagnosis (15.5 years) and median age of diagnosis (7 years) underscored the maximum risk in childhood and adolescence.[54]

Dysplastic melanocytic nevi typically are 5 mm or larger in diameter; usually have fuzzy, irregular borders; and

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have variegated color. Dysplastic nevi are considered precursor lesions that increase melanoma risk.[55] The familial dysplastic nevus syndrome is a disorder with the following features: (1) a distinctive appearance of abnormal melanocytic nevi; (2) unique histologic features of the nevi; (3) autosomal dominant pattern of inheritance; and (4) hypermutability of fibroblasts and lymphoblasts. Fibroblasts and lymphoblasts from patients with this syndrome are abnormally sensitive to UV damage, and people with this syndrome are at markedly higher risk of developing melanoma.[56] Certain families with germ-line mutations in *CDKN2A*, *CDK4*, and other genes are at increased risk of developing dysplastic nevi and melanoma.[57]

History of Exposure to Artificial UVR

Exposure to tanning beds and sunlamps, which produce primarily UVA, is associated with increased risk of developing BCC, SCC, and melanoma.

UVR EFFECTS ON THE EYE

In adults, more than 99% of UVR is absorbed by the anterior structure of the eye, although some of it reaches the retina.[58] Acute exposure to UVR can result in photokeratitis.[59] Gazing directly into the sun (as can occur during an eclipse) can cause focal burns to the retina (solar retinopathy).[60]

Exposure to solar UVB radiation is associated with an increased risk of cataracts.[61] UVR can contribute to the development of pterygium, corneal degenerative changes, and cancer of the skin around the eye.[58] There is evidence for a probable relationship between UVR exposure and squamous intraepithelial neoplasms of the conjunctiva or cornea, but there is insufficient evidence to determine if there is a relationship between UVR exposure and the development of macular degeneration.[62] Melanoma of the uveal tract, the most common primary intraocular malignant neoplasm in adults, is associated with light skin color, blond hair, and blue eyes. There is contradictory evidence regarding the role of UVR in causing uveal melanoma.[63,64]

UVR EFFECTS ON THE IMMUNE SYSTEM

Exposure to UVR contributes to immunosuppression, which is increasingly recognized as important in the development of skin cancer. UVR exposure is thought to have 2 effects: skin-cancer induction and immune suppression.[65] Experiments in mice chronically exposed to UVR have shown that tumors induced by UVR are highly antigenic and are recognized and rejected by animals with normal immune systems. The tumors grow progressively, however, when transplanted into mice with immune systems that are compromised.[65] UVR exposure induces "systemic" immune suppression so that exposure on 1 body site suppresses the immune response when the antigen is introduced at a distant site that was not irradiated. Soluble factors implicated in systemic immune suppression include platelet-activating factor (PAF), prostaglandin E2 (PGE2), *cis*-urocanic acid, histamine, interleukin 4, interleukin 10, and α-melanocyte-stimulating hormone.[65]

Skin cancers are common in people exposed to immunosuppressive agents, which further illustrates the role of the immune system. In people who have had renal transplants, lifelong immunosuppressive treatment needed for adequate graft function leads to a reduction of immunosurveillance and an increased risk of various cancers. With increased duration of transplantation, skin cancer is now one of the commonest causes of death in renal transplant recipients. Twenty years after transplantation, approximately 40% to 50% of white recipients in Western countries and 70% to 80% of those in Australia will have developed at least 1 NMSC (mostly SCC).[66] People who have had renal transplants also have an increased incidence of melanoma.[67] Because ongoing immunosurveillance has been lacking, skin cancers in people who have received organ transplants are likely to behave aggressively with a higher rate of local recurrence and a greater tendency to be invasive and metastatic.[66]

ARTIFICIAL SOURCES OF UVR

People may be exposed to artificial sources of UVR in several ways, including as treatment for medical conditions (such as psoriasis), in occupational settings (such as welding), and for cosmetic purposes. Sunlamps and tanning beds are the main sources of artificial UVR used for deliberate purposes.[68] Artificial tanning is a relatively new phenomenon that results in potentially large exposures to UVA and UVB. The "tanning industry" has grown quickly; it takes in $5 billion in annual revenue, up from $1 billion in 1992.[69] Each day, more than 1 million people tan in one of 50 000 tanning facilities in the United States.[69] Indoor tanning also is popular in northern Europe and is gaining popularity in Australia.[68]

Artificial tanning is a common practice among teenagers. In a national sample of non-Hispanic white teenagers 13 to 19 years of age in the United States, 24% of respondents—representing 2.9 million teenagers—reported using a tanning facility at least once in their lives.[70] In another national survey, 10% of youth 11 to 18 years of age reported using indoor tanning beds or sunlamps in the previous year.[71] Women and girls represent the majority of people who artificially tan. Of the 1 million people daily who are tanning-salon customers, 70% are females 16 to 49 years of age.[69] Twenty-eight per-

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cent of white US teenaged girls interviewed in 1996 had used tanning salons 3 or more times during their lives.[70] Tanning-bed use increases with age, from 7% among 14-year-old girls to 16% among 15-year-old girls and to 35% among 17-year-old girls.[72]

Tanning-bed use by adolescent girls is often associated with other unhealthy behaviors. In 1 study, frequent tanning-bed use was associated with smoking cigarettes, binge-drinking, being highly concerned about weight, and other risk behaviors.[73]

Evidence That Tanning May Be Addictive

Exposure to UVR from sunlight or tanning parlors may be addictive. Beachgoers aged 18 years and older in Galveston, Texas, were interviewed using questions to evaluate dependence on tanning. Subjects completed surveys that included a tanning-specific modification of a screening instrument for alcoholism and questions to evaluate criteria for tanning-specific substance-related disorder. Of 145 subjects, 26 (18%) screened positive on both measures, and 63 (43%) screened positive on 1 measure. The authors concluded that those who chronically and repeatedly expose themselves to UVR to tan may have a type of UVR substance-related disorder.[74] In a study of 14 adults, tanners overwhelmingly preferred UVR-emitting beds when asked to choose blindly between UVR-emitting and non–UVR-emitting tanning beds. A more relaxed and less tense mood was reported after UVR exposure compared with after non-UVR exposure.[75] In another study, the opioid antagonist naloxone was given to 8 frequent salon tanners and 8 people who were infrequent tanners. Withdrawal-like symptoms were induced in 4 of 8 frequent salon tanners; no symptoms occurred in the 8 infrequent tanners. It is conjectured that ultraviolet light exposure results in induction of cutaneous endorphins; thus, endorphin release may play a role in driving UVR-exposure behavior. If cutaneous endorphins are induced, an endorphin blockade would be expected to block the effect.[76] A recent study assessed the prevalence of addiction to indoor tanning among college students and its association with substance use and symptoms of anxiety and depression. Two written measures, the CAGE (cut down, annoyed, guilty, eye-opener) Questionnaire, used to screen for alcoholism, and the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR) criteria for substance-related disorders were modified to evaluate study participants for addiction to indoor tanning. Self-report measures of anxiety, depression, and substance use were administered. Among the 229 study participants who had tanned indoors, 70 (30.6%) met CAGE criteria and 90 (39.3%) met DSM-IV-TR criteria for addiction to indoor tanning. Indoor tanners reported significantly greater symptoms of anxiety and greater use of alcohol, marijuana, and other substances than those who did not meet these criteria. Depressive symptoms did not significantly vary according to indoor-tanning-addiction status.[77]

Effects of Artificial UVR on Human Skin

Tanning beds primarily emit UVA radiation, although a small amount (<5%) is in the UVB range.[68] In terms of biological activity, the intensity of UVA radiation produced by large, powerful tanning units may be 10 to 15 times higher than that of the midday sun. Frequent indoor tanners may receive 1.2 to 4.7 times the annual dose of UVA than is received from the sun, in addition to doses from sun exposure.[68] This intensity of exposure is not found in nature and is a new phenomenon in people.[78]

Artificial UVR exposure has been shown repeatedly to induce erythema and sunburn. Erythema or burning effects were reported by 18% to 55% of users of indoor tanning equipment in Europe and North America.[68] Although UVB is much more potent than UVA in causing sunburn, high fluxes of UVA can cause erythema in people who are sensitive to sunlight. In people who tan easily, exposure to tanning appliances will lead first to immediate pigment-darkening. A more permanent tan will occur with accumulated exposure, depending on individual tanning ability and the amount of UVB present in the light spectrum of the tanning lamps. Immediate pigment-darkening has no photoprotective effect against UVR-induced erythema or sunburn. In addition, the permanent tan induced by UVA and UVA-induced skin-thickening provides little photoprotection.

