



Published in final edited form as:

*Dermatol Clin.* 2009 April ; 27(2): 205–viii. doi:10.1016/j.det.2008.12.002.

## Melanoma Epidemiology and Public Health

Marianne Berwick, PhD, MPH<sup>a,b,\*</sup>, Esther Erdei, PhD<sup>a</sup>, and Jennifer Hay, PhD<sup>c</sup>

<sup>a</sup>Division of Epidemiology and Biostatistics, Department of Internal Medicine, 1 University of New Mexico, MSC08 4360, CRF 103A, Albuquerque, NM 87131 0001, USA

<sup>b</sup>Population Science, University of New Mexico Cancer Center, Albuquerque, NM 87131, USA

<sup>c</sup>Department of Psychiatry and Behavioral Sciences, Memorial Sloan-Kettering Cancer Center, 641 Lexington Avenue, 7th Floor, New York, NY 10022, USA

### Abstract

Melanoma has presented a conundrum to physicians and prevention researchers. We should be able to reduce incidence and mortality rather easily because evolving melanoma lesions are observable on the surface of the skin. However, melanoma has not proved tractable. The incidence and mortality have risen in all developed countries during the past 50 years. However, mortality appears to be abating among younger cohorts even though the reason for this is not clear.

### Keywords

Melanoma; Mortality; Incidence

## EPIDEMIOLOGY

The epidemiology of melanoma is marked by contrasts. The majority of melanomas are diagnosed at very thin (<1 mm Breslow thickness) and highly curable stages—about 70 percent in most series. However, melanomas diagnosed later (those with Breslow thickness >1 mm) have much poorer survival rates and there are no satisfactory cures for advanced melanoma.

Furthermore, melanoma is more often diagnosed among advantaged individuals within a population, but the disadvantaged individuals are more likely to have advanced disease.<sup>1–3</sup> Whether this is due to host factors associated with deprivation, such as life stress, or to factors associated with access to care, such as distance to providers, is not well understood. Finally, cutaneous melanoma is most common among developed countries with large numbers of white residents, while it is rare and presents with different phenotype and histology in less developed countries and in other ethnic groups. Current estimates place the number of new cases of melanoma at 160,177 worldwide and deaths from melanoma at 40,781.<sup>4</sup> In the United States, there were an estimated 62,480 new cases of melanoma diagnosed in 2008, and 8420 deaths from melanoma.<sup>5</sup> Although the number of new cases appears to be increasing, the mortality rates appear to be decreasing among younger cohorts of melanoma patients.<sup>6</sup> In addition, some of the incidence increase is certainly artifactual as we are certainly looking harder for melanoma as evidenced by the increasing rates of biopsy, and we are finding lesions today that 20 years ago would never have been called

melanoma.<sup>7,8</sup> In the meantime, scientists cannot differentiate melanomas that will progress from those that are indolent; therefore, it is imperative that research continues to investigate the best means for prevention.

## Risk Factors

Risk factors for melanoma can generally be divided into environmental and genetic. The search to understand the interaction of the two has been facilitated by advances in genomics.

### Environmental risk factors

**Sunlight:** Ultraviolet radiation (UVR) is the major known etiologic agent associated with melanoma. Many individuals however do not know that different patterns of sun exposure have different effects in the development of melanoma. For example, chronic sun exposure, that which one receives during outdoor work on a daily basis, does not increase risk for melanoma and is even associated with inhibition of melanoma.<sup>9–11</sup> On the other hand, intermittent sun exposure, large blasts of UVR, received on weekends or holidays, is the major form of UVR promoting the development of melanoma.<sup>12</sup>

Measurement of intermittent sun exposure or recreational sun exposure represents an important research challenge because measures of past sun exposure necessarily depend on subject recall of exposure, which is not always reliable.<sup>13</sup> Epidemiologic studies have shown weak associations between episodes of sunburn and melanoma incidence.<sup>14</sup> Estimates of the effect of intermittent exposure have ranged from a protective effect of (a relative risk) of 0.44 (0.21–0.91)<sup>15</sup> to an adverse risk of 8.41 (3.63–19.6).<sup>16</sup> Imprecise measurement associated with self-reports is an important rate-limiting factor in determining the relationship between intermittent sun exposure and melanoma incidence.

Furthermore, excessive sunlight exposure in youth, among individuals with aberrant neovogenesis, increases the risk of melanoma, particularly for melanoma associated with BRAF mutations;<sup>17,18</sup> whereas continuous, intermittent sun exposure over a lifetime, are associated with NRAS mutations among older individuals.

**Other sources of ultraviolet radiation:** Mounting evidence points to the negative effects of tanning beds.<sup>19</sup> In 2006, the International Agency for Research on Cancer convened an expert panel of epidemiologists to evaluate the risks for melanoma and other skin cancers from use of sun-beds.<sup>20</sup> They performed a meta-analysis of 19 studies that have evaluated the association between sunbed exposure and melanoma and other skin cancers. The study showed that early life exposure is the most damaging. In this meta-analysis the relative risk for melanoma was 1.75 (95% confidence interval [CI], 1.35–2.26) for “first exposure under the age of 35;” a relative risk of 1.15 that is statistically significant (95% CI, 1.00–1.31) for “ever use;” a summary relative risk of 1.49 (95% CI, 0.93–2.38) for exposure distant in time; and a summary relative risk of 1.10 (95% CI, 0.76–1.60) for recent exposure. All of the relative risks are higher than 1.0. The most persuasive study a (prospective cohort study of 106,379 women in Sweden and Norway) found, not only a similar level of risk, but that the increased risk was not due to the type of UV lamps used before 1983 but likely due to more recent types of sunbeds.<sup>21</sup>

