

Ultraviolet radiation and skin cancer

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Financial Disclosure: None.
Conflict of Interest: None.

Abstract

Skin cancer is the most common type of cancer in fair-skinned populations in many parts of the world. The incidence, morbidity and mortality rates of skin cancers are increasing and, therefore, pose a significant public health concern. Ultraviolet radiation (UVR) is the major etiologic agent in the development of skin cancers. UVR causes DNA damage and genetic mutations, which subsequently lead to skin cancer. A clearer understanding of UVR is crucial in the prevention of skin cancer. This article reviews UVR, its damaging effects on the skin and its relationship to UV immunosuppression and skin cancer. Several factors influence the amount of UVR reaching the earth's surface, including ozone depletion, UV light elevation, latitude, altitude, and weather conditions. The current treatment modalities utilizing UVR (i.e. phototherapy) can also predispose to skin cancers. Unnecessary exposure to the sun and artificial UVR (tanning lamps) are important personal attributable risks. This article aims to provide a comprehensive overview of skin cancer with an emphasis on carefully evaluated statistics, the epidemiology of UVR-induced skin cancers, incidence rates, risk factors, and preventative behaviors & strategies, including personal behavioral modifications and public educational initiatives.

Burden of skin cancer

Skin cancer is the most common type of cancer in light skinned populations around the world.¹ Skin cancers are mainly divided into melanoma, and nonmelanoma skin cancers (NMSCs), the latter including basal and squamous cell carcinomas (BCC and SCC, respectively). Melanoma is responsible for most of the cancer related mortalities, and NMSCs are typically described as having a more benign course with locally aggressive features. Nevertheless, they represent “the most common type” of cancer in humans and they can result in significant disfigurement, leading to adverse physical and psychological consequences for the affected patients.²

Nonmelanoma skin cancers

It is estimated that 2–3 million cases of NMSCs occur worldwide each year.^{3,4} The incidence varies with very high rates in the Caucasian populations of the world.² For incidence, the overall upward trend observed in most parts of Europe, Canada, USA and Australia shows an average increase between 3% and 8% a year.¹ The incidence of NMSCs is over 1.3 million cases each year in the U.S.; in fact, this incidence rate is “expected to double in the next 30 years.”⁵ Approximately 30% of all newly diagnosed cancers in the U.S. are BCC, making it the most commonly diagnosed cancer in this country.⁶

Basal cell carcinoma, which accounts for 80–85% of all NMSCs, rarely metastasizes to other organs.² It is the

most common malignancy in white people. Its worldwide incidence is increasing by up to 10%, “with highest rates in elderly men and increasing incidence in young women.”⁷ Although mortality is low, this malignancy causes considerable morbidity and places a huge burden on healthcare systems worldwide.⁷ SCC, which accounts for 15–20% of all NMSCs, is more likely to invade other tissues and can cause death.²

As a result of the benign nature of NMSC characteristics, some patients may remain unregistered and undiagnosed, leading to an under-representation of the number of cases.² Moreover, as NMSCs have localized symptoms and primarily manifest in older individuals, they may remain undiagnosed.

Basal cell carcinoma and SCC are usually found in sun-exposed areas, especially the head and neck regions.^{8,9} They are both positively related to the amount of ultraviolet radiation (UVR) received and inversely proportional to the “degree of skin pigmentation in the population.”² Women have higher occurrences than men for both types of cancers on the legs, consistent with greater sun exposure at this site.⁹ In 2006, a study noted that the ratio of BCC to SCC is 4 : 1 for the head and neck.¹⁰ The probability of getting SCC is less than getting BCC; however, SCC “carries a > 10-fold higher risk of metastasis and mortality.”⁶

Melanoma

It is estimated that 132,000 new cases of melanoma occur worldwide each year.^{3,4} Incidence rates are at least 16

times greater in Caucasians than African Americans and 10 times greater than Hispanics.^{10,11} The WHO also “estimates that as many as 65,161 people a year worldwide die from malignant skin cancer,” approximately 48,000 of whom are registered.^{3,12} Melanoma represents only about 3% of all skin cancers in the U.S., but it accounts for about 75% of all skin cancer deaths.^{12–14} The American Academy of Dermatology (AAD) estimated that in 2009, there will be about 121,840 new melanoma cases in the U.S. with 8650 deaths (~1 death every hour).¹² This mortality value is remarkably high considering the fact that melanoma is nearly always curable in its early stages; however, this high number can be attributed to the late diagnosis of the disease in which the cancer spreads to other parts of the body. Over the last three decades, the incidence and mortality rates of melanoma have increased in the U.S.¹⁵ In particular, of all neoplasms, approximately 20–30% of skin cancers are diagnosed in Caucasians, 2–4% are in Asians and 1–2% are in blacks and Asian Indians.¹⁰ In 2006, of all skin cancers, melanoma represented 1–8% in blacks, 10–15% in Asian Indians and 19% in Japanese.¹⁰ Moreover, even though skin cancers are not as prevalent in individuals with darker skin, they can have more morbidity and fatalities as they may go undiagnosed for a while.¹⁰ Melanoma most often appears on the trunk of men and the lower legs of women, although it can be found on the head, neck, or elsewhere.^{10,13} Researchers estimate that 1 of 50 people in the U.S. in 2010 will be diagnosed with melanoma at some point in their lives. Specifically, among Caucasians, the rate of increase of melanoma incidence is 3–7% each year. Intermediate skin pigmentations, (i.e., Hispanics and Asians) have skin cancers resembling both Caucasians and dark-skinned groups in terms of clinical presentation and epidemiology. This issue is an important

public health concern because by 2050, it is estimated that about half of the U.S. population will consist of Hispanics, Asians and blacks.¹⁰