Other frequently reported effects of artificial tanning include skin dryness, pruritus, nausea, photodrug reactions, disease exacerbation (eg, systemic lupus erythematosus), and disease induction (eg, polymorphous light eruption). Long-term health effects include skin-aging, effects on the eye (eg, cataract formation), and carcinogenesis.

In 1992, the IARC[1] classified the "use of sunlamps and sunbeds" as "probably carcinogenic to humans." In 2000, the National Institutes of Health stated that "exposure to sunlamps or sunbeds is known to be a human carcinogen, based on sufficient evidence of carcinogenicity from studies in humans, which indicate a causal relationship between exposure to sunlamps or sunbeds and human cancer."[79]

A case-control study demonstrated a significant association between using any tanning device and the incidence

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of SCC and BCC.[80] A prospective cohort study of 106 379 women in Scandinavia examined melanoma risk in females who reported having used a sunbed or sunlamp. A 55% increase in melanoma risk was found in women who reported having used a tanning device at least once per month in at least 1 of the 3 decades between 10 and 39 years of age, compared with those who had never or rarely used a tanning device during those 3 decades.[81]

In 2006, the IARC published an updated analysis of studies of the carcinogenicity of artificial UVR with regard to melanoma, SCC, and BCC.[68] On the basis of 19 studies, any previous use of sunbeds was positively associated with melanoma (summary relative risk: 1.15 [95% confidence interval: 1.00–1.31]), although there was no consistent evidence of a dose-response relationship. First exposure to sunbeds before 35 years of age significantly increased the risk of melanoma on the basis of 7 studies (summary relative risk: 1.75 [95% confidence interval: 1.35–2.26]). The summary relative risk of 3 studies of SCC showed an increased risk. Studies did not support an association for BCC. The evidence did not support a protective effect of the use of sunbeds against damage to the skin from subsequent sun exposure.

Biological evidence supports the epidemiologic studies. The skin of volunteers exposed to UVA lamps used in tanning appliances showed DNA damage.[68] The IARC concluded that young adults should be discouraged from using indoor tanning equipment and that restricted access to sunbeds by minors should be strongly considered.

Tanning-Industry Response

The tanning industry has fought vigorously to allow teenagers access to tanning salons and promotes the purported health benefits and safety of artificial tanning. The Indoor Tanning Association, an industry group founded in 1999, promotes "a responsible message about moderate tanning and sunburn prevention."[82] Their mission is to "protect the freedom of individuals to acquire a suntan, via natural or artificial light."[83] The Indoor Tanning Association claims that "controlled" salon tanning is safer than "uncontrolled" beach tanning; this concept is not supported by laboratory, behavioral, or epidemiologic data.[78] Another commonly held misconception is that getting a "prevacation tan"—when people visit tanning salons to prepare skin for a sunny vacation—will protect against subsequent skin damage during the vacation. This practice actually leads to extra radiation exposure not only before the vacation but also afterward, because people use fewer sun-protection precautions during the vacation because of a mistaken belief that the tan will protect them.[69] A prevacation tan results in minimal protection (an SPF of 3),[78] which provides virtually no protection against sun-induced DNA damage.[68]

Antitanning Legislation and Recommendations

Because of mounting evidence about the carcinogenicity of artificial UVR, support for regulations to limit teenagers' access to tanning facilities has been widespread. The World Health Organization,[84] the American Medical Association,[85] and the American Academy of Dermatology[86] all support legislation to ban the use of artificial tanning devices by people younger than 18 years. The IARC review concluded that young adults should be discouraged from using indoor tanning equipment and that restricted access to sunbeds by minors should be strongly considered.[68]

France has banned indoor tanning for people younger than 18 years since 1997; indoor tanning for those younger than 18 years also is prohibited in the province of New Brunswick, Canada.[87] Currently (as of February 2011), more than 60% of US states regulate tanning facilities for minors.[88] Some states completely ban salon access to children younger than 14 years, whereas other states ban access to adolescents 15 or 16 years of age. Some states require written parental consent or written consent with the parent present at the facility or a doctor's prescription. In California, where tanning-salon use is banned for children younger than 14 years, recent legislation made annual signed parental consent required for tanning-facility use by adolescents 14 to 17 years of age.[89] During the 2010 legislative session, 20 states introduced bills to regulate tanning facilities for minors.[88,*]

The Indoor Tanning Association has fought against legislative initiatives and stated that legislation will harm business[90] and that tanning is an issue of parental rights: "When it involves a suntan, the State has no business inserting itself between child and parent. This notion that government knows more about child rearing than parents is preposterous."[89] Pediatric health advocates have countered this argument by stating that laws to limit minors' access to tanning parlors should be thought of in the same way as laws that limit youth access to tobacco.[87,89] All states prohibit the purchase of tobacco products by those younger than 18 years; some prohibit tobacco sales to those younger than 19 years.[87] Tanning legislation is often not enforced.[91]

Artificial Tanners (Spray Tans and Sunless Tanning Lotions)

Several organizations have suggested that people who wish to obtain the look of a tanned skin use artificial (or "sunless") tanning products to substitute

*For more information on current state laws that restrict the use of tanning beds by children and teenagers, please contact the AAP Division of State Government Affairs.

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for tanning obtained by going outside or at tanning salons. Sunless tanners contain dihydroxyacetone, a chemical that reacts with amino acids in the stratum corneum to form brown-black compounds—melanoidins—that deposit in skin. Dihydroxyacetone is a mutagen that induces DNA strand breaks in certain strains of bacteria; it has not been shown to be carcinogenic in animal studies.[92]

Dihydroxyacetone is the only color additive approved by the US Food and Drug Administration (FDA) for use as a tanning agent.[93] Dihydroxyacetone-containing tanning preparations may be applied to the consumer's bare skin by misters at sunless tanning booths. Bronzers are water-soluble dyes that temporarily stain the skin. Bronzers are easily removed with soap and water.

The prevalence of sunless tanner use in Australia has ranged from 9% to 22%[94]; 28% of women between 18 and 24 years of age reported using sunless tanners.[95] A survey of young adults 18 to 24 years of age in the United States revealed that 22% had used sunless tanners in the previous 12 months, and another 22% who had not used these products would consider doing so in the next 12 months.[94] Sunless-tanning-product users were more likely to be female, to be younger, and to report having sunburned during the previous summer than potential users or nonusers.

Dihydroxyacetone-induced tans become apparent within 1 hour; maximal darkening occurs within 8 to 24 hours. Most users report that color disappears over 5 to 7 days. Because neither dihydroxyacetone nor melanoidins afford any significant UV protection, consumers must be advised that sunburn and sun damage may occur unless they use sunscreen and other sun-protection methods. Consumers must also be warned that any sunless products that contain added sunscreen provide UVR protection only during the first few hours after application and that additional sun protection must be used during the duration of the artificial tan.