### Host factors

**Melanocytic nevi:** Nevi are the strongest risk factor for the development of melanoma. Studies have shown that those with a higher than average number of moles (often counted on the back, and 40 or more on the back can be considered high). The way in which nevi might be involved in risk has been considered in different ways. One theory is that nevi are actually on the causal pathway, and that some nevi develop into melanoma. This theory is

supported by the approximately 40% of melanomas that have an adjacent nevus or nevus remnant still observable by pathology.<sup>22,23</sup>

Individuals with fewer nevi require repeated exposure to sunlight to drive carcinogenesis because they are more likely to develop melanomas on high sun exposure areas such as the head and neck, while those with many nevi, who are more likely to develop melanoma of the trunk, may possess host factors that drive carcinogenesis after minimal sunlight exposure.<sup>18</sup> Therefore, it is plausible that the oncogenic pathway characterized by pigment cell instability is associated with a higher number of nevi and may lead to more aggressive melanoma and a worse prognosis.

In any case, having more nevi than average, or having dysplastic nevi, increases risk for melanoma substantially.<sup>24-26</sup>

**Family history:** First-degree relatives of melanoma patients have a higher risk of the disease than individuals without positive family history, suggesting that a distinct hereditary component exists. Familial melanoma accounts for an estimated 5 to 10 percent of all cases of melanoma.<sup>27</sup> Characteristics which distinguish the familial from the nonfamilial form of the disease include younger age at first diagnosis, better survival, thinner lesions, multiple primary lesions, and increased occurrence of nonmelanoma cancers.<sup>28</sup> Pooled data from eight case-control studies showed that an individual's risk of melanoma increases by about twofold with an affected first-degree relative.<sup>29</sup> This effect was independent of host factors such as age, nevus count, hair and eye color, and freckling. Familial relative risk remained similar in all of the studies, even though melanoma incidence varied by about 10-fold in the study areas.

**Phenotypic characteristics:** Phenotype is independently important as a risk factor for the development of melanoma: light hair color, light eye color, and light skin color, including skin that freckles easily. These phenotypic characteristics are often subsumed under the heading of “skin type.” Fitzpatrick<sup>30</sup> classified skin type into six groups that are frequently used even though the reproducibility of these groupings is relatively poor (Table 1).<sup>31</sup>

**Variation in susceptibility genes—**Major advances in genetics have made it possible to identify genetic factors that are critical to susceptibility to melanoma. The Genome Wide Association Studies (GWAS) have identified new variants in genes that may be found to play an important role in the development of melanoma.

**Pigmentation genes:** Foremost among the pigmentary genes known to be associated with melanoma is the melanocortin 1 receptor (MC1R). Data show that some MC1R variants are associated both with melanoma and phenotype—particularly red hair. Others are only associated with melanoma development, suggesting that MC1R variants could play a role in melanoma development by way of pigmentary and nonpigmentary pathways.<sup>32</sup> Pigmentary and other genes have recently been identified by GWAS, and their effects remain to be evaluated.<sup>33,34</sup>

**DNA repair genes:** Genetic variants in DNA repair genes are obvious candidates for melanoma susceptibility based on the xeroderma pigmentosum (XP) paradigm.<sup>35</sup> XP patients have an approximately 1,000-fold increased risk for developing melanoma and variants of XP, and other repair genes have recently been associated with risk.<sup>36,37</sup>

**Cell cycle genes:** To date, few mutations have been found in cell cycle genes in melanoma etiology.<sup>38,39</sup> The familial melanoma gene, CDKN2A, is rarely mutated among those with

sporadic melanoma, although it accounts for approximately 30 percent of mutations in those with a hereditary form of the disease.<sup>40</sup>

**Gene–environment interaction**—The pattern of sun exposure that appears to induce skin cancer, in particular melanoma, development is complex and is clearly different by skin type (ie, propensity to burn, ability to tan). Armstrong and colleagues<sup>41</sup> have proposed a model consistent with data from other epidemiologic studies where risk for melanoma increases with increasing sun exposure among those who tan easily, but only with a small amount after which risk decreases with increasing exposure. Among subjects who are intermediate in their ability to tan, risk continues to increase slowly and then, at some point, declines with increasing exposure. On the other hand, those subjects who have great difficulty tanning have an almost linear increase in risk with increasing sun exposure. This model recognizes that individuals are differentially susceptible to sun exposure and have different levels of risk based on skin type. Moreover, it suggests that different types or patterns of sun exposure are associated with different levels of risk for melanoma.

## PUBLIC HEALTH

Unfortunately, there is little sound evidence on which to move forward in public education and awareness of skin cancer. This is a critical juncture for the development of such evidence. Currently, different factions promulgate somewhat confusing recommendations: some advise avoiding the sun, and others advise enjoying a moderate amount of sun exposure. The tanning industry is even promoting the use of tanning parlors to develop a “safe” tan.

The American Cancer Society<sup>42</sup> recommends sun protection strategies including sun avoidance between 10<sup>AM</sup> and 4<sup>PM</sup> and, when sun cannot be avoided, use of umbrellas, protective hats, clothing, and sunscreen with a sun protection factor of 15 or more. Total avoidance of artificial UV sources such as tanning beds is also recommended. Unfortunately, less than half (47%) of the United States population engages in any sun protection,<sup>43</sup> and 59% report that they have sunbathed in the past year.<sup>44</sup> Data from over 28,000 individuals in the 2005 National Health Interview Survey<sup>45</sup> showed that only about half (43%–51% across age groups) reported frequent (sometimes/most of the time/always) use of sunscreen, 65% to 80% did not usually stay in the shade when outside on a sunny day, and 15% to 51% used sun protection clothing. For each behavior, younger age was related to higher levels of risk behavior.