As the incidence of skin cancer is increasing at an alarming rate, it is one of the greatest threats to public health. The pathogenesis of skin cancer is multifactorial. However, UVR (a potent carcinogen) is a major contributing factor.^{8,9,13} Our aim was to provide an overview of skin cancer, its epidemiology, incidence and the relationship of UVR-induced immunosuppression with skin cancer; protective measures and preventative strategies are also mentioned.

Ultraviolet radiation

Sunlight is a continuous spectrum of electromagnetic radiation that is divided into three major spectrums of wavelength: ultraviolet, visible and infrared.¹⁶ The UV range is the most significant spectrum of sunlight that causes photoaging and skin cancer. UVR is subdivided into ultraviolet A [UVA (315–400 nm)], ultraviolet B [UVB (280–315 nm)] and ultraviolet C [UVC (100–280 nm)].¹⁷ Approximately 90–99% of the solar UVR energy that reaches the earth’s surface is UVA, where only 1–10% is UVB (Table 1).^{18,19} One study indicated that about 65–90% of all melanomas are attributable to UVR exposure.²⁰

UVR and skin cancer pathogenesis

The damaging effects of UVR on the skin are thought to be caused by direct cellular damage and alterations in immunologic function. UVR produces DNA damage (formation of cyclobutane pyrimidine dimers), gene mutations, immunosuppression, oxidative stress and inflammatory responses, all of which have an important role in photoaging of the skin and skin cancer.²¹ In addition to

Table 1 Types of ultraviolet radiation and their properties^{16–20}

Type of UV radiation	General properties
Ultraviolet A radiation (UVA)	Approximately 90–99% reaches the earth’s surface Is not filtered by the stratospheric ozone layer in the atmosphere Long wavelength & low energy- can penetrate deeper into the skin Once considered harmless, but now believed to be harmful if one has excessive and long-term exposure Causes aging of the skin; induces immediate and persistent pigmentation (tanning) Passes through glass
Ultraviolet B radiation (UVB)	Approximately 1–10% reaches the earth’s surface Filtered by the stratospheric ozone layer in the atmosphere Short wavelength & high energy- can penetrate the upper layers of the epidermis Responsible for causing sunburns, tanning, wrinkling, photoaging and skin cancer Carcinogenic and a thousand times more effective in causing sunburns than UVA Does not pass through glass
Ultraviolet C radiation (UVC)	Filtered by the stratospheric ozone layer in the atmosphere before reaching earth Major artificial sources are germicidal lamps Burns the skin and causes skin cancer

this, UVR creates mutations to p53 tumor suppressor genes; these are genes which are involved in DNA repair or the apoptosis of cells that have lots of DNA damage. Therefore, if p53 genes are mutated, they will no longer be able to aid in the DNA repair process; as a result, there is “dysregulation of apoptosis, expansion of mutated keratinocytes, and initiation of skin cancer.”²² UVA radiation has an important role in the carcinogenesis of stem cells of the skin.²² UVB radiation induces DNA damage, which causes inflammatory responses and tumorigenesis.²¹

Skin color and photoprotection

The “low incidence of cutaneous malignancies in darker-skinned groups is primarily a result of photoprotection provided by increased epidermal melanin, which provides an inherent sun protection factor (SPF) of up to 13.4 in blacks. Epidermal melanin in blacks filters twice as much UVB radiation as does that in Caucasians. Black epidermis transmits 7.4% of UVB and 17.5% of ultraviolet A rays, compared with 24% and 55% in Caucasian epidermis, respectively.¹⁰ This is because the larger, more melanized melanosomes in the epidermis of dark skin absorb and scatter more light energy than the smaller, less melanized melanosomes of white skin. The dose of UVR required to produce a minimally perceptible erythema has been estimated to be 6–33 times greater in blacks than in whites.”¹⁰

Factors which influence the emission of UVR

Ultraviolet radiation that reaches the earth’s surface can increase or decrease based on a variety of factors. One factor is the ozone layer, which forms a thin shield in the stratospheric atmosphere, protecting life on earth from the sun’s UV rays; this layer absorbs all UVC radiation, most UVB radiation and very little UVA radiation.²³ Since the mid 1980s, scientists began to be concerned that the ozone layer was being depleted.^{24,25} The reason for thinning of the stratospheric ozone is resulting from the release of ozone-depleting substances and chemicals (chlorofluorocarbons) that are released from industry and motor vehicle exhaust into the atmosphere.²⁴ An approximate 1% decrease in ozone levels corresponds to a 1–2% increase in the mortality caused by melanoma.²³ Likewise, a 10% decrease in the ozone levels will cause 300,000 new nonmelanoma and 4500 new melanoma skin cancer cases.⁴ Depletion of the ozone layer results in increased UVR, (especially UVB, which is the most carcinogenic UVR), reaching the earth’s surface. UVB is directly absorbed by DNA and causes structural DNA damage. UVA causes indirect DNA damage through the formation of reactive oxygen species, which create breaks in DNA.