PREVENTION

The incidence of skin cancer continues to rise despite public health efforts to increase awareness of sun-safety measures. Children and teenagers continue to sunburn: in 1 large study of more than 10 000 white teenagers 12 to 18 years of age, most respondents (83% [$n = 8355$]) reported sunburning at least once, and 36% of children reported 3 or more burns during the previous summer.[72] Only one-third of respondents reported routine use of sunscreen during the past summer. Sunburning during the summer was reported in a nationwide survey of youth, although many reported using sunscreen before their most serious sunburn.[96] Among adolescents 16 to 18 years of age, the prevalence of sunburn and the average number of days spent at the beach increased between surveys conducted in 1998 and 2004.[97]

It has been estimated that sun avoidance could reduce the number of lifetime NMSC cases by almost 80%.[98] Although other risk factors (eg, precursor lesions, older age, race, previous melanoma, and family history) are more closely associated with melanoma than sunburns, exposure to UVR is the only risk factor that is avoidable. Leading organizations (the American Cancer Society,[99] Centers for Disease Control and Prevention,[100] Healthy People,[101] National Council on Skin Cancer Prevention[102]) have recommended sun-safe behaviors. UVR-protective messages include:

1. Do not burn; avoid suntanning and tanning beds.
2. Wear protective clothing and hats.
3. Seek shade.
4. Use extra caution near water, snow, and sand.
5. Apply sunscreen.
6. Wear sunglasses.

Clothing and Hats

Clothing can be an excellent UVR barrier, because it offers a simple and practical means of sun protection. In contrast to sunscreens, the photoprotection afforded by clothing does not diminish throughout the day unless the clothing becomes wet. Infants and children may be dressed in cool, comfortable clothing and wear hats with brims. One study revealed that wearing clothing decreases the development of nevi.[103] Protective factors in clothing include fabric type, thickness, color, and chemical enhancement.[2] Wool and synthetic materials such as polyester are more protective, whereas cotton, linen, acetate, and rayon are less protective. A tighter weave lets in less sunlight than a looser weave. Darker colors are more protective than lighter ones. Clothes that cover more of the body provide more protection; sun-protective styles cover to the neck, elbows, and knees. Treating fabrics with chemical absorbers or washing them with optical brighteners increases UVR protectiveness.

In 1996, Australia and New Zealand established standards for the UVR protectiveness of clothing. The United States developed standards in 2001. The ultraviolet protection factor (UPF) measures a fabric's ability to block UVR from passing through the fabric and reaching the skin. The UPF is classified from 15 to ≥50 as follows: 15 to 24 is rated as "good"; 25 to 39 is rated as "very good"; and 40 to ≥50 is rated as "excellent." Although garments with a UPF above 50 may be labeled "UPF 50+," these garments may not offer substantially more protection than those with a UPF of 50. Any garment

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with a UPF lower than 15 should not be labeled as "sun protective" or "UV protective."[104] Denim provides a UPF of 1700.[2] Typical summer cotton T-shirts provide a UPF of 5 to 9. The UPF of fabrics can be increased by shrinking and decreased by stretching. If cotton fabrics get wet, the UPF decreases. The US Federal Trade Commission monitors advertising claims about sun-protective clothing.[105]

Hats provide variable sun protection for the head and neck, depending on the brim width, material, and weave. A wide-brimmed (3-in) hat provides an SPF of 7 for the nose, 3 for the cheek, 5 for the neck, and 2 for the chin. Medium-brimmed (1- to 3-in) hats provide an SPF of 3 for nose, 2 for the cheek and neck, and none for the chin. A narrow-brimmed hat provides an SPF of 1.5 for the nose but little protection for the chin and neck.[2]

Shade

Infants younger than 6 months should be kept out of direct sunlight. Whenever possible, children's outdoor activities should be planned to minimize peak-intensity midday sun (10 AM to 4 PM). Seeking shade is somewhat useful, but people can still sunburn, because light is scattered and reflected. A fair-skinned person sitting under a tree can burn in less than an hour. Shade provides relief from heat and possibly provides a false sense of security about UVR protection. Clouds decrease UVR intensity but not to the same extent that they decrease heat intensity and, thus, may promote a misperception of protection.[6]

Sunscreen

Sunscreen is the main form of protection used by the population, including parents who use sunscreen to protect children.[106–109] Sunscreens reduce the intensity of UVR affecting the epidermis, thus preventing erythema and sunburn. Most FDA-approved sunscreen agents are organic chemicals that absorb various wavelengths of UVR, primarily in the UVB range; others are effective in the UVA range.[110] Some agents are not photostable in the UVA range and degrade with sun exposure. Combinations of chemicals are needed to provide broad-spectrum protection and increase photostability.[110]

The 2 FDA-approved inorganic physical sunscreens are zinc oxide and titanium dioxide, which prevent penetration of skin by UVB, UVA1, and UVA2. Physical sunscreens are usually white or tinted after application; some newer formulations are less visible on the skin but may be less effective.[110] Physical sunscreens are useful for people with photosensitivity disorders and other conditions that require protection from full-spectrum UVR.[3] Table 3 lists the FDA-approved sunscreen agents.

SPF is a grading system developed to quantify the degree of protection from erythema provided by using a sunscreen; the higher the SPF, the greater the protection. For example, a person who would normally experience sunburn in 10 minutes can be protected up to approximately 150 minutes (10 × 15) with an SPF-15 sunscreen. SPF pertains only to UVB. The SPF is determined indoors according to a standard protocol that uses artificial light sources and application of a defined amount of sunscreen (2 mg/cm^2). An SPF-2 sunscreen applied at this thickness blocks approximately 50% of UVB radiation; an SPF-10 blocks 90%; an SPF-15 blocks 94%; and an SPF-30

TABLE 3 FDA-Approved Sunscreens[110,115]

Sunscreen	Range of protection	Comments
Organic		
PABA derivatives	UVB	—
PABA[a]		
Padimate O (octyl dimethyl PABA)		
Cinnamates	UVB	—
Octinoxate (octyl methoxycinnamate)		
Cinoxate		
Salicylates	UVB	—
Octisalate (octyl salicylate)		
Homosalate		
Trolamine salicylate[a]		
Benzophenones	UVB, UVA2	Penetrates skin; estrogenicity in animal studies
Oxybenzone (benzophenone 3)		
Sulisobenzone (benzophenone 4)		
Dioxybenzone (benzophenone 8)[a]		
Others		
Octocrylene	UVB	In combination with other sunscreen agents, improves product photostability
Ensulizole (phenylbenzimidazole sulfonic acid)	UVB	—
Avobenzone (butyl methoxybenzoyl methane, Parsol 1789)	UVA1, UVA2	Photolabile; efficacy decreases by ~60% after 60 min of exposure
Ecamsule (terephthalylidene dicamphor sulfonic acid)	UVB, UVA2	Photostable; particularly effective for UVA2; approved by the FDA in 2007
Meradimate (menthyl anthranilate)[a]	UVA2	—
Inorganic		
Titanium dioxide	UVB, UVA2/UVA1	—
Zinc oxide	UVB, UVA2/UVA1	—

Note that other agents are approved for use in the European Union.
[a] These agents are rarely used in sunscreen formulations.

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blocks 97%. However, sunscreens block the pre-D_3 effective radiation more effectively than the erythemally effective radiation.[111]

In actual use, the SPF often is substantially lower than expected, because the amount applied to the skin is less than half the recommended amount (2 mg/cm^2).[112] To adequately cover all sun-exposed areas of an average adult wearing a bathing suit, 1 oz (30 mL) of sunscreen would be needed. It is recommended that sunscreen with an SPF of at least 15 be applied liberally 15 to 30 minutes before sun exposure to allow for absorption into the skin and to decrease the likelihood that the sunscreen will be washed off. Furthermore, it is recommended that sunscreens be reapplied every 2 hours and after swimming, sweating, or drying off with a towel.[110] Sunscreen products with a greater SPF provide somewhat greater protection. Products with a higher SPF have been recommended for some people, including those who have had skin cancer.[113] For most users, however, proper application and reapplication are more important factors than using a product with a higher SPF.

