Published in final edited form as:
Curr Opin Oncol. 2009 March ; 21(2): 144–149. doi:10.1097/CCO.0b013e3283252fc5.

Indoor UV tanning and skin cancer: health risks and opportunities

Joshua M. Schulman and **David E. Fisher**
Department of Dermatology, Cutaneous Biology Research Center, and Melanoma Program, Massachusetts General Hospital, Boston, MA, USA

Abstract

Purpose of review—Skin cancer incidence is higher than that of any other human malignancy, and yet one of its root causes (ultraviolet radiation) is perhaps better understood than any other human carcinogen. The roles of ultraviolet radiation exposure and indoor tanning behaviors on skin cancer risk are explored here.

Recent findings—Studies from the past several years have shown a significant association between ever-use of an indoor tanning facility and an increased risk of basal cell carcinoma, squamous cell carcinoma, and melanoma. The association between indoor tanning and skin cancer is particularly strong among those who first used a tanning facility in early adulthood. Elevated vitamin D levels have been suggested to protect against various internal malignancies and other disease states, but sources of vitamin D that do not require UV exposure are easily available.

Summary—Although additional research is needed to understand fully the relationship between UV and skin cancer, it is already clear that indoor tanning bed use represents an avoidable risk factor for melanoma and non-melanoma skin cancer – both of which may be lethal. Acting upon this information provides a unique opportunity for protecting the public health.

Keywords

indoor tanning; skin cancer; vitamin D; regulation

One of the striking paradoxes of modern medicine is the extremely high frequency of skin cancer despite an enormous body of evidence that identifies UV radiation as a skin carcinogen. Among UV sources, indoor tanning represents a growing, multi-billion dollar industry, though its growth comes at a time when there is scientific consensus that exposure to ultraviolet (UV) radiation increases the risk of developing skin cancer. As evidence of the dangers of UV exposure mounts, the indoor tanning industry has responded with an aggressive public relations campaign, which appears designed to reassure the general public that health benefits of tanning may outweigh the health risks. This public relations campaign reached a peak on March 26, 2008, when the industry published a full-page advertisement in *The New York Times*, claiming that an association between tanning and melanoma was "hype." The advertisement continued:

> "Recent research indicates that the benefits of moderate exposure to sunlight outweigh the hypothetical risks. Surprisingly, there is no compelling scientific evidence that tanning causes melanoma. Scientists have proven, however, that exposure to all forms of ultraviolet light – both indoors and out – stimulates the

Corresponding Author: David E. Fisher, Massachusetts General Hospital, 55 Fruit St, Bartlett Hall 622, Boston, MA 02114, USA. Tel: 617-643-5428. dfisher3@partners.org.

> natural production of vitamin D. And research has proven that vitamin D protects against heart disease and many types of cancer, in addition to providing other important health benefits. It's time to rethink sun bathing" [1].

This powerful advertisement bears scrutiny, because it simultaneously conveys certain scientific truths while confusing or distorting others, in a fashion that may dangerously affect the public's perception of true medical risk. This article presents a review of the current state of scientific evidence concerning tanning and skin cancer risk (including the risk of melanoma), the health outcomes associated with vitamin D, and the true importance of "rethinking sun bathing" to protect the public health.

Tanning and Skin Cancer

Skin is the most common organ to be affected by cancer in the United States, and the incidence of skin cancer has been rising rapidly over the past several decades, especially among younger adults. The causes of this trend are multi-factorial, but UV exposure patterns are broadly accepted as contributory, as fits the extensive evidence that UV radiation induces DNA damage in the skin, which can initiate carcinogenesis [2,3]. In particular, the aromatic heterocyclic bases in DNA absorb strongly in the wavelength range of UVB, leading to the generation of cyclobutane pyrimidine dimers that cause C→T and CC→TT mutations [4]. Recent evidence suggests that radiation in the UVA range can also trigger DNA damage via cyclobutane pyrimidine dimer formation [5]. This DNA photodamage can be repaired through mechanisms involving the tumor suppressor p53 (or, in cases of more extensive damage, p53 plays a role in regulating apoptosis), though p53 itself is subject in the skin to dipyrimidine mutagenesis. Thus, UV-mediated DNA damage has carcinogenic potential both by initiating mutations throughout the genome and by contributing to loss of p53 function [6,7].