## Educational Strategies

**Programs for the general population**—General population approaches to improve overall sun protection have been adopted most prominently in Australia. For example, the Australian state of Victoria adopted the SunSmart program integrating environmental change and mass media public education to address sun protection across the entire population. Documentation of this program shows that a mix of strategies is effective but quite expensive in improving sun protection behaviors.<sup>46</sup>

The “Slip, Slap, Slop” campaign has promoted sun protection for more than 20 years throughout Australia.<sup>47</sup> In Queensland, Australia, Stanton and colleagues<sup>48</sup> evaluated the effectiveness of one intensive campaign. They found that individuals did protect themselves when in the sun or, if they did not, they did not think they were out long enough to be sunburned. The motivation for protection stemmed from a desire to prevent future health problems, and it seems that the sun protective behaviors were a direct result of public health campaigns. However, they suggest that public health campaigns need to move beyond the current efforts to increase awareness and knowledge of skin cancer to provide supportive

environments (shade is an issue in places like Australia and New Mexico) and enhance individual skills (such as recognition of the UV Index [UVI]).

**Physician counseling**—The Task Force on Community Preventive Services,<sup>49</sup> a Centers for Disease Control and Prevention group, has found adequate evidence to show that sun exposure is associated with the development of melanoma, but inadequate evidence to indicate that physician counseling will change patient behaviors to reduce risk.

### Programs for high risk populations

**Children:** In the United States, targeted approaches to specific higher-risk populations have been the predominant approach to intervention development. For example, interventions targeted to children are arguably quite important given that children spend a greater proportion of their time outdoors, and because sun exposure and sunburns (in particular before age 18) represent a large proportion of the lifetime environmental risk of skin cancer. Sun protection programs for children include interventions focused on parent behaviors, preschool children, and school-age children. Many of these interventions have been implemented in schools and hospitals. In general, those multiunit programs that occur over several sessions with intensive instruction work best.<sup>50</sup> However, widespread dissemination of these interventions may be limited by shrinking resources and competing demands in the school and other institutional environments.

Unfortunately, the Community Preventive Services Task Force<sup>49</sup> reported that education and policy approaches to increasing sun protective behaviors were effective when implemented in primary schools and in recreational or tourism settings, but found insufficient evidence to determine effectiveness when implemented in other settings, such as child care centers, secondary schools and colleges, and occupational settings. They also found “insufficient evidence to determine the effectiveness of interventions oriented to healthcare settings and providers, media campaigns alone, interventions oriented to parents or caregivers of children, and community-wide multicomponent interventions.”

**Family history of melanoma:** Another population that has been recently targeted for intervention has included those with a family history of melanoma, who face increased risks for skin cancer over and above the general population. For instance, Geller and colleagues<sup>51</sup> developed a novel telephone motivational interviewing and tailored education material approach, but the intervention did not show differential improvement in the treatment over the control group.

**Interventions in high risk environments:** A final distinct intervention approach includes those interventions that are specific to various higher risk environments, and thus capitalize on the local situational cues in certain environments. For example, a program implemented at a zoo which included signage linking sun protection to animals' strategies of skin protection, tip sheets for parents, children's activities, and discounted sun protection was useful in increasing sales of sunscreen and hats compared with a control zoo.<sup>52</sup> Environmentally specific interventions have also been instituted at beaches, ski resorts, and swimming pools.<sup>53–55</sup>

### The Use of Sunscreens

It is still not clear that sunscreens of any sort will provide protection from developing melanoma although they clearly prevent sunburn—a sign that sensitive skin has had too much UV. Suggestions have been made that sunscreens actually increase risk for skin cancer,<sup>56</sup> although the most likely way that this association might occur is when individuals use sunscreens to prolong their stay in the sun. Therefore, it is possible that the use of

sunscreens for intentional sun exposure may actually increase risk. Sunscreen sun protection recommendations have been made by the International Agency for Research on Cancer.<sup>57</sup> No conclusion could be drawn about the cancer-preventive activity of topical use of sunscreens against basal-cell carcinoma and cutaneous melanoma. Unfortunately, based on data from carefully conducted studies, sunscreens are being used as tanning aids to avoid sunburn.<sup>58</sup>

The use of sunscreens has long been promoted as the first line of defense for prevention all types of skin cancer. However, during the last 10 years, the public has been warned not to rely on sunscreens alone, but to practice overall sun protection. Uncontrollable confounding limits the ability of an observational study to assess the efficacy of sunscreens. Therefore, individuals who are most at risk, those who are redheaded or a very light phenotype with poor tanning ability, or those who are planning to spend time at the beach after being in the office all day, are the ones who are most likely use sunscreens.

Since the 1990s, however, improvements have been made and sunscreens now cover a much broader portion of the UV spectrum.<sup>59</sup> We will be able to evaluate their effectiveness after a suitable lag period, such as 20 years, has passed.

A few randomized trials have evaluated the effects of sunscreens on a putative precursor lesion for melanoma, the development of nevi. Gallagher and colleagues<sup>60</sup> found a small effect of sunscreens in reducing nevus formation among those who freckled; the effect was extremely small although statistically significant. Additionally, an observational study in Queensland, found that sun protection, including frequent reapplication of sunscreen, was associated with a reduced number of nevi in children between 1 and 3 years old.<sup>61</sup> In addition, van der Pols and colleagues<sup>62</sup> found that after participation in a trial of the use of sunscreens, those who had been randomized to daily use continued to use sunscreens. Thus, although the investigators limit the interpretation of their findings because of a 63% response to their follow up, intensive encouragement over multiple time periods with a simple message may be more effective than the multicomponent messages delivered by community sun protection campaigns. A German study, using multiple methods for intervention, found no evidence that education and sunscreen use would affect the development of nevi in 1,232 children 2 to 7 years old after a 3-year intervention.<sup>63</sup>

### **Clothing as Protection**

The most consistent and robust protection from UVR in relationship to melanoma is the physical barrier provided by hats, long sleeves, and long pants. Clothing is an ideal protective agent against the sun and has been used for hundreds of years in sunny countries like India and Egypt where people tend to cover up to prevent excessive sun exposure with long sleeves, long pants, and hats or turbans.