The formulating, testing, and labeling of sunscreen products is regulated by the FDA. The FDA has approved 17 sunscreen chemicals for use in the United States. Several more are available in the European Union. Four chemicals effective in the UVA range have been approved for use in the United States, although others are available in the European Union. In May 1999, the FDA published its final rule for over-the-counter sunscreen products that protect against UVB. Regulations concerning UVA were delayed until reliable testing methods could be developed. In August 2007, the FDA proposed new sunscreen regulations that focused on manufacturing, testing, and labeling of UVA sunscreens using a 4-star rating system that would rate product protection from "low" (1 star) to "highest" (4 stars).[114] The FDA also proposed that the "sun-protection factor" be changed to "UVB sunburn-protection factor."[115] The proposed grading system would divide sunscreens into 4 protection categories: low (SPF-2–SPF-15); medium (SPF-15 to lower than SPF-30); high (SPF-30–SPF-50); and highest (higher than SPF-50). Manufacturers would be unable to label their products with specific SPF values higher than 50, because the FDA believes that there are no data showing accuracy and reproducibility of SPF determinations higher than 50.[115] The proposed FDA rule had not been finalized as of February 2011.

The regular use of a broad-spectrum sunscreen preparation can prevent solar (actinic) keratoses, which are precursor lesions of SCC.[116,117] One randomized clinical trial revealed that sunscreen also decreases the risk of developing SCC.[118] The role of sunscreen in preventing BCC and melanoma has not been fully elucidated. No studies have demonstrated that sunscreen use prevents melanoma or BCC. Some research has revealed that sunscreen users have a higher risk of melanoma and BCC and more nevi.[103] These observations have led to concern that people who use sunscreens also spend more time in the sun because they do not sunburn.[119] The American College of Preventive Medicine found "insufficient evidence to recommend for or against sunscreen use. Nonmelanoma skin cancers may be reduced with regular, daily sunscreen use. There is insufficient evidence that chemical sunscreens protect against malignant melanoma and they may, in fact, increase risk."[120] Two reviews, however, did not support the association between sunscreen use and an increased risk of melanoma.[121,122] Sunscreen continues to be recommended by the American Academy of Dermatology[123] and many other organizations as part of a total program of sun protection.

Sunscreens may be systemically absorbed. In 1 study, sunscreen products were studied in vitro to assess the extent of absorption after application to excised human skin. Half of the products were marketed specifically for children. Of the 5 chemical sunscreen ingredients present in the products, only oxybenzone (benzophenone 3) penetrated skin.[124] In another report, researchers from the Centers for Disease Control and Prevention examined more than 2500 urine samples collected during 2003–2004 for oxybenzone. The samples selected were representative of the US population aged 6 years and older as part of the National Health and Nutrition Examination Survey (NHANES), an ongoing survey that assesses the health and nutritional status of the US civilian population. The analysis found oxybenzone in 97% of the samples,[125] which suggests widespread exposure to the population. Females and non-Hispanic white people had the highest concentrations regardless of age. Data are not available for children younger than 6 years.

Results of animal studies have shown alterations in liver, kidney, and reproductive organs in rats given oral or transepidermal doses of oxybenzone.[126] A study of 6 commonly used UVB and UVA sunscreens was conducted to determine estrogenicity in vivo and in vitro. Five of the 6 sunscreen ingredients (benzophenone 3, homosalate, 4-methyl-benzylidene camphor [4-MBC], octyl methoxycinnamate [OMC] and octyl-dimethyl-PABA) increased cell proliferation in breast cancer cells, and the sixth sunscreen ingredient, butyl-methoxydibenzoylmethane (avobenzone), was inactive. In the in vivo analysis, rats fed the sunscreen ingre-

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dients OMC, 4-MBC, and benzophenone 3 showed dose-dependent increases in uterine weight. Epidermal application of 1 of the products (4-MBC) also increased uterine weight.[127] Researchers investigating human prenatal exposures to phthalate and phenol metabolites and their relationship to birth weight found that higher maternal concentrations of benzophenone 3 were associated with a decrease in birth weight in girls but a greater birth weight in boys.[128] A study in young men and postmenopausal women given generous daily applications of benzophenone 3, OMC, and 4-MBC revealed detectable levels of these chemicals in plasma and urine.[129] There were no effects on serum concentrations of reproductive hormones related to sunscreen exposure in men or women. The authors concluded that although the data showed skin penetration of these sunscreen chemicals, there did not seem to be an effect on the hypothalamic-pituitary-gonadal axis. Caution, however, was suggested for children. Researchers in Europe investigated analyzed samples of human milk for the presence of sunscreens and other chemicals with possible endocrine activity. Mothers were asked about their use of sunscreens and cosmetics that contained sunscreen ingredients (benzophenone 2, benzophenone 3, 3-benzylidene camphor, 4-MBC, OMC, homosalate, octocrylene, and octyl-dimethyl PABA). Responding to questionnaires, 78.8% of the women reported using products that contained sunscreens; 76.5% of human milk samples contained these chemicals. There was a high correlation reported between mothers' use of these chemicals and their concentrations in human milk. The authors concluded that except for lipsticks (the ingestion of which is probably important), their results agree with studies in animals and humans showing dermal absorption of sunscreens. Given that some of these chemicals have endocrine activity in animals, the authors suggested that exposure could be lessened if mothers abstained from using these products during their children's sensitive life stages.[130]

Sunscreens are lipophilic and can bioaccumulate in the environment. Sunscreen ingredients have been identified in fish.[127] Because of recent data on bioaccumulation in humans and wildlife, researchers have called for an in-depth analysis of the systemic toxicology of sunscreen ingredients.[127] Sunscreen ingredients are not listed as known or suspected human carcinogens.[131]

Sunscreen products that contain zinc and titanium oxides are increasingly manufactured by using nanotechnology—the design and manipulation of materials on atomic and molecular scales. Nanoscale particles are measured in nanometers, or billionths of a meter. Using nanoscale particles renders products that contain zinc and titanium oxides nearly transparent and increases cosmetic acceptability. Concerns have been raised, however, about the dearth of safety information available about nanoscale ingredients, including the effect on skin that is damaged by sunburn.[132] There are no data available about the effects of these products on infants and children. Advocacy groups have called on the FDA to require more testing and increased regulatory oversight.

To our knowledge, toxicity in infants and children from absorption of sunscreen ingredients has not been reported. Permeability of skin to topically applied products is, however, of concern for infants and young children, especially preterm infants, in whom the stratum corneum of the epidermis is thinner and a less effective barrier than that of term newborn infants and adults. Well-known toxicity from percutaneous absorption in infants and children include the adverse effects of alcohol, boric acid (in diaper powder), hexachlorophene (in antiseptic cleansers), and mercuric chloride (in diaper rinses).[133] Risks from cutaneous exposure to environmental toxicants and chemicals may be heightened in children compared with adults for reasons that include differing behavior patterns; anatomic and physiologic differences in absorption, metabolism, distribution, and excretion; and developmental differences of vital organs that may result in different end organ effects.[133] Infants have a greater ratio of surface area to body weight compared with older children and adults, which allows infants to percutaneously absorb proportionately greater quantities of topical medications or other preparations.[134]

The development of barrier function of infant skin has been investigated. The skin barrier limits water loss, protects the body from entry by toxic substances, and resists mechanical trauma.[135] Vernix caseosum provides a barrier during fetal life. Once the vernix is removed after birth, the stratum corneum of the epidermis provides protection. It was previously thought that the stratum corneum assumed adult function in the first few weeks of life. Accumulating research suggests, however, that the stratum corneum continues to develop through the early years of life. One study[136] assessed the dynamic transport and distribution of water in the stratum corneum in infants (3–12 months of age) and adults (14–73 years of age) by measuring transepidermal water loss (TEWL) ("insensible water loss," a measure of the amount of water that passively diffuses through the epidermis), capacitance (a measure of skin hydration), rates of absorption and desorption, and concentration of water and natural moisturizing factor (NMF) in the skin. Infants' skin had greater hydration, greater TEWL, greater water ab-

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sorption and desorption, and lower concentration of NMF. NMFs normally take up water; lower levels may contribute to faster water desorption, which possibly affects barrier function. The authors concluded that the unique properties of infant skin continue to persist at least through the first 12 months of life. In an Italian study,[137] TEWL, capacitance, and pH were measured in 70 infants (8–24 months of age) without skin disease and 30 healthy adult women (25–35 years of age). TEWL measurements did not differ between the infants and adults. Capacitance values and pH were higher in infants. The authors concluded that, despite the similarities in TEWL, differences found in capacitance and pH indicated functional immaturity, possibly resulting in increased permeability.