Importantly, DNA damage also initiates the tanning pathway. When activated in response to DNA damage in keratinocytes, p53 binds to and upregulates transcription of the pro-opiomelanocortin (POMC) gene [8**] (Figure 1). The POMC polypeptide is post-translationally cleaved into several products, including α-melanocyte-stimulating hormone, which then signals to melanocytes via the melanocortin 1 receptor (MC1R) [9]. If signaling through this receptor is disrupted, tanning does not occur, as in red-haired individuals who harbor loss-of-function polymorphisms of MC1R and burn in response to sun exposure without tanning [10]. In other words, UV-mediated DNA damage can occur in some individuals in the absence of tanning, but tanning does not apparently occur without antecedent DNA damage. These data cast significant doubt over the theoretical possibility of a truly "safe tan."

While sun exposure is a known risk factor for developing skin cancer, particularly squamous cell carcinoma (SCC) and basal cell carcinoma (BCC), comparatively less attention has been directed at indoor tanning as a risk factor. An important study from 2002 addressed this question, finding that indoor tanning does contribute significantly to the risk of developing SCC and BCC [11]. Based on a sample of 603 BCC patients and 293 SCC patients, along with 540 controls, this study found that ever having used a tanning device resulted in a 50% increase in BCC risk (OR = 1.5, 95% CI = 1.1-2.1) and more than doubled the risk of developing SCC (OR = 2.5, 95% CI = 1.7-3.8). Age at first use of a tanning device was also a significant risk factor, with age less than 20 years associated with the highest risk for both BCC and SCC.

The tanning industry has attempted to downplay the significance of the elevated BCC and SCC risk, arguing that these forms of skin cancer are generally less aggressive than melanoma. Such an argument ignores the fact that non-melanoma skin cancer, especially

SCC, does have metastatic potential and is likely responsible for thousands of potentially avoidable deaths each year in the United States [3]. Additionally, the morbidity associated with surgical excision of non-lethal BCC and SCC and the utilization of healthcare resources to treat these preventable cancers are substantial.

Nonetheless, by shifting the discussion away from BCC and SCC and toward melanoma, the tanning industry has all but invited scientific inquiry into the association between indoor tanning and melanoma risk. Early studies of this association produced equivocal results, perhaps because indoor tanning has gained in popularity only over the past thirty years, and, due to the lag time characteristic of melanoma development, early studies may not have allowed for a sufficiently long post-exposure period to adequately assess melanoma risk. Indeed, a recent case-control study by Clough-Gorr et al produced equivocal results for the same reason; the study found a non-statistically significant increase in melanoma risk for individuals who had ever used a tanning bed (OR = 1.14, 95% CI = 0.80-1.61), but their study population had, on average, only begun using tanning beds 17 years prior to the time of data collection, which corresponded to a later age at first use [12*]. By contrast, a recent report by Ting et al enrolled a slightly younger population, composed of 1518 dermatologic patients, among whom there were 79 cases of malignant melanoma [13*]. This study found that ever using a tanning bed was associated with an increased risk of developing malignant melanoma (OR = 1.64, 95% CI = 1.01-2.67), and, importantly, this risk was particularly pronounced among women aged 45 years or younger (OR = 3.22, 95% CI = 1.01-11.46).

A landmark study published in 2007 by the International Agency for Research on Cancer confirms the association between indoor tanning and melanoma [14**]. The authors conducted a meta-analysis of all 19 reports published to that time that evaluated indoor tanning behavior and malignant melanoma risk. The included reports represented a total of 7,355 cases across 3 continents and spanned 24 years of publication. In total, having ever used a tanning bed was associated with an increased risk of melanoma (summary RR = 1.15, 95% CI = 1.00-1.31). Out of the 19 reports initially included in the meta-analysis, 7 contained data on age at first use of a tanning bed. Based on those 7 studies, first exposure to indoor tanning before age 35 years was associated with a 75% increased risk of developing melanoma (summary RR = 1.75, 95% CI = 1.35-2.26), as shown in Figure 2.