These events lead to mutations and then skin cancer.²⁶ Another factor affecting the level of UVR that reaches the earth’s surface is the time of day and the time of year. The sun exerts its highest peak between 10 AM to 4 PM. During this time, the sun’s rays have the least distance to travel through the atmosphere and UVB levels are at their highest. In the early morning and late afternoon, the sun’s rays pass through the atmosphere at an angle and their intensity is greatly reduced. The sun’s angle varies with the seasons, causing the intensity of UV rays to change. UV intensity tends to be the highest during the summer season.^{23,27} Environmental factors that increase the amount of UVR exposure to humans include latitudes closer to the equator. At higher latitudes the sun is lower in the sky, so UV rays must travel a greater distance through ozone-rich portions of the atmosphere and in turn, less UVR is emitted. Hence, living closer to the equator increases UV exposure, thus increasing the incidence of skin cancers.²³ For every 1000 meters increase in elevation, the UVR intensity increases by 10–12%.¹⁷ UV levels also depend on cloud cover; thus, there are lower UV levels at higher cloud cover densities.¹⁷ In the summer, “the sun is higher in the sky, and less ultraviolet radiation is absorbed during its passage through the atmosphere. Fog, haze, clouds, and pollutants can reduce ultraviolet levels by 10–90%. Snow, sand, and metal can reflect up to 90% of ultraviolet [radiation]. Sea water can reflect up to 15%, whereas little reflection occurs on still water (e.g., a pool). Shade alone reduces solar UVR by 50–95%. The amount of protection varies considerably between different shade settings, with a beach umbrella showing the least and dense foliage the most protection. The best technique for reducing ultraviolet exposure is to avoid the sun, especially in the middle of the day.”²³

UVR and damaging effects

Lifestyle changes during the past five decades, with an increase in exposure to sunlight because of outdoor activities and worsening sunbathing habits often result in skin cancers.^{8,13} Among Caucasians, “intense early sunburns and blistering sunburns are closely associated with the development of melanoma.”¹⁰ As a result of chronic UV exposure, skin aging, “wrinkles, uneven skin pigmentation, loss of skin elasticity and a disturbance of skin barrier functions” result. These “changes in the skin that superimpose the alterations of chronological aging” refer to photoaging.²⁸ The development of SCCs, BCCs and malignant melanoma is often associated with painful sunburns.²⁹ In fact, “more than 1 severe sunburn in childhood is associated with a 2-fold increase in melanoma risk.”³⁰ Chronic exposure to UVR is known as the most important risk factor for the development of actinic

keratoses (precursors of SCC).²⁹ Exposure to UVR during childhood and adolescence plays a role in the future development of skin cancer.^{31,32} It was noted that in the US, most people receive 22.73% of their lifetime exposure to the sun by 18 years of age.¹⁴ This meant that during childhood (1–18 years of age), most people received approximately one-fifth of their total sun exposure.¹⁴ The total amount of sun received over the years, and overexposure resulting in sunburns are associated with skin cancers.³¹ A history of exposure to sunlight, particularly sunburns, during childhood is also the most important behavioral risk factor for the development of NMSC and melanoma.^{32,33}

The epidemiology implicating UV exposure as a cause of melanoma is further supported by biological evidence that damage caused by UVR, particularly damage to DNA, plays a central role in the development of melanoma.¹³ The relative risk of skin cancer is three times as high among people born in areas that receive high amounts of UVR from the sun than those who move to those areas in adulthood.³¹ Likewise, outdoor workers have a higher risk than indoor workers.²⁰

The aforementioned citations conclude that there is a dose-related relationship between sunlight exposure and the incidence of skin cancer. For the development of BCC and melanoma, intermittent intense exposures appear to carry a higher risk than lower level chronic exposures, even if the total UV dose is the same. By contrast, the risk for SCC is strongly associated with chronic UV exposure but not with intermittent exposure.³³ Taken together, epidemiologic studies and experimental studies in laboratory animals indicate that intermittent intense and chronic exposures to solar UVR are the primary cause of NMSCs and melanoma.

UVR-induced immunosuppression

Ultraviolet immunosuppression is considered an important event in skin carcinogenesis.²⁶ UV exposure adversely affects the skin's immune system by: (1) diminishing antigen-presenting cell function, (2) inducing immunosuppressive cytokine production and (3) modulating contact and delayed-type hypersensitivity reactions.³⁴ The role of UV-induced immunosuppression and its relevance to the mechanism of skin cancer has not been fully elucidated. One study showed that renal transplant recipients, who are taking immunosuppressive therapies, have increased immunosuppression and decreased immunosurveillance; therefore, they are more susceptible to cancers, especially skin cancers (90% being NMSCs). Such individuals are more prone to skin cancer if they have UVR exposure and if they are light-skinned.³⁵ However, skin cancers tend to develop in sun-exposed areas regardless

of sun exposure before or after transplantation.^{23,36,54} Among transplant recipients, BCC risk had a linear increase and SCC risk had an exponential increase. Additionally, within this group, older individuals have a faster incidence of skin cancer because of longer UVR exposure over their lifetimes.^{35,37}