Infrequently, topical sunscreen agents can have adverse effects, including erythema, itching, burning, or stinging. Allergic contact dermatitis and photoallergic and phototoxic reactions occur rarely.[110]

It is generally recommended that infants younger than 6 months be kept out of direct sunlight. The Australasian College of Dermatologists recommends the use of a sunscreen for infants when exposure to the sun cannot be prevented by other avoidance measures: "Shade, clothing and broad rimmed hats are the best sun protection measures for infants. Sunscreens should be applied to areas of the skin not protected by clothing."[138] The American Academy of Pediatrics (AAP) has stated that sunscreen may be used on infants younger than 6 months on small areas of skin if adequate clothing and shade are not available.[139]

Sunscreens may increase absorption of the insect repellent DEET (*N,N*-dimethyl-metatoluamide), especially when DEET is applied first.[140] Products that combine a sunscreen agent with an insect repellent such as DEET are available. Using individual DEET and sunscreen products at the same time is an acceptable practice, but the use of combination products is not recommended. A sunscreen should be reapplied after swimming or sweating, whereas insect repellent generally does not need to be reapplied.[141] Furthermore, concerns have been raised about potential toxicity of percutaneously absorbed repellents in children, especially with repeated application.[142]

Window Glass

Standard clear window glass absorbs wavelengths below 320 nm (UVB). UVA, visible light, and infrared radiation are transmitted through standard clear window glass. Large window areas are now commonly part of residential and commercial architectural design. Modern windows increasingly incorporate energy-efficient glazes that decrease heat gain and loss through windows. Many of these energy-efficient glazes provide some UVR protection, but only a few provide full UVR protection.[143]

Transmission of UVR through automobile glass depends on the type and the tint of the glass. Because of safety reasons, all windshields are made of laminated glass, a product made stronger through bonding with a tough, clear plastic. Laminated glass filters out most UVA. Side and rear windows, however, are usually made from nonlaminated glass, which allows significant UVA exposure, especially UVA1. Tinted glass removes more UVA than does clear glass; it is possible for automobile owners to add tinted window films to side and rear windows to reduce transmission of light, UVR, and heat.[143] The parts of a driver's or passenger's body closest to a window receive the most radiation. Individuals with photosensitivity disorders can experience exacerbations of their disease while riding in a car.[143] Most states do not allow plastic films with less than 35% visible light transmittance. The minimum allowable visible light transmission levels for side and rear windows are determined by each state[143] and are available from the International Window Film Association.[144]

It has been hypothesized that the increase in melanoma in indoor workers (but not outdoor workers) during the last century may be a result of their exposure to UVA passing through windows.[145] These researchers agree that overexposure to UVB initiates melanoma but that increased UVA exposures (which can cause mutations and break down cutaneous vitamin D_3) and low vitamin D_3 concentrations in the skin promote melanoma.

Sunglasses

Sunglasses protect against sun glare and harmful radiation. The first sunglass standard was published in Australia in 1971; standards were subsequently adopted in Europe and the United States. The latest US sunglass standard was published in 2001 by the American National Standards Institute. This standard is voluntary and is not followed by all manufacturers.[143]

Major US visual health organizations recommend that sunglasses that absorb 97% to 100%[146] or 99% to 100%[59] of the full UV spectrum (up to 400 nm) should be worn. Expensive sunglasses do not necessarily provide better UVR protection. Purchasing sunglasses that meet standards for a safe level of UVR should be the goal. Wearing a hat with a brim can greatly reduce the UVR exposure to the eyes and surrounding skin. It is recommended that people wear sunglasses outdoors when working, driving, participating in sports, taking a walk, or running errands.[147]

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Sunglasses for infants and children are available.

The UV Index

The UV index was developed in 1994 by the National Weather Service in consultation with the US Environmental Protection Agency and the Centers for Disease Control and Prevention. The UV index predicts the intensity of UV light for the following day on the basis of the sun's position, cloud movements, altitude, ozone data, and other factors.[148] It is conservatively calculated on the basis of effects on skin types that burn easily. Higher numbers predict more intense UV light during midday of the following day: 0 to 2, minimal; 3 to 4, low; 5 to 6, moderate; 7 to 9, high; and 10 or higher, very high. Sun-protection strategies should be applied at even minimal levels of the UV index, and increasing stringency should be used as the UV index increases (eg, avoiding outdoor exposures from 10 AM to 4 PM if the UV index is 7 or higher). The index is available online for thousands of cities at www.weather.com. It is printed in the weather section of many daily newspapers and reported through weather reports of local radio, television, and weather stations. The UV index can be used to plan outdoor activities.

VITAMIN D

Sun exposure and vitamin D concentrations are intricately intertwined. Thus, effects of limiting sun exposure on vitamin D status must be understood and addressed.

Metabolism

Humans get vitamin D from exposure to sun, dietary sources (such as fortified milk and oily fish), and vitamin supplements. After sunlight exposure, 7-dehydrocholesterol in the skin is converted to previtamin D_3; previtamin D_3 is then converted to vitamin D_3 (cholecalciferol). Vitamin D from the skin and diet is metabolized primarily in the liver to 25-hydroxyvitamin D (25[OH]D), which is used to determine a patient's vitamin D status; 25(OH)D is metabolized in the kidneys to its active form, 1,25-dihydroxyvitamin D, also known as calcitriol.

Vitamin D synthesis in skin depends on skin type. A person with skin type I who burns easily after a first moderate UVR exposure will rapidly achieve maximal vitamin D synthesis. In contrast, a person with skin type VI will have relatively limited vitamin D synthesis, because UVR will be absorbed by melanin rather than other cellular targets.[149] Because excess previtamin D_3 or vitamin D_3 is destroyed by sunlight, exposure to sunlight does not cause vitamin D intoxication.[150] The action spectrum that induces cutaneous vitamin D_3 synthesis is in the UVB range.[151]

Vitamin D Health Effects

Vitamin D is essential for normal growth and skeletal development.[152] At a 25(OH)D concentration lower than 50 nmol/L (<20 ng/mL), children are at increased risk of developing rickets[153]; concentrations below this amount are considered to be deficient. In adults, a 25(OH)D concentration of 80 nmol/L (32 ng/mL) is generally recognized as the threshold of an optimal level, and a concentration of 50 to 79 nmol/L is considered "insufficient."[150,154,155] The AAP recommends that pregnant women maintain a 25(OH)D concentration of 80 nmol/L or higher.[156]

The benefits of vitamin D in adults are many and include improved bone health, prevention of fractures, better muscle health, and reduced risk of falling in older people.[150] The actions of vitamin D that extend beyond bone mineral metabolism are increasingly being understood. Many human tissues, including brain, prostate, breast, and colon, as well as immune cells, have vitamin D receptors, and some have enzymes capable of producing 1,25-dihydroxyvitamin D from circulating vitamin D.[157] 1,25-Dihydroxyvitamin D controls more than 200 genes, including those responsible for regulating cellular proliferation, differentiation, apoptosis, and angiogenesis.[150] These cell-regulation actions are found at 25(OH)D concentrations higher than 75 nmol/L.[152]