The use of clothing appears to reduce the number of nevi formed. However, these studies are of young children who may eventually achieve their full nevus density later. An intervention to retard the development of nevi with clothing and sun protection was only moderately successful—more so among boys.<sup>64</sup> In an observational analysis of the same subjects in this randomized study, clothing and staying indoors were associated with a reduced number of nevi, but wearing sunscreen while unclothed was not.<sup>65</sup> Others found<sup>66</sup> that children who wore clothing had fewer nevi and those who used sunscreen had more.

### **The Controversy About Vitamin D and Sun Exposure**

Some sun exposure produces vitamin D synthesis, improves seasonal affective disorder, may help lower blood pressure, may be inversely associated with the incidence and mortality from a number of cancers, and is the treatment for polymorphic light eruption. Excessive

sun exposure, particularly the intermittent pattern, is responsible for much of cutaneous malignant melanoma. Overexposure to UVR can lead to sunburn, immunologic changes, precipitation and exacerbation of photosensitivity, accelerated skin aging and skin cancer. These important problems need to be addressed with effective education.

It has been hypothesized that, in some cases, even more sun may be beneficial. One explanation for the rise in melanoma incidence that takes into account the different effects of chronic (or daily) and intermittent sun exposure (the type office workers get on weekends and holidays at the beach) proposes that as people have replaced outdoor occupations with indoor ones, they have experienced more intermittent sun exposure.<sup>67</sup> The decrease in outdoor occupations, or chronic exposure which is actually protective for melanoma, could explain the increase in melanoma incidence in Canada.

Diffey<sup>68</sup> effectively argues that the population attains adequate vitamin D through recreational activities. He carefully points out that increasing solar exposure would lead to an increase in skin cancer. The point of the present discussion is that individuals should take care in the sun and, at the same time, realize that a small amount of sun exposure is not bad.

**Confusion due to messages about vitamin D and sun exposure**—Lately, the public has received confusing messages owing to the increasing recognition of the role of Vitamin D in protecting against many internal cancers as well as other diseases. In the United Kingdom, Hiom<sup>69</sup> worries that “a growing body of literature suggesting a cancer protective role for vitamin D and sun exposure presents further challenges for skin cancer prevention campaigns, no more so than when exaggerated claims for the health benefits of sunbathing make the media spotlight. The UK population tends to need little encouragement to make the most of sunshine, and this is especially true for the younger generation who most need to take care. Public health messages to avoid the midday sun, not to burn and to protect children should not adversely affect outdoor activity or population vitamin D levels, but it is important that they are targeted to those most at risk and are consistent.” A number of groups—the Australian and New Zealand Bone and Mineral Society, Osteoporosis Australia, Australasian College of Dermatologists, the Cancer Council Australia, and others—have recently modified a long-standing message to the public from one of staying out of the sun to one of short periods of exposure for health.<sup>70</sup> Some argue in favor of “Love the sun and protect your skin” as a public health message.<sup>71</sup>

## SOLUTIONS

### The UV Index

The UVI is used in most developed countries and although many programs have been developed in the countries where light-skinned individuals predominate and also have high rates of skin cancer, the UVI is not used as widely as it might be.

The UVI should be studied more thoroughly to understand just how to best communicate levels of ground-level sun effects. Brooks and colleagues<sup>72</sup> have recently suggested that advocacy groups should work with the World Health Organization to lead such efforts. When comparing the promulgation of the UVI in three countries, the United States, the United Kingdom, and Australia, one sees widely differing presentation of the index, none of which appears to be effective as yet. As this is a widely-reported measure to assist individuals in enhancing their time outdoors, the New South Wales Cancer Council and the Anti-Cancer Council of Victoria<sup>73</sup> have suggested that it would be valuable to learn more about public perceptions of the index and how to enhance its use. Efforts should be made to seek media and other settings through which information concerning the UVI can be disseminated to reach population subgroups involved in activities and situations identified as

at high risk for UV exposure. The groups suggest that priority be given to developing the following specific uses of the UVI:

In daily weather forecasts to prompt appropriate sun protection behavior

To improve understanding of the relationship between ambient temperature and ambient UV

As a guide to seasonal changes in the times of and level of protection necessary during childhood outdoor activities, particularly in schools

In sections of newspapers, magazines, radio programs and special cable TV channels aimed at high-risk groups or activities (eg, sports channels to target fishermen, cricketers, and sailors)

In travel information targeted at Australian and international tourists.

### Sun Exposure Behavior

The UVI can guide the need for protection. Sun exposure and sun protection are behaviorally controlled. It has recently been shown that the general population has a relatively accurate concept of skin cancer prevention.<sup>50</sup> However, high levels of knowledge about the risks of skin cancer do not necessarily translate into consistent sun protection. Indeed, this is the case for knowledge concerning diet and exercise recommendations as well, where knowledge is necessary but not sufficient to motivate behavior change.<sup>74–76</sup>

Given the prevailing belief that a suntan is attractive, concerns for appearance is a barrier to sun protection campaigns.<sup>77</sup> Accordingly, another important approach to promoting sun protection involves the use of appearance appeals, which are designed to emphasize the harm to physical appearance associated with sun exposure, or to increase the perceived attractiveness of untanned skin. Because sunless tanning is prevalent, it represents another important focus for intervention is amenable to appearance appeals.<sup>78</sup>