Implications of UVR in clinical therapies and skin cancer

Phototherapy

Phototherapy is currently being used to treat various cutaneous diseases.^{38,39} These phototherapies are conducted using broadband UVB (290–320 nm), narrowband UVB (311–313 nm), UVA-1 (340–400 nm), and psoralens plus UVA (PUVA).^{38–40} Despite the positive aspects of PUVA, its short-term usage is associated with erythema, edema, and sunburns, while its long-term usage is correlated with photoaging and skin cancers.^{38–40} PUVA therapy is mutagenic and carcinogenic. The determinants of the risk of NMSC and melanomas in PUVA-treated patients can vary with the dose and length of time of exposure to PUVA. Both European and American studies have demonstrated that patients exposed to high doses of PUVA therapy have a substantially increased risk of SCC and melanoma.^{41–43} Some data also indicate a small but significant increase of BCC.^{41–43} Patients treated with PUVA may also develop large, irregular, unevenly pigmented, dark lentigines known as “PUVA lentigines,” which may be precursors of melanoma.⁴³ A recent large-scale cohort study revealed that patients who had undergone PUVA therapy were still susceptible to developing skin cancers even after 25 years of discontinuation of PUVA. Therefore, even though PUVA can be a useful tool for treating several skin conditions, it is still a major risk factor for many skin conditions.⁴⁴

UVR relating to tanning beds and lamps

Every year, approximately 28 million Americans are reported to use artificial UV tanning.⁴⁵ The National Institute of Environmental Health Sciences (NIEHS) warns that solar UVR and exposure to sunlamps and tanning beds are carcinogenic. It has been suggested that artificial UVR is linked to melanoma development.^{46,47} The effects of natural and artificial UV exposure may take 20 or more years to produce skin cancer.⁴⁸ In a study, it was estimated that people using artificial UV tanning have a 2–3 fold increased risk of NMSCs.^{48,49} A recent study showed that tanning-bed bulbs emit mostly UVA radiation and ~5% UVB.⁵⁰ In general, young women were more frequent users of tanning beds than men. In addition, there is a positive correlation between tanning

bed usage and melanoma.⁵⁰ The other risk factors, in addition to UVR, are mentioned in Table 2.

Protective behaviors and preventive strategies

Personal behavioral modification

By being informed of the risks/effects of UV radiation, a few simple changes in behavior and lifestyle may prevent repeated sun damage and skin cancer (Table 3). This can be achieved by (1) minimizing sun exposure (seeking shade) during peak hours (10 AM–4 PM), (2) wearing sun-protective clothing (including hats with a brim all around, wrap-around sunglasses, which block both UVA and UVB rays, etc.), (3) using sunscreen with both UVA/UVB & physical blocks on your body and lips, and (4) avoiding

tanning beds/lamps – interestingly, “many tanning salon patrons erroneously believe that an artificial tan prevents subsequent sunburn and is safer than tanning outdoors.”^{50,51} Several studies have demonstrated that the use of sun-protective wear can decrease the number of moles and pre-malignant lesions.⁵²

Sunscreens are an important adjunct to other types of protection against UV exposure. Many studies have demonstrated that regular sunscreen use is effective in reducing the incidence of actinic keratoses and SCCs.²³ Another randomized trial demonstrated that among children who are at high risk for developing melanoma, sunscreens are effective in reducing moles, the precursors and the strongest risk factors for melanoma.⁵³ The AAD recommends broad-spectrum sunscreen covering both UVA & UVB with a Sun Protection Factor (SPF) of at least 15 with reapplication every 2 h when outdoors, even on cloudy days; additionally, one should make efforts to

Table 2 Risk factors for skin cancer^{8,10,13,20,23,26,28–50}

UV Radiation exposure
UVA
UVB
Immunosuppression
UV radiation
Immunosuppressive medications
Organ transplant recipients (especially with Fitzpatrick skin types I, II and III)
Acquired immune deficiency syndrome (AIDS)
Major genetic syndromes
Xeroderma pigmentosum
Oculocutaneous albinism
Epidermodysplasia verruciformis
Basal cell nevus syndrome
Predisposing clinical treatments
Phototherapy
PUVA (Psoralen + UVA therapy)
Viruses & infections
Human papilloma virus (HPV)
Human immunodeficiency virus (HIV)
Environmental pollutants & chemical carcinogens
Polycyclic aromatic hydrocarbons (PAHs)
Exposures to chemical carcinogens
Ionizing radiation
X-rays
Other risk factors
Artificial UV radiation (Tanning)
Smoking
Color of the skin (Having fair skin, especially with blue, or hazel eyes, skin types I and II and blond and red hair)
History and precursor lesions
Dermatoses and keratoses
Chronically injured or nonhealing wounds
Scars
Diet
Alcohol intake
Secondary to other cancers
Increasing age
Working outdoors