Relationships between 25(OH)D status and markers of functional outcomes in children and adolescents vary according to age, race, environment, and genetic predisposition.[153,158,159] A study of bone health in Australian adolescent boys identified a 25(OH)D concentration of at least 43 to 55 nmol/L as optimal.[160] Several studies reviewed by Wagner et al[156] noted associations of low 25(OH)D concentration with increased parathyroid hormone and reduced bone metabolism. Hypovitaminosis D may reduce maximum peak bone mass in pubertal girls.[161] A subsequent study supported these associations for girls,[162] but another study reported a larger gain in bone area and bone mineral content for children with lower 25(OH)D concentration.[163] In a randomized controlled trial, vitamin D supplementation in girls 10 to 17 years of age was associated with higher lean mass and higher bone mineral content.[164] Authors of a study of girls 11 to 15 years of age reported calcium absorption to be unrelated to 25(OH)D status and to vary according to race; relationships between 25(OH)D status and parathyroid hormone were also found to vary according to race.[165] In observational studies (ie, studies in which people are observed or certain outcomes are measured), higher intakes of vitamin D via food in pregnancy and supplementation in infancy were associated with a lower risk of type 1 diabetes in children.[166,167]

Ecologic studies have revealed a lower incidence of breast, colon, and prostate cancers in areas of higher sun ex-

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posure.[150,168–170] A recent meta-analysis of observational studies provided evidence of a decreased risk of colorectal cancer and colorectal adenoma associated with higher serum 25(OH)D concentrations.[171] The analysis also found nonsignificant reduced risk of breast cancer and no evidence for association of vitamin D and prostate cancer. Two double-blind placebo-controlled randomized trials with vitamin D supplementation at 400 IU (10 μg)/day failed to demonstrate effects on colorectal[172] or breast cancer[173] incidence, which suggests that ecologic studies may not adequately control for confounding variables[171] or that vitamin D supplementation needs to be substantially higher to achieve a protective effect.[168,171] Low vitamin D status also is directly associated with increased all-cause mortality; reasons for this association merit further examination.[171]

Ecologic studies have revealed a lower risk of multiple sclerosis in areas of high sun exposures[174]; dietary vitamin D lowers risk of multiple sclerosis,[175] possibly through regulating genetic susceptibility.[176] People with low vitamin D concentrations are at higher risk of insulin resistance and the metabolic syndrome,[177] future abnormal glucose regulation,[178] periodontal disease,[179] diminished heart health,[180] and other conditions. To date, however, the relationships between vitamin D supplementation and cancer risks have not been evaluated by randomized controlled trials providing vitamin D at doses that would be high enough to achieve 25(OH)D concentrations similar to those thought to provide protective effects. Until such data are available, questions will remain about the influence of confounding factors (eg, diet, latitude, skin melanin) on previously reported cancer-related outcomes. New randomized controlled trials are needed to guide the development of guidelines for vitamin D supplementation in relationship to cancer prevention[171] and other outcomes.

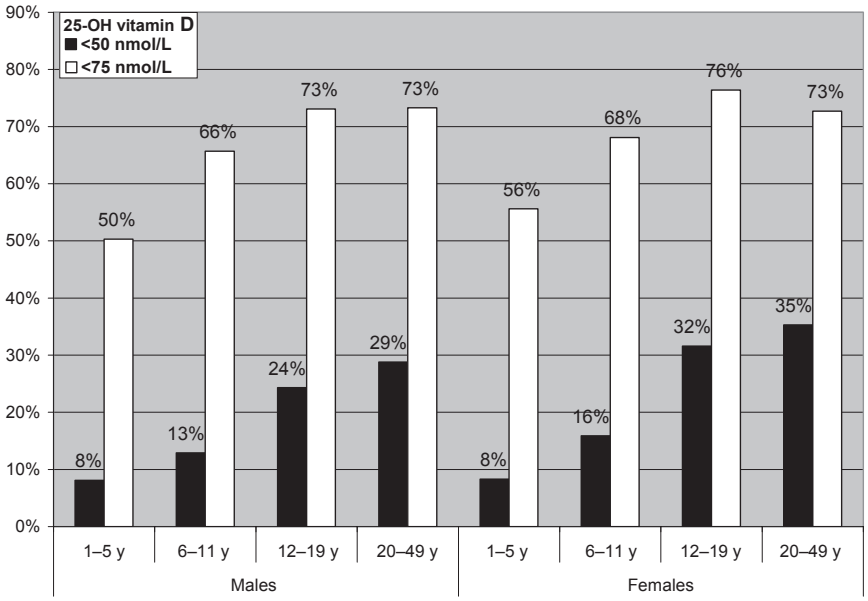

FIGURE 1
Prevalence of serum 25(OH)D concentrations below selected thresholds according to age and gender: NHANES 2000–2004.[183]

Prevalence of Hypovitaminosis D

Hypovitaminosis D is common among US children.[181,182] Data from the 2000–2004 NHANES indicate that approximately 30% of US teenagers and young adults have 25(OH)D deficiency (ie, 25[OH]D $<$ 50 nmol/L), as do approximately 15% of children 6 to 11 years of age and 8% of children 1 to 5 years of age (Fig 1).[183] Because NHANES data are collected in the South in winter months and the North in the summer, these national data may underestimate seasonal variations in 25(OH)D concentrations. The prevalence of hypovitaminosis D is high among samples of women in many areas including Australia,[184] Bangladesh, and Hong Kong, where cutaneous vitamin D synthesis can take place year-round.[185,186]

Risk Factors for Vitamin D Deficiency

Risk factors for hypovitaminosis D in US youth include increasing age, low vitamin D intake, dark skin, winter season, and higher BMI.[181] 25(OH)D deficiency is common in populations such as infants born to mothers at high risk of hypovitaminosis D; breastfed infants; children with sickle cell disease, type 1 diabetes, malabsorption, or obesity; and those who take medications such as anticonvulsants or glucocorticoids. In US non-Hispanic white adults, 25(OH)D concentrations were 5 to 9 nmol/L higher in 1988–1994 compared with 2000–2004; increasing BMI, decreased milk intake, and increased use of sun-protection methods between study periods seemed to be factors accounting for approximately 20% of the difference.[183]

Sun-Exposure Considerations

Many factors influence the efficiency of vitamin D production resulting from sunlight exposures. The amount of skin exposed to the sun results in differences in vitamin D synthesis. In a 1985 study of young infants in Cincinnati, Ohio, who were fully clothed (without hats) and exclusively breastfed, it was determined that 2 hours of sun expo-

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sure weekly were needed to maintain 25(OH)D concentrations higher than 27.5 nmol/L, compared with infants who were wearing only a diaper, for whom only 30 minutes/week of sun exposure were required.[187]

It has been stated that at least 20% of the body surface needs to be exposed to UVB for vitamin D concentrations to increase.[152] Dark-skinned people require exposures approximately 5 to 10 times as long as do light-skinned people to achieve similar levels of cutaneous vitamin D production.[188] At latitudes above 35°N (eg, north of Memphis, TN; Kyoto, Japan; and Cyprus) and below 35°S (eg, south of Adelaide, South Australia; and Montevideo, Uruguay), UVB photons do not penetrate to the earth's surface in winter months, which makes cutaneous vitamin D production negligible in those months. Because of the scatter of UVB, sun exposure outside the peak sun hours of 10 AM to 3 PM in the spring, summer, and fall has limited impact on cutaneous vitamin D synthesis.[188] It has been stated that brief exposures to high overhead sunlight have maximum vitamin D benefit with most limited erythema risk.[111]