On the whole, these educational messages may, however, be based on an inaccurate assessment of the causes of skin cancer.<sup>79</sup> Although sunscreen use is important in reducing erythema (sunburn), clear evidence that it is associated with a reduction in melanoma or basal cell skin cancer is lacking, and the public may realize this. In the coming years, behavioral and psychosocial approaches will be important in addressing challenges on the horizon for skin cancer control. For example, the availability of genomic testing for melanoma may raise new questions on how to encourage sun protection in those who are tested to be genetically susceptible to melanoma or other skin cancers.<sup>80</sup>

### Screening

Finally, the value of sun protection versus screening in melanoma control will have implications for the focus of behavioral intervention approaches, with both higher-risk and general population cohorts.<sup>81</sup> Secondary prevention is a potentially useful tool in the armamentarium against morbidity and mortality from skin cancer. Although there are no randomized trials supporting widespread population-based screening for melanoma, the idea of skin screening remains appealing for melanoma—a visible malignancy that appears to have mortality benefit when detected early. Persons with a family history of melanoma or with multiple or atypical moles, and those with a previous diagnosis of melanoma are candidates for intensive skin examination, either by themselves or by a physician.

Although physicians have been shown to diagnose melanoma at a thinner stage,<sup>82</sup> many more opportunities for physicians to provide skin cancer prevention counseling exist as only 1.5% of adults and fewer adolescents received such counseling. Although the evidence for



the value of a melanoma screening program has been found to be insufficient by the US Preventive Services Task Force,<sup>83</sup> Geller and colleagues<sup>84</sup> are concerned that we may never have the randomized trial evidence that would convince individuals that screening will prevent mortality from melanoma. They point out the low screening rates in the population and suggest a rational program to find those at highest risk. In the first place, a baseline total skin examination of all white men 50 years and older seems rational. These are the individuals who most often have deep lesions and who die from melanoma; half of the melanoma-related deaths occur in this group. Geller and colleagues<sup>84</sup> lobby for expansion of public outreach, public and professional education and also legislative efforts.

Optimism is provided by Pennie and colleagues<sup>85</sup> who find that primary care providers are able to identify and treat melanomas appropriately—at almost the level of trained dermatologists. The barriers, however, are critical to consider: physicians need more changing rooms to facilitate full body examinations; there is no payment mechanism currently for a physician conducted skin examination; and there is little assistance for the uninsured.

## SUMMARY

The many complexities described lead to difficulty when implementing skin cancer programs. Protection from UV radiation exposure—shade seeking, staying out of the sun during the peak hours of UV radiation, and wearing protective clothing—is likely to be effective. However, the use of any one of these preventive factors is infrequent. Current attempts to limit solar exposure through reduced exposure and sun protection are only partially successful. Despite extensive publicity campaigns, rates of melanoma in Australia are still increasing. In Queensland, the most recent analysis shows that invasive melanoma is increasing at a rate of 2.5 percent per year in males and 1.2 percent per year in females; with most of the increase among the thinner lesions, although rates appear to be stabilizing among those under 35 years old.<sup>6</sup> There is some evidence that individuals can be taught skin self-examination (SSE). The Check It Out Project has shown that individuals can be relatively easily trained to reliably examine their own skin.<sup>86</sup> Some evidence suggests that those who feel comfortable with SSE do better, and that might account for the difference in mortality (under study now).

Until we have better data on how to prevent skin cancer of all types, it makes sense to practice as much sun protection as possible (after getting that 15 minutes of UV exposure three times a week for vitamin D synthesis) and to practice SSE in combination with physician skin examination (either primary care practitioner or dermatologist) to evaluate any concerning skin lesions that may appear.

## Acknowledgments

This work was supported by Grant CA112524 from the National Cancer Institute.

## REFERENCES

1. Mouw T, Koster A, Wright ME, et al. Education and risk of cancer in a large cohort of men and women in the United States. *PLoS ONE*. 2008; 3:e3639. [PubMed: 18982064]
2. Shack L, Jordan C, Thomson CS, et al. Variation in incidence of breast, lung and cervical cancer and malignant melanoma of the skin by socioeconomic group in England. *BMC Cancer*. 2008; 8:271. [PubMed: 18822122]
3. Birch-Johansen F, Huilsum G, Kjaer T, et al. Social inequality and incidence of and survival from malignant melanoma in a population-based study in Denmark, 1994–2003. *Eur J Cancer*. 2008; 44:2043–9. [PubMed: 18664405]