Table 3 Prevention strategies for skin cancer^{10,23,28,49–63}

Minimize sun exposure
Minimize sun exposure, especially from 10 AM–4 PM (peak sun hours)
Sun protection
Use wrap-around sunglasses, which block both UVA and UVB rays
Wear clothing, which use tightly-woven fabric
Wear a hat with a brim all around, especially during peak sun hours
Sunscreens
Use an effective and protective sunscreen (high SPF [at least SPF 15], with UVA/UVB and physical blocks) as needed all over exposed skin and even on your lips
Reapply sunscreen every 2 h when outdoors, even on cloudy days
Apply sunscreen to children 6 months and older
Avoid artificial tanning
Avoid the usage of tanning beds and lamps
Policies
Plan, implement and follow policies, which reduce UV exposure
Sun safety education
Age-appropriate health education to public and all patients (regardless of age, gender, health status and skin color)
Education efforts and public health campaigns for patients, caregivers, populations at risk, children and public
Skin cancer education for family members, teachers, administrators, coaches and healthcare personnel (including primary care physicians and dermatologists)
Services & organizations
Promotion of health services and organizations, which support skin cancer education
Skin examinations
Perform frequent skin examinations and seek a health professional on a routine basis to get full skin exams
Seek medical attention if you feel there is a mole that is getting bigger or if you have any questions
Screening
Promotion and usage of free skin cancer screening programs
Environments
Creating and maintaining environments, which promote sun safety

minimize sun exposure and to apply sunscreen to children 6 months of age and older.⁵⁴ The AAD and the NIEHS also recommend that the public avoid tanning beds because of their possible association with skin cancers.^{49,54} Additionally, patients are advised to perform frequent skin examinations and get complete examinations from health care professionals on a routine basis. Moreover, if a mole is changing color or increasing in size, one should seek medical attention.¹⁰

In regard to sunscreen usage, many studies have shown a reduction in several skin conditions and some skin cancers in people, who use sunscreens regularly.²³ Moreover, UVR “is responsible for the production of vitamin D₃ in the skin. Vitamin D₃ is hydroxylated in the liver and kidney to produce 1,25 (OH)₂ vitamin D, a hormone that regulates calcium homeostasis and bone maintenance. There has been controversy over the belief that people need to receive sunlight to maintain adequate vitamin D levels in the body. It has been suggested that current sun-avoidance practices, including the use of sunscreen products, may or will contribute to a widespread vitamin D deficiency. Evidence to support this emerges from clinical studies demonstrating that the application of sunscreen products will reduce artificial, UV-induced vitamin D in sunscreen users. However, studies examining actual vitamin D status in populations using sunscreen products have not found deficiencies in vitamin D or clinical evidence of such a deficiency. This could be because the sun is not the only source of vitamin D synthesis. Supplements can provide adequate intake, as can milk and orange juice fortified with vitamin D. The U.S. Department of Agriculture recommends 200 IU/d for children and younger adults (<50 years old), 400 IU/d for older adults (>50 years old), and 600 to 800 IU/d for the elderly (>70 years old). Eight ounces of fortified milk or orange juice contains 100 IU (2.5 g), which is also the amount of vitamin D found in approximately half a teaspoon of cod liver oil. The sun-and-vitamin-D issue has become less significant because of the fact that most foods are now enriched with vitamin D” and there are vitamin D supplements available to alleviate any deficiencies.⁵⁵ Nevertheless, clothing and sunscreen usage has been shown, for the most part, to reduce UVR exposure.²³

Public interventions and education

Various skin cancer task forces have proposed several important guidelines to decrease the rising skin cancer incidence.^{56,57} These briefly include the following: (1) the establishment of policies that reduce exposure to UVR; (2) providing and maintaining physical and social environments, which support sun safety and are consistent with the development of other healthful habits; (3) professional pre-service and in-service skin cancer education

for school administrators, teachers, physical education teachers and coaches, nurses, and others working in healthcare; (4) health services and organizations to increase skin cancer prevention education, sun-safety environments and making these policies readily available to the public; (5) lastly, the promotion of free skin cancer screening programs are also highly encouraged.^{56,57}

Several studies “support the notion that individuals’ beliefs about sun-risk and sun-safe behaviors have a major influence on their intentional sunbathing and sunbathing consequences.”⁵⁸ Education can also be greatly influential for people during decision making. However, education should be age-appropriate and it should create awareness, knowledge, attitudes and behavioral skills that people need to prevent skin cancer. Moreover, this education should be linked to opportunities for practicing sun-safety behaviors. Primary care physicians can have larger roles in preventing skin cancer if they are trained to “recognize and educate patients at risk, as well as direct them to be followed under dermatology care.”⁵⁹ Therefore, there is a need for education related to UV exposure and skin cancer risk. To address this issue, it would be beneficial to implement educational programs tailored for schools/workplaces, homes and doctors’ visits. Despite the importance of patient education by physicians, Freiman *et al.*, determined that only 19% of selected patients received sun advice from their physicians before they were diagnosed with melanoma and 49% received advice after diagnosis. The authors go on to say that “patients with known risk factors were not preferentially targeted for advice before their diagnosis.”⁵⁹ This shows how important it is for physicians and dermatologists to provide sun-related education. Patient education can include advice pertaining to sunscreen usage, reapplication methods, risk factors and tanning bed dangers. In addition to this, visual aids can be valuable in physicians’ offices as they can display the results of people after receiving a great deal of UVR.⁶⁰ Naylor and Robinson mention sun-protection strategies, which physicians can utilize to promote safe sun behaviors. These include: (1) setting a date to end intentional tanning, (2) determining which past behaviors were helpful in protecting against sun exposure and trying to incorporate them (as well as other techniques) in the future, (3) making strategies to overcome obstacles and (4) involving family members so everyone would remind each other about using sun protection. These authors suggest that the techniques listed above will be beneficial if they are implemented on individual patients rather than the entire public.⁶¹