One author has recommended that children and healthy adults practice "sensible sun exposure" (ie, exposure of arms and legs for 5–30 minutes, depending on the time of day, season, latitude, and skin pigmentation, between 10 AM and 3 PM twice weekly) as a way to maintain vitamin D concentrations and avoid deficiency.[150] This same author recommended that, after such exposure, a sunscreen with an SPF-15 or greater can be applied if the person wishes to remain outdoors.[189] In contrast, the American Academy of Dermatology has stated that maximum production of vitamin D occurs after only brief exposure to UVR; this amount of time is 2 to 5 minutes of midday summer exposure for a light-skinned person living in New York, New York, or Boston, Massachusetts. Although leaders in skin-cancer prevention agree that vitamin D is important for good health, they oppose intentional sun exposure to induce vitamin D production, because UVR is a known human carcinogen.[151,190] There are no studies of children suggesting a level of sun exposure that would negate the need to comply with dietary vitamin D recommendations. Thus, use of deliberate sun exposure to maintain vitamin D sufficiency is not recommended. Given the high prevalence of hypovitaminosis D, however, it seems clear that renewed attention must be paid to evaluating the adequacy of dietary and supplemental vitamin D intake and how much, if any, unprotected sun exposure is beneficial. It is important to keep in mind that infants younger than 6 months should be kept out of direct sunlight as much as possible.

Dietary and Supplemental Vitamin D Recommendations

Many children get less than 400 IU (10 μg) of vitamin D daily from their diets. Approximately 22% of US children 1 to 8 years of age, 50% of girls 9 to 18 years of age, and 35% of boys 9 to 18 years of age get less than 200 IU (5 μg) of vitamin D daily from food or supplements.[191] No primary care–based studies have examined the sensitivity of evaluations of dietary vitamin D intake or sunlight-exposure assessments to determine vitamin D adequacy in children.

The main source of vitamin D is exposure to sunlight,[192–195] which makes it difficult to establish a dietary requirement that has broad generalizability, especially because of the many variables (eg, skin pigmentation, body mass, season, outdoor exposure, clothing, sunscreen use, etc) associated with differences in vitamin D concentrations.[156] In a 1997 report, the Institute of Medicine recommended an adequate intake level for vitamin D of 200 IU/day.[196] An updated report on vitamin D from the Institute of Medicine was released in November 2010.[197] However, because 200 IU/day of vitamin D is insufficient to maintain a 25(OH)D concentration higher than 50 nmol/L, the AAP and others currently recommend that exclusively and partially breastfed infants receive supplements of 400 IU/day of vitamin D shortly after birth and continue to receive these supplements until they are weaned and consume 1000 mL/day or more of vitamin D–fortified formula or vitamin D–fortified milk.[152,156] This level of supplementation in a breastfed infant will generally achieve a 25(OH)D concentration of more than 70 nmol/L and prevent vitamin D deficiency rickets.[151] All nonbreastfed infants who ingest less than 1000 mL/day of vitamin D–fortified formula should receive a vitamin D supplement of 400 IU/day. Formulas for term infants sold in the United States generally provide approximately 400 IU of vitamin D per L, and the majority of vitamin D–only and multivitamin liquid supplements provide 400 IU per dose. The AAP also recommends that older children and adolescents who do not obtain 400 IU/day through vitamin D–fortified milk and foods should take a 400-IU vitamin D supplement daily. The effect of such routine supplementation on 25(OH)D status in children and adolescents has not yet been evaluated. The extent to which 25(OH)D increases with supplementation will vary depending on the amount of vitamin D synthesized over the summer months and the dosage and duration of use.[198] Children at high risk of vitamin D deficiency, such as those with chronic fat malabsorption and those who chronically take antiseizure medications, may need vitamin D doses higher than 400 IU/day,[156] and studies that have tested supplementation in special populations have used

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much larger doses safely.[199,200] Treatment of hypovitaminosis D in infants and toddlers is safely done with 2000 IU of vitamin D daily for 6 weeks.[201]

Influence of Vitamin D Supplementation

Several studies have evaluated the influence of the amount of vitamin D supplementation on 25(OH)D levels. In adults, daily supplementation with 400 IU of vitamin D increases 25(OH)D concentration by 7.0 nmol/L.[202] Supplementation of a pregnant woman with 400 IU of vitamin D, as in prenatal vitamins, has little effect on her 25(OH)D concentration.[160] For adult men at latitude 41°N, the amount of supplemental vitamin D (in addition to dietary vitamin D) needed to maintain baseline 25(OH)D concentration over the winter was 500 IU/day.[202] In a sample of young, healthy adults living at ≥51° latitude, the vitamin D supplemental requirement (in addition to dietary vitamin D) for 97.5% of the sample to maintain a 25(OH)D concentration higher than 25 nmol/L was 348 IU of vitamin D per day; the supplemental dose of vitamin D required to maintain a 25(OH)D concentration higher than 50 nmol/L for 97.5% of the sample was 1120 IU of vitamin D per day and was 1644 IU of vitamin D per day to maintain a 25(OH)D concentration higher than 80 nmol/L.[203] The Institute of Medicine report released in November 2010 provided an extensive review of the effects of vitamin D supplementation.[197]

Influence of Sun Protection on 25(OH)D

A few studies of adults, but none of children, have examined associations with sun protection or sunscreen use and 25(OH)D concentrations. Higher use of sun protection was associated with statistically significantly lower 25(OH)D concentrations in non-Hispanic white adult subjects in the 2000–2004 NHANES, compared with similar subjects in the 1988–1994 NHANES.[183] Other studies of adults found expected relationships between reported levels of sun exposure and 25(OH)D concentration but no association between reported sunscreen use and 25(OH)D concentration.[204,205] Authors of a small, controlled trial that involved 24 elderly adult sunscreen users and 19 controls over 2 years reported that lower 25(OH)D concentration in sunscreen users did not result in increases in parathyroid hormone or increases in bone biological markers.[206] Sunscreen users generally apply insufficient amounts to meet the expected SPF level. Sunscreen efficacy also depends on uniform application to exposed body parts, the sunscreen's durability and substantivity (a measure of the sunscreen's ability to be adsorbed by or adhered to the skin while swimming, bathing, or perspiring), and reapplication. Thus, evaluation of sunscreen use without also considering other sun-protection measures may not accurately indicate risk of low 25(OH)D status.

PEDIATRICIAN COUNSELING

Pediatricians can play important roles in counseling about sun protection. In a 2003 report, the US Preventive Services Task Force (USPSTF) determined that clinician counseling may have an effect on parents' use of sunscreen for their children but not for using other sun-protection measures such as wearing protective clothing, reducing excessive sun exposure, avoiding sunlamps/tanning beds, or practicing skin self-examination. The USPSTF noted that only limited data exist about potential harm of counseling or of specific skin-protection behaviors.[207] Harm could include the possibility that focusing on sunscreen use may result in a false sense of security and more time spent in the sun because users do not sunburn.[119] Other harmful outcomes include the possibility that vitamin D deficiency results from sunscreen use; according to the USPSTF, a randomized controlled trial in people older than 40 years found that sunscreen use over the summer had no effect on 25(OH)D concentrations.[207] There are concerns that sun avoidance may result in reduced physical activity levels among children and negative effects on mental health; there have been no studies regarding the effects of protection behaviors on these outcomes.[207]

In a survey of children's caregivers attending a university-based clinic in Florida, only 30% of caregivers reported having been counseled by their physician about sun protection. Caregivers who were counseled had greater sun-protection knowledge, were more likely to report regular use of sun protection for their child, and were more likely to report teaching their child about sun protection.[208] In surveys of Massachusetts and Texas pediatricians, approximately three-quarters of them indicated that they recommended safe sun practices or sunscreen use to a majority of their patients. However, the messages they presented included a limited number of the available sun-protection strategies.[209,210] Counseling regarding sun protection is prioritized lower than counseling on other safety issues.[209,211] Time constraints are often mentioned as a main barrier to providing counseling.[211] A "teachable moment" may arise when the child or adolescent presents with a sunburn.