4. Parkin DM, Bray F, Ferlay J, et al. Global cancer statistics, 2002. *CA Cancer J Clin.* 2005; 55:74–108. [PubMed: 15761078]
5. American Cancer Society. *Cancer facts & figures 2008.* American Cancer Society; Atlanta (GA): 2008.
6. Baade P, Coory M. Trends in melanoma mortality in Australia: 1950-2002 and their implications for melanoma control. *Aust N Z J Public Health.* 2005; 29(4):383–6. [PubMed: 16222938]
7. Qin J, Berwick M, Ashbolt R, et al. Quantifying the change of melanoma incidence by Breslow thickness. *Biometrics.* 2002; 58(3):665–70. [PubMed: 12230002]
8. Welch HG, Woloshin S, Schwartz LM. Skin biopsy rates and incidence of melanoma: population based ecological study. *BMJ.* 2005; 331:481. [PubMed: 16081427]
9. Gandini S, Sera F, Cattaruzza MS, et al. Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *Eur J Cancer.* 2005; 41(1):45–60. [PubMed: 15617990]
10. Elwood JM, Jopson J. Melanoma and sun exposure: an overview of published studies. *Int J Cancer.* 1997; 73:198–203. [PubMed: 9335442]
11. Nelemans PJ, Rampen FH, Ruiten DJ, et al. An addition to the controversy on sunlight exposure and melanoma risk: a meta-analytical approach. *J Clin Epidemiol.* 1995; 48(11):1331–42. [PubMed: 7490596]
12. Armstrong BK. Epidemiology of malignant melanoma: intermittent or total accumulated exposure to the sun? *J Dermatol Surg Oncol.* 1988; 14(8):835–49. [PubMed: 3397443]
13. Berwick M, Chen YT. Reliability of reported sunburn history in a case-control study of cutaneous malignant melanoma. *Am J Epidemiol.* 1995; 141(11):1033–7. [PubMed: 7771439]
14. Whiteman D, Green A. Melanoma and sunburn. *Cancer Causes Control.* 1994; 5(6):564–72. [PubMed: 7827244]
15. MacKie RM, Aitchison T. Severe sunburn and subsequent risk of primary cutaneous malignant melanoma in Scotland. *Br J Cancer.* 1982; 46(6):955–60. [PubMed: 7150488]
16. Grob JJ, Gouvernet J, Aymar D, et al. Count of benign melanocytic nevi as a major indicator of risk for nonfamilial nodular and superficial spreading melanoma. *Cancer.* 1990; 66(2):387–95. [PubMed: 2369719]
17. Thomas NE, Edmiston SN, Alexander A. Number of nevi and early-life ambient UV exposure are associated with BRAF-mutant melanoma. *Cancer Epidemiol Biomarkers Prev.* 2007; 16(5):991–7. [PubMed: 17507627]
18. Whiteman DC, Watt P, Purdie DM, et al. Melanocytic nevi, solar keratoses, and divergent pathways to cutaneous melanoma. *J Natl Cancer Inst.* 2003; 95(11):806–12. [PubMed: 12783935]
19. Berwick M. Are tanning beds “safe”? Human studies of melanoma. *Pigment Cell Melanoma Res.* 2008; 21(5):517–9. [PubMed: 18821856]
20. The International Agency for Research on Cancer Working Group on artificial ultraviolet (UV) light and skin cancer. *Int J Cancer.* 2006; 120:1116–22.
21. Veierød M, Weiderpass E, Thöm M, et al. A prospective study of pigmentation, sun exposure, and risk of cutaneous malignant melanoma in women. *J Natl Cancer Inst.* 2003; 95(20):1530–8. [PubMed: 14559875]
22. Massi D, Carli P, Franchi A, et al. Naevus-associated melanomas: cause or chance? *Melanoma Res.* 1999; 9(1):85–91. [PubMed: 10338338]
23. Skender-Kalnenas TM, English DR, Heenan PJ. Benign melanocytic lesions: risk markers or precursors of cutaneous melanoma? *J Am Acad Dermatol.* 1995; 33(6):1000–7. [PubMed: 7490345]
24. Olsen CM, Zens MS, Stukel TA, et al. Nevus density and melanoma risk in women: A pooled analysis to test the divergent pathway hypothesis. *Int J Cancer.* 2008; 124(4):937–44. [PubMed: 19035450]
25. Chang YM, Newton-Bishop JA, Bishop DT, et al. A pooled analysis of melanocytic nevus phenotype and the risk of cutaneous melanoma at different latitudes. *Int J Cancer.* 2008; 124(2):420–8. [PubMed: 18792098]
26. Gandini S, Sera F, Cattaruzza MS, et al. Meta-analysis of risk factors for cutaneous melanoma: I. Common and atypical naevi. *Eur J Cancer.* 2005; 41(1):28–44. [PubMed: 15617989]