In addition to the above, it is also advisable to provide children with sun-safety education, which can also influence their caregivers.⁶² By encouraging and teaching youngsters about healthy sun exposure habits, one

can influence them to make sound judgments as they get older. Robinson and Rademaker suggest that by educating and promoting healthy sun-protection behaviors in adults, children will be able to learn these positive behaviors and apply them in their lives. Moreover, if children practice sun-protective techniques, they will reduce their cumulative lifelong sun exposure and intense episodic sun exposure, hence reducing their risk for skin cancer (NMSCs and melanoma).⁶³ In addition to the youth, such sun safety education programs are also vital for people at high risk of getting skin cancer.²⁸

Besides patient education, public health campaigns, which are broadcast via the media, can also serve to teach the public.⁶⁰ These messages should be concise, easily attainable and they should target specific audiences (public and healthcare professionals).⁶² In conclusion, by providing proper education, people will be more aware of the truths and consequences of excessive UV exposure and skin cancer. This will hopefully allow them to take preventive steps, to reduce their risk of skin cancer. Through public education, healthcare agencies/providers can increase the public's awareness of skin cancers, their causes and preventative measures, which may be both life-changing and life-saving.^{56,57}

Questions

1. How many cases of NMSCs occur worldwide each year?
 - a. 50,000
 - b. 500,000
 - c. 1 million
 - d. 2–3 million
 - e. 5 million
2. How many new cases of melanoma occur worldwide each year?
 - a. 11,000
 - b. 27,000
 - c. 58,000
 - d. 104,000
 - e. 132,000
3. According to the WHO, how many people die a year worldwide from malignant skin cancer?
 - a. 1,562
 - b. 14,789
 - c. 22,974
 - d. 65,161
 - e. 81,678
4. According to a study, what percentage of all melanomas are attributable to UVR exposure?
 - a. 10–15%
 - b. 20–25%
 - c. 30–85%
 - d. 60–75%
 - e. 65–90%
5. A 10% decrease in ozone levels will cause...
 - a. 300,000 new NMSC cases and 4,500 new melanoma cases
 - b. 150,000 new NMSC cases and 2,250 new melanoma cases
 - c. 100,000 new NMSC cases and 2,000 new melanoma cases
 - d. 50,000 new NMSC cases and 500 new melanoma cases
 - e. 10,000 new NMSC cases and 250 new melanoma cases
6. Based on a recent study, what percentage of lifetime exposure to the sun do people receive by the age of 18?
 - a. About 15%
 - b. About 23%
 - c. About 55%
 - d. About 78%
 - e. About 90%
7. According to a study, approximately how much does the risk of getting NMSCs increase when using artificial UV tanning?
 - a. 2–3 fold
 - b. 5 fold
 - c. 10 fold
 - d. 15 fold
 - e. 20 fold
8. What can one do to prevent skin cancer?
 - a. Minimize sun exposure, especially from 10AM to 4PM
 - b. Use sun-protective clothing
 - c. Use sunscreen
 - d. Avoid artificial tanning
 - e. All of the above
9. According to a study, what percentage of patients received sun advice from their physicians prior to being diagnosed with melanoma?
 - a. 2%
 - b. 16%
 - c. 19%
 - d. 24%
 - e. 32%
10. What percentage UVR protection does a sunscreen with SPF 15 provide?
 - a. 96%
 - b. 75%
 - c. 54%
 - d. 23%
 - e. 15%

See answers on page 986.