EARLY DETECTION

The US Preventive Services Task Force concluded that evidence is insufficient to recommend for or against routine screening for skin cancer in adults by using a total-body skin examination for the early detection of cutaneous melanoma, BCC, or SCC in people without a

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history of skin cancer or otherwise at high risk.[212] However, because early detection increases survival rates,[213,214] it has been recommended that complete cutaneous examinations be performed by physicians and other health care providers, coupled with periodic self-examination of the skin by the individual person.[214] Skin lesions with malignant features noted in physical examinations should be biopsied. There are no recommendations on skin-cancer screening in children. Because melanoma occurs in teenagers and is a common cancer among young adults, it seems prudent to recommend that clinicians caring for these groups include a skin examination as part of a complete physical examination.

PREVENTION IN SCHOOLS

The Centers for Disease Control and Prevention has published guidelines to protect schoolchildren from excessive sun exposure in schools. Recommendations include reducing skin-cancer risks through policies; creation of physical, social, and organizational environments that facilitate protection from UVR; education of young people; professional development of staff; involvement of families; work by nurses and other school health services staff; and program evaluation.[100] Authors of a systematic review published in 2004 (search updated to June 2000) concluded that efforts to teach children how to protect themselves from UVR were effective when implemented in primary schools and in recreational settings. There was insufficient evidence, however, about the effectiveness of implementation in other settings.[215]

Schools have a role in determining children's attitudes and behaviors. The SunWise Program, developed by the Environmental Protection Agency, is a brief, standardized sun-protection education program.[216] It is the first environmental education program for sun safety designed to teach children in elementary and middle schools (and their caregivers) how to protect themselves from overexposure to the sun. The SunWise program has been shown to promote improvement in knowledge, intentions to play in the shade and to use sunscreen, and attitudes regarding healthiness of a tan.[217] A recent study of the SunWise program demonstrated that every federal dollar invested in it generates $2 to $4 in public health benefits.[218]

COMMUNITY-BASED PROGRAMS

Multicomponent community-wide approaches have been recommended by health education experts[219] and can be effective. Several community-directed campaigns have addressed sun protection in younger children. A randomized controlled trial of the SunSafe project, an intervention in New England, involved schools, child care settings, primary care offices, and beach settings. The SunSafe program was effective in changing sun-protection practices observed at community beaches for children 2 to 10 years of age.[220,221]

Use of sun-protection practices begins to decline in early adolescence[222] as media and peer influences on teen attitudes and behaviors increase and parental influences decrease.[223] A randomized controlled trial of the SunSafe program was conducted in 5 intervention and 5 control communities to assess the impact of a sun education program in the middle school years. The SunSafe in the Middle School Years program augmented the original program by involving sports teams and peer-led activities. After 2 years of intervention, adolescents in intervention communities had less of the expected deterioration in sun-safety practices compared with adolescents in control communities.[224]

Other interventions were not effective. Australian adults who received solar UV forecasts and supporting communications did not implement markedly enhanced personal sun-protection practices.[225] A randomized trial of an educational intervention to reduce sunburn rates and improve sun-protection behavior in schoolchildren showed no difference in sunburn episodes between the study and control groups.[226]

PUBLIC HEALTH CAMPAIGNS

Australia, the country with the highest incidence of skin cancer in the world, has been in the forefront of the public health response to this disease. SunSmart, a population-based skin-cancer–prevention program run by the Australian state of Victoria since 1988, incorporates substantial public education efforts as well as structural and environmental change strategies in schools, workplaces, local government settings, and pools. Paid television advertising has been part of a strategy of public education. The authors of a recent assessment of the SunSmart program concluded that sun-protection methods and rates of sunburn showed substantial general improvement over time but stalled in recent years. Most initial gains were sustained over 15 years of assessments, but there was no further progress with regard to sunburn, sunscreen use, body exposure, and attitudes.[227]

In a 2008 editorial, Martin Weinstock, MD, an internationally known dermatologist and researcher, concluded that data suggest that public health efforts at skin-cancer prevention are inadequate.[228] Four challenges to effective skin-cancer prevention campaigns were identified. First, sun-protection messages to avoid or limit time during peak sun hours may conflict with health-promotion messages regarding physical activity. This potential conflict may be resolved by following the "slip, slop, slap" motto of the Australians to slip on a shirt, slop on sunscreen, and slap on a hat—a message consistent with conducting outdoor physical activity in a sun-protective manner. Next, there is controversy about how much

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sun exposure is needed for vitamin D synthesis, which possibly results in excessive exposure to sun and deliberate exposure to artificial UVR. Third, it has been reported that skin-cancer risk behaviors cluster with other risky behaviors, such as smoking and risky drinking. A greater understanding of these behaviors may help with interventions. Fourth, the increasingly profitable tanning industry benefits from unrestrained selling of UVR. These challenges suggest that it is uncertain whether primary prevention efforts to reduce skin cancer through UVR protection will be successful.

RESEARCH NEEDS

Outstanding research questions exist in many areas, including relationships of sunscreen use to melanoma and BCC; safety of absorbed ingredients in sunscreens; effects of long-term use of sunscreen, especially when this practice begins early in life; the role of vitamin D in preventing cancer and other health conditions; the relationship of 25(OH)D to functional outcomes for children; developing and assessing strategies for estimating vitamin D status during a clinical encounter; determining how much sun exposure and vitamin D supplementation is "enough" depending on a person's age and gender, his or her geographic location, the season of the year, and other factors; effects of long-term use of vitamin D supplementation at various levels; and utility of routine counseling on sun-avoidance strategies in clinical encounters.

CONCLUSIONS

UVR is a known human carcinogen and has numerous other adverse health effects. Skin-cancer rates have reached epidemic proportions, and skin cancers occur in young people and sometimes result in death. Excessive exposure to UVR during childhood and adolescence is thought to confer an increased risk of developing skin cancer. Morbidity and deaths from skin cancer are preventable. Pediatricians may play an important role in providing education about skin-cancer prevention to patients and their parents, yet many do not take opportunities to do so. Pediatricians are urged to provide advice on hundreds of topics,[229] so it may be impractical to expect pediatricians to discuss skin-cancer prevention and sun protection during every health-maintenance visit. It is, however, reasonable to expect that skin-cancer prevention be discussed on at least a few visits during the course of a pediatrician's long-term relationship with a child and his or her family. Because parents' comprehensive sun-protection practices for children start to decline when children are very young,[230] it is important to begin discussions early in the child's life. Discussions are especially important for children at high risk of developing skin cancer—children with light skin, those with nevi and/or freckling, and those with a family history of melanoma. Melanoma is rare in children, but moles are not rare. Education can include a discussion of moles and the need to be aware of changes in them. As children approach puberty, it is important to include information about the dangers of artificial tanning. Pediatricians also have an important role as advocates in helping to support legislation to ban minors' access to tanning salons.

Lifelong sun protection is recommended beginning at an early age. Although sunscreen is the most commonly used method of sun protection, patients should be counseled to not overly rely on sunscreen. A complete program of sun protection includes wearing clothing and hats, timing activities to minimize peak hours of the sun, and wearing sunglasses. Advice should be framed in the context of promoting regular outdoor play and other physical activity.

Vitamin D is available through foods, supplements, and incidental sun exposure. Because current intake levels of vitamin D by children and adolescents may not prevent vitamin D deficiency, it is recommended that all infants, children, and adolescents receive 400 IU of vitamin D per day. Additional vitamin D supplementation and laboratory evaluations of vitamin D status may be needed for some children in some areas. Overexposure to UVR from sunlight and exposure to UVR from artificial sources raise the risk of skin cancer, photoaging, and other adverse effects and should be avoided.

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Sophie J. Balk, the Council on Environmental Health and Section on Dermatology

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