27. Greene, MH.; Fraumeni, JJ. The hereditary variant of malignant melanoma. In: Clark, WH.; Goldman, LI.; Mastrangelo, MJ., editors. Human malignant melanoma. Grune & Stratton; New York: 1979. p. 139-66.
28. Kopf AW, Hellman LJ, Rogers GS, et al. Familial malignant melanoma. *J Am Med Assoc.* 1986; 256:1915-9.
29. Ford D, Bliss JM, Swerdlow AJ, et al. Risk of cutaneous melanoma associated with a family history of the disease. *Int J Cancer.* 1995; 62:377-81. [PubMed: 7635561]
30. Fitzpatrick TB. The validity and practicality of sun-reactive skin types I through VI. *Arch Dermatol.* 1988; 124:869-71. [PubMed: 3377516]
31. Rampen FH, Fleuren BA, de Boo TM, et al. Unreliability of self-reported burning tendency and tanning ability. *Arch Dermatol.* 1988; 124(6):885-8. [PubMed: 3377517]
32. Raimondi S, Sera F, Gandini S, et al. MC1R variants, melanoma and red hair color phenotype: a meta-analysis. *Int J Cancer.* 2008; 122(12):2753-60. [PubMed: 18366057]
33. Gudbjartsson DF, Sulem P, Stacey SN, et al. MC1R. *Nat Genet.* 2008; 40(7):886-91. [PubMed: 18488027]
34. Brown KM, Macgregor S, Montgomery GW, et al. Common sequence variants on 20q11.22 confer melanoma susceptibility. *Nat Genet.* 2008; 40(7):838-40. [PubMed: 18488026]
35. Kertat K, Rosdahl I, Sun XF, et al. The Gln/Gln genotype of XPD codon 751 as a genetic marker for melanoma risk and Lys/Gln as an important predictor for melanoma progression: a case control study in the Swedish population. *Oncol Rep.* 2008; 20(1):179-83. [PubMed: 18575735]
36. Millikan RC, Hummer A, Begg C, et al. Polymorphisms in nucleotide excision repair genes and risk of multiple primary melanoma: the Genes Environment and Melanoma Study. *Carcinogenesis.* 2006; 27(3):610-8. [PubMed: 16258177]
37. Kadearo AL, Wakamatsu K, Ito S, et al. Cutaneous photoprotection and melanoma susceptibility: reaching beyond melanin content to the frontiers of DNA repair. *Front Biosci.* 2006; 11:2157-73. [PubMed: 16720302]
38. Han J, Colditz GA, Hunter DJ. Lack of associations of selected variants in genes involved in cell cycle and apoptosis with skin cancer risk. *Cancer Epidemiol Biomarkers Prev.* 2006; 15(3):592-3. [PubMed: 16537723]
39. Berwick M, Orlow I, Hummer AJ, et al. The prevalence of CDKN2A germ-line mutations and relative risk for cutaneous malignant melanoma: an international population-based study. *Cancer Epidemiol Biomarkers Prev.* 2006; 15(8):1520-5. [PubMed: 16896043]
40. Kefford R, Bishop JN, Tucker M, et al. Genetic testing for melanoma. *Lancet Oncol.* 2002; 3(11):653-4. [PubMed: 12424065]
41. Armstrong BK, Kricger A, English DR. Sun exposure and skin cancer. *Australas J Dermatol.* 1997; 38(Suppl 1):S1-6. [PubMed: 10994463]
42. American Cancer Society. [Accessed 2009] Available at: [www.cancer.org](http://www.cancer.org).
43. Hall HI, May DS, Lew RA, et al. Sun protection behaviors of the U.S. white population. *Prev Med.* 1997; 26(4):401-7. [PubMed: 9245656]
44. Koh HK, Bak SM, Geller AC, et al. Sunbathing habits and sunscreen use among white adults: results of a national survey. *Am J Public Health.* 1997; 87(7):1214-7. [PubMed: 9240117]
45. Coups EJ, Manne SL, Heckman CJ. Multiple skin cancer risk behaviors in the U.S. population. *Am J Prev Med.* 2008; 34(2):87-93. [PubMed: 18201637]
46. Dobbins SJ, Wakefield MA, Jansen KM, et al. Weekend sun protection and sunburn in Australia trends (1987-2002) and association with SunSmart television advertising. *Am J Prev Med.* 2008; 34(2):94-101. [PubMed: 18201638]
47. Montague M, Borland R, Sinclair C. Slip! Slop! Slap! and SunSmart, 1980-2000: Skin cancer control and 20 years of population-based campaigning. *Health Educ Behav.* 2001; 28(3):290-305. [PubMed: 11380050]
48. Stanton WR, Moffatt J, Clavarino A. Community perceptions of adequate levels and reasons for skin protection. *Behav Med.* 2005; 31(1):5-15. [PubMed: 16078522]

49. Saraiya M, Glanz K, Briss P. 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. [PubMed: 14561953]
50. Rutten L, Hesse BW, Moser RP, et al. Public understanding of cancer prevention, screening, and survival: Comparison with state-of-science evidence for colon, skin, and lung cancer. *J Cancer Educ.* in press.
51. Geller AC, Emmons KM, Brooks DR, et al. A randomized trial to improve early detection and prevention practices among siblings of melanoma patients. *Cancer.* 2006; 107(4):806–14. [PubMed: 16832795]
52. Mayer JA, Lewis EC, Eckhardt L, et al. Promoting sun safety among zoo visitors. *Prev Med.* 2001; 33(3):162–9. [PubMed: 11522156]
53. Weinstock MA, Rossi JS, Redding CA, et al. Randomized controlled community trial of the efficacy of a multicomponent stage-matched intervention to increase sun protection among beachgoers. *Prev Med.* 2002; 35(6):84–92.
54. Buller DB, Andersen PA, Walkosz BJ, et al. Randomized trial testing a worksite sun protection program in an outdoor recreation industry. *Health Educ Behav.* 2005; 32:514–35. [PubMed: 16009748]
55. Glanz K, Geller AC, Shigaki D, et al. A randomized trial of skin cancer prevention in aquatic settings: the Pool Cool program. *Health Psychol.* 2002; 21(6):579–87. [PubMed: 12433010]
56. Gorham ED, Mohr SB, Garland CF, et al. Do sunscreens increase risk of melanoma in populations residing at higher latitudes? *Ann Epidemiol.* 2007; 17(12):956–63. [PubMed: 18022535]
57. Vainio H, Miller AB, Bianchini F. An international evaluation of the cancer-preventive potential of sunscreens. *Int J Cancer.* 2000; 88(5):838–42. [PubMed: 11072258]
58. Autier P, Boniol M, Dore JF. Sunscreen use and increased duration of intentional sun exposure: still a burning issue. *Int J Cancer.* 2007; 121(1):2755–9.
59. Diffey BL. Sunscreens and melanoma: the future looks bright. *Br J Dermatol.* 2005; 153(2):378–81. [PubMed: 16086753]
60. Gallagher RP, Rivers KJ, Lee TK. Broad-spectrum sunscreen use and the development of new nevi in white children: a randomized controlled trial. *JAMA.* 2000; 283(22):2955–60. [PubMed: 10865273]
61. Whiteman DC, Brown RM, Purdie DM, et al. Melanocytic nevi in very young children: the role of phenotype, sun exposure, and sun protection. *J Am Acad Dermatol.* 2005; 52(1):40–7. [PubMed: 15627079]
62. van der Pols JC, Xu C, Boyle GM, et al. Expression of p53 tumor suppressor protein in sun-exposed skin and associations with sunscreen use and time spent outdoors: a community-based study. *Am J Epidemiol.* 2006; 163(11):982–8. [PubMed: 16624969]
63. Bauer J, Büttner P, Wiecker TS, et al. Effect of sunscreen and clothing on the number of melanocytic nevi in 1,812 German children attending day care. *Am J Epidemiol.* 2005; 161(7):620–7. [PubMed: 15781951]
64. English DR, Milne E, Simpson JA. Sun protection and the development of melanocytic nevi in children. *Cancer Epidemiol Biomarkers Prev.* 2005; 14(12):2873–6. [PubMed: 16365003]
65. English DR, Milne E, Simpson JA. Ultraviolet radiation at places of residence and the development of melanocytic nevi in children (Australia). *Cancer Causes Control.* 2006; 17(1):103–7. [PubMed: 16411059]
66. Autier P, Doré 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(24):1873–80. [PubMed: 9862624]
67. Gallagher RP, Elwood JM, Yang CP. Is chronic sunlight exposure important in accounting for increases in melanoma incidence? *Int J Cancer.* 1989; 44(5):813–5. [PubMed: 2583861]
68. Diffey B. Do white British children and adolescents get enough sunlight? *BMJ.* 2005; 331:3–4. [PubMed: 15994663]
69. Hiom S. Public awareness regarding UV risks and vitamin D—the challenges for UK skin cancer prevention campaigns. *Prog Biophys Mol Biol.* 2006; 92(1):161–6. [PubMed: 16624385]