References

- 1 Breitbart EW, Greinert R, Volkmer B. Effectiveness of information campaigns. *Prog Biophys Mol Biol* 2006; 92: 167–172.
- 2 Suárez B, López-Abente G, Martínez C, *et al.* Occupation and skin cancer: the results of the HELIOS-I multicenter case-control study. *BMC Public Health* 2007; 7: 180.
- 3 Foster PJ, Dunn EA, Karl KE, *et al.* Cellular magnetic resonance imaging: in vivo imaging of melanoma cells in lymph nodes of mice. *Neoplasia* 2008; 10: 207–216.
- 4 World Health Organization. Skin cancers [online]. Available from URL: <http://www.who.int/uv/faq/skincancer/en/print.html>. [Accessed 2009 September 14].
- 5 Rhee JS, Matthews BA, Neuburg M, *et al.* The skin cancer index: clinical responsiveness and predictors of quality of life. *Laryngoscope* 2007; 117: 399–405.
- 6 Rittié L, Kansra S, Stoll SW, *et al.* Differential ErbB1 signaling in squamous cell versus basal cell carcinoma of the skin. *Am J Pathol* 2007; 170: 2089–2099.
- 7 O'Driscoll L, McMorro J, Doolan P, *et al.* Investigation of the molecular profile of basal cell carcinoma using whole genome microarrays. *Mol Cancer* 2006; 5: 74.
- 8 Preston DS, Stern RS. Nonmelanoma cancers of the skin. *N Engl J Med* 1992; 327: 1649–1662.
- 9 Diepgen TL, Mahler V. The epidemiology of skin cancer. *Br J Dermatol* 2002; 61: 1–6.
- 10 Gloster HM Jr, Neal K. Skin cancer in skin of color. *J Am Acad Dermatol.* 2006; 55: 741–760; quiz 761–764.
- 11 Albert VA, Koh HK, Geller AC, *et al.* Years of potential life lost: another indicator of the impact of cutaneous malignant melanoma on society. *J Am Acad Dermatol* 1990; 23: 308–310.
- 12 American Academy of Dermatology. 2008 Skin cancer fact sheet [online]. Available from URL: http://www.aad.org/media/background/factsheets/fact_skincancer.html. [Accessed 2009 September 14].
- 13 Gilchrist BA, Eller MS, Geller AC, *et al.* The pathogenesis of melanoma induced by ultraviolet radiation. *N Engl J Med* 1999; 340: 1341–1348.
- 14 The Skin Cancer Foundation. Skin cancer facts [online]. Available from URL: <http://www.skincancer.org/Skin-Cancer-Facts/> [Accessed 2009 September 14].
- 15 Petermann KB, Rozenberg GI, Zedek D, *et al.* CD200 is induced by ERK and is a potential therapeutic target in melanoma. *J Clin Invest* 2007; 117: 3922–3929.
- 16 Soehnge H, Ouhtit A, Ananthaswamy ON. Mechanisms of induction of skin cancer by UV radiation. *Front Biosci* 1997; 2: D538–D551.
- 17 World Health Organization. Ultraviolet radiation and health [online]. Available from URL: http://www.who.int/uv/uv_and_health/en/index.html. [Accessed 2009 September 14].
- 18 Miller SA, Hamilton SL, Wester UG, *et al.* An analysis of UVA emissions from sunlamps and the potential importance for melanoma. *Photochem Photobiol* 1998; 68: 63–70.
- 19 Pastila R, Leszczynski D. Ultraviolet-A radiation induces changes in cyclin G gene expression in mouse melanoma B16-F1 cells. *Cancer Cell Int* 2007; 7: 7.
- 20 Glanz K, Buller DB, Saraiya M. Reducing ultraviolet radiation exposure among outdoor workers: state of the evidence and recommendations. *Environ Health* 2007; 6: 22.
- 21 Meeran SM, Punathil T, Katiyar SK. Interleukin-12-deficiency exacerbates inflammatory responses in UV-irradiated skin and skin tumors. *J Invest Dermatol* 2008; 128: 2716–2727.
- 22 Benjamin CL, Ananthaswamy HN. p53 and the pathogenesis of skin cancer. *Toxicol Appl Pharmacol* 2007; 224: 241–248.
- 23 Lautenschlager S, Wulf HC, Pittelkow MR. Photoprotection. *Lancet* 2007; 370: 528–537.
- 24 Armstrong BK, Kricger A. The epidemiology of UV induced skin cancer. *J Photochem Photobiol* 2001; 63: 8–18.
- 25 Kripke ML. Impact of ozone depletion on skin cancers. *J Dermatol Surg Oncol* 1988; 14: 853–857.
- 26 Brenner M, Hearing VJ. The Protective Role of Melanin Against UV Damage in Human Skin. *Photochem Photobiol* 2008; 84: 539–549.
- 27 Hill D, White V, Marks R, *et al.* Changes in sun-related attitudes and behaviours, and reduced sunburn prevalence in a population at high risk of melanoma. *Eur J Cancer Prev* 1993; 2: 447–456.
- 28 Schroeder P, Haendeler J, Krutmann J. The role of near infrared radiation in photoaging of the skin. *Exp Gerontol* 2008; 43: 629–632.
- 29 Kennedy C, Bajdik CD, Willemze R, *et al.* The influence of painful sunburns and lifetime sun exposure on the risk of actinic keratoses, seborrheic warts, melanocytic nevi, atypical nevi, and skin cancer. *J Invest Dermatol* 2003; 120: 1087–1093.
- 30 Ma F, Collado-Mesa F, Hu S, *et al.* Skin cancer awareness and sun protection behaviors in white Hispanic and white non-Hispanic high school students in Miami, Florida. *Arch Dermatol* 2007; 143: 983–988.
- 31 Whiteman DC, Whiteman CA, Green AC. Childhood sun exposure as a risk factor for melanoma: a systematic review of epidemiologic studies. *Cancer Causes Control* 2001; 12: 69–82.
- 32 Kricger A, Armstrong BK, English DR. Sun exposure and nonmelanocytic skin cancer. *Cancer Causes Control* 1994; 5: 367–392.
- 33 Westerdahl J, Olsson H, Ingvar C. At what age do sunburn episodes play a crucial role for the development of malignant melanoma. *Eur J Cancer* 1994; 30A: 1647–1654.
- 34 Beissert S, Schwarz T. Mechanisms involved in ultraviolet light-induced immunosuppression. *J Invest Dermatol Symp Proc* 1999; 4: 61–64.
- 35 Ho WL, Murphy GM. Update on the pathogenesis of post-transplant skin cancer in renal transplant recipients. *Br J Dermatol* 2008; 158: 217–224.

- 36 Euvrard S, Kanitakis J, Claudy A. Skin cancers after organ transplantation. *N Engl J Med* 2003; 348: 1681–1691.
- 37 Berg D, Otley CC. Skin cancer in organ transplant recipients: epidemiology, pathogenesis, and management. *J Am Acad Dermatol* 2002; 47: 1–17.
- 38 Parrish JA, Fitzpatrick TB, Tanenbaum L, et al. Photochemotherapy of psoriasis with oral methoxsalen and longwave ultraviolet light. *N Engl J Med* 1974; 291: 1207–1211.
- 39 Greaves MW, Weinstein GD. Treatment of psoriasis. *N Engl J Med* 1995; 332: 581–588.
- 40 Dawe RS. Ultraviolet A1 phototherapy. *Br J Dermatol* 2003; 148: 626–637.
- 41 Stern RS, Lieberman EJ, Vakeva L. Oral psoralen and ultraviolet-A light (PUVA) treatment of psoriasis and persistent risk of nonmelanoma skin cancer. PUVA Follow-up Study. *J Natl Cancer Inst* 1998; 90: 1278–1284.
- 42 Stern RS, Laird N, Melski J, et al. Cutaneous squamous-cell carcinoma in patients treated with PUVA. *N Engl J Med* 1984; 310: 1156–1161.
- 43 Stern RS, Nichols KT, Vakeva LH. Malignant melanoma in patients treated for psoriasis with methoxsalen (psoralen) and ultraviolet A radiation (PUVA). The PUVA Follow-Up Study. *N Engl J Med* 1997; 336: 1041–1045.
- 44 Nijsten TE, Stern RS. The increased risk of skin cancer is persistent after discontinuation of psoralen+ultraviolet A: a cohort study. *J Invest Dermatol* 2003; 121: 252–258.
- 45 American Academy of Dermatology. Indoor tanning fact sheet [online]. Available from URL: http://www.aad.org/media/background/factsheets/fact_indoortanning.html [Accessed 2009 September 14].
- 46 Swerdlow AJ, Weinstock MA. Do tanning lamps cause melanoma? An epidemiologic assessment *J Am Acad Dermatol* 1998; 38: 89–98.
- 47 Spencer JM, Amonette RA. Indoor tanning: risks, benefits, and future trends. *J Am Acad Dermatol* 1995; 33: 288–298.
- 48 Chen YT, Dubrow R, Zheng T, et al. Sunlamp use and the risk of cutaneous malignant melanoma: a population-based case-control study in Connecticut, USA. *Int J Epidemiol* 1998; 27: 758–765.
- 49 Karagas MR, Stannard VA, Mott LA, et al. Use of tanning devices and risk of basal cell and squamous cell skin cancers. *J Natl Cancer Inst* 2002; 94: 224–226.
- 50 Ting W, Schultz K, Cac NN, et al. Tanning bed exposure increases the risk of malignant melanoma. *Int J Dermatol* 2007; 46: 1253–1257.
- 51 Centers for Disease Control and Prevention. Skin cancer: prevention. Available from URL: http://www.cdc.gov/cancer/skin/basic_info/prevention.htm. [Accessed 2009 September 14].
- 52 Autier P, Dore JF, Cattaruzza MS, et al. Sunscreen use, wearing clothes, and number of nevi in 6- to 7-year-old European children. European Organization for Research and Treatment of Cancer Melanoma Cooperative Group. *J Natl Cancer Inst* 1998; 90: 1873–1880.
- 53 Gallagher RP, Rivers JK, Lee TK, et al. Broad-spectrum sunscreen use and the development of new nevi in white children: A randomized controlled trial. *JAMA* 2000; 283: 2955–2960.
- 54 The American Academy of Dermatology. Ultraviolet Index: What You Need to Know [online]. Available from URL: http://www.aad.org/public/publications/pamphlets/sun_ultraviolet.html. [Accessed 2009 September 14].
- 55 Skolnick BA, Saladi RN, Fox JL. An Update on Sunscreens. *J Am Osteopath Coll Dermatol* 2007; 8: 23–29.
- 56 Glanz K, Saraiya M, Wechsler H. Centers for disease control and prevention. Guidelines for school programs to prevent skin cancer. *MMWR Recomm Rep*. 2002; 51: 1–18.
- 57 Saraiya M, Glanz K, Briss P, et al. Task force on community preventive services on reducing exposure to ultraviolet light. Preventing skin cancer: findings of the task force on community preventive services on reducing exposure to ultraviolet light. *MMWR Recomm Rep*. 2003; 52: 1–12.
- 58 Turrisi R, Hillhouse J, Heavin S, et al. Examination of the short-term efficacy of a parent-based intervention to prevent skin cancer. *J Behav Med* 2004; 27: 393–412.
- 59 Freiman A, Yu J, Loutfi A, et al. Impact of melanoma diagnosis on sun-awareness and protection: efficacy of education campaigns in a high-risk population. *J Cutan Med Surg*. 2004; 8: 303–309.
- 60 Soto E, Lee H, Saladi RN, et al. Behavioral factors of patients before and after diagnosis with melanoma: a cohort study – are sun-protection measures being implemented?. *Melanoma Res*. In Press.
- 61 Naylor M, Robinson JK. Sunscreen, sun protection, and our many failures. *Arch Dermatol* 2005; 141: 1025–1027.
- 62 Robinson JK, Hornung RL. Non-profit organizations and public education: a compendium of resources for physicians. *Clin Dermatol* 1998; 16: 461–465.
- 63 Robinson JK, Rademaker AW. Sun protection by families at the beach. *Arch Pediatr Adolesc Med* 1998; 152: 466–470.

Answers

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3. d
4. e
5. a
6. b
7. a
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9. c
10. a