70. Working Group of the Australian and New Zealand Bone and Mineral Society; Endocrine Society of Australia and Osteoporosis Australia. Vitamin D and adult bone health in Australia and New Zealand: a position statement. *Med J Aust.* 2005; 182(6):281–5. [PubMed: 15777143]
71. Breitbart EW, Greinert R, Volkmer B. Effectiveness of information campaigns. *Prog Biophys Mol Biol.* 2006; 92(1):167–72. [PubMed: 16595143]
72. Brooks KR, Brooks DR, Hufford D, et al. Are television stations and weather pages still reporting the UV index? A national media follow-up study. *Arch Dermatol.* 2005; 141(4):526. [PubMed: 15837879]
73. Dixon, H.; Armstrong, B. Report of a national workshop on its role in sun protection. NSW Cancer Council and Anti-Cancer Council of Victoria; Sydney: 1999. The UV index.
74. Arthey S, Clarke VA. Suntanning and sun protection: a review of the psychological literature. *Soc Sci Med.* 1995; 40(2):265–74. [PubMed: 7899938]
75. Berwick M, Fine JA, Bolognia JL. Sun exposure and sunscreen use following a community skin cancer screening. *Prev Med.* 1992; 21(3):302–10. [PubMed: 1614992]
76. Grob JJ, Guglielmina C, Gouvernet J, et al. Study of sunbathing habits in children and adolescents: application to the prevention of melanoma. *Dermatology.* 1993; 186(2):94–8. [PubMed: 8428054]
77. Jackson KM, Aiken LS. A psychosocial model of sun protection and sunbathing in young women: the impact of health beliefs, attitudes, norms, and self-efficacy for sun protection. *Health Psychol.* 2000; 19(5):469–78. [PubMed: 11007155]
78. Hoerster KD, Mayer JA, Woodruff SI, et al. The influence of parents and peers on adolescent indoor tanning behavior: findings from a multi-city sample. *J Am Acad Dermatol.* 2007; 57(6):990–7. [PubMed: 17658194]
79. Young AR, Potten CS, Chadwick CA, et al. Photoprotection and 5-MOP photochemoprotection from UVR-induced DNA damage in humans: the role of skin type. *J Invest Dermatol.* 1991; 97(5):942–8. [PubMed: 1919058]
80. Hay JL, Meischke HW, Bowen DJ, et al. Anticipating dissemination of cancer genomics in public health: a theoretical approach to psychosocial and behavioral challenges. *Ann Behav Med.* 2007; 34(3):275–86. [PubMed: 18020937]
81. Wartman D, Weinstock M. Are we overemphasizing sun avoidance in protection from melanoma? *Cancer Epidemiol Biomarkers Prev.* 2008; 17(3):469–70. [PubMed: 18319330]
82. Epstein DS, Lange JR, Gruber SB, et al. Is physician detection associated with thinner melanomas? *JAMA.* 1999; 281(7):640–3. [PubMed: 10029126]
83. US Preventive Services Task Force. Recommendations and rationale for screening for skin cancer. *Am J Prev Med.* 2001; 203(Suppl):44–6.
84. Geller AC, Sober AJ, Zhang Z, et al. Strategies for improving melanoma education and screening for men age > or = 50 years: findings from the American Academy of Dermatological National Skin Cancer Screening Program. *Cancer.* 2002; 95(7):1554–61. [PubMed: 12237925]
85. Pennie ML, Soon SL, Risser JB, et al. Melanoma outcomes for Medicare patients: association of stage and survival with detection by a dermatologist vs a non-dermatologist. *Arch Dermatol.* 2007; 143(4):488–94. [PubMed: 17438181]
86. Weinstock MA, Risica PM, Martin RA, et al. Melanoma early detection with thorough skin self-examination: the “Check It Out” randomized trial. *Am J Prev Med.* 2007; 32(6):517–24. [PubMed: 17533068]

**Table 1**

## Fitzpatrick classification of skin type

<b>Skin Type</b>	<b>Skin Color</b>	<b>Characteristics</b>
I	White, very fair, red or blond hair, blue eyes, freckles	Always burns, never tans
II	White; fair; red or blond hair; blue, hazel, or green eyes	Usually burns, tans with difficulty
III	Cream white, fair with any eye or hair color, very common	Sometimes mild burn, gradually tans
IV	Brown, typical Mediterranean Caucasian skin	Rarely burns, tans with ease
V	Dark Brown, Mid-Eastern skin types	very rarely burns, tans very easily
VI	Black	Never burns, tans very easily

*From Fitzpatrick TB. The validity and practicality of sun-reactive skin types I through VI. 1988; Arch Dermatol 124:869-71; with permission.*