Importantly, numerous laboratory models suggest that relatively low-dose UV exposure, which would not be sufficient to generate erythema (“sunburn”), is still able to induce skin carcinogenesis [15*]. Thus, the notion of “responsible tanning” is, together with “safe tanning” mechanistically incongruous since the ability to induce measurable tanning shares a carcinogenic intermediate.

Vitamin D and Disease Prevention

In addition to causing DNA damage and promoting carcinogenesis, UV exposure to the skin does have a positive effect in its ability to convert 7-dehydrocholesterol to pre-vitamin D, which is converted by the liver and kidneys to active vitamin D. Vitamin D has long been known to be critical for skeletal health, and more recently it has been suggested to reduce the risk of mortality from a variety of cancers, presumably by signaling events at vitamin D receptors that can induce cellular differentiation and inhibit proliferation [16]. While the data supporting these activities are reviewed below, it is important to emphasize that the virtues of vitamin D can be fully uncoupled from the risks related to UV exposure, because vitamin D can be easily obtained in oral supplements (pill form) and is routinely recommended by internists, frequently together with appropriate monitoring of levels during routine blood chemistry analyses. Thus it is possible to embrace the benefits of vitamin D without subjecting oneself to UV carcinogenic exposure.

Colon Cancer

Some of the strongest epidemiological evidence supporting an association between vitamin D levels and cancer mortality comes from the study of colon cancer. In one of the largest such studies, cancer mortality and baseline vitamin D status were assessed for 16,818 volunteers enrolled in the NHANES III study, from which there were 66 colon cancer deaths over a period of 146,578 person-years [17*]. Serum levels of vitamin D greater than or equal to 80 nmol/L were associated with lower colorectal mortality risk than levels below 50 nmol/L (RR = 0.28, 95% CI = 0.11-0.68). In a separate case-control study nested within the Health Professionals Follow-up Study (HPFS) (n = 179 cases), similar results were reported: men in the highest quartile for serum vitamin D level (median 96.8 nmol/L) had a significantly reduced risk of colon cancer compared to men in the lowest quartile (median 48.2 nmol/L) (adjusted OR = 0.46, 95% CI = 0.24-0.89) [18*]. When pooled with women from the Nurses Health Study (n = 193 additional cases), this association for colon cancer risk persisted (adjusted OR = 0.54, 95% CI = 0.34-0.86). As with many studies described here, it is obviously important to interpret association studies cautiously, since the relationship may be indirect (eg low vitamin D may be coincidently present, but mechanistically uninvolved in colorectal cancer risk, and thus an inappropriate target for modulating risk).

Prostate Cancer

For non-colon cancers, an association between vitamin D status and disease risk is somewhat less clear. In one recent case-control study of prostate cancer, for example, Ahn et al found that higher pre-diagnosis levels of vitamin D correlated with more aggressive disease (Gleason 7 or above), whereas disease incidence did not vary with vitamin D level [19*]. In another study, Li et al examined circulating vitamin D levels and vitamin D receptor polymorphisms in 1066 prostate cancer cases and 1618 matched controls; overall, there was no association between vitamin D status and prostate cancer, except in a subgroup analysis of men with below-median levels of both 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D (the two circulating forms, the latter of which is the active hormone), who had an increased risk of aggressive disease (OR = 2.06, 95% CI = 1.24-3.43) [20*]. The implications of this finding require further clarification, as low levels of each vitamin D form individually did not correlate with disease state. However, some insight may come from the analysis of vitamin D receptor polymorphisms, where it was found that among men with low 25-hydroxyvitamin D levels, those who also carried a loss-of-function polymorphism (FokI ff) had an increased risk of total (OR = 1.9, 95% CI = 1.1-3.3) and aggressive (OR = 2.5, 95% CI = 1.1-5.8) prostate cancer [20*]. Thus, it is likely not vitamin D levels alone, but rather the function of the entire vitamin D axis, that may influence the development and progression of prostate cancer.

Non-Hodgkin Lymphoma

Significant attention has also been directed recently at non-Hodgkin lymphoma, though with this malignancy studies have focused more on sun exposure than on vitamin D levels. For example, in a case-control study from Germany involving 710 lymphoma patients and an equal number of controls, increased number of vacation days spent at sunny locations was associated with a decreased risk of developing lymphoma (OR = 0.6, 95% CI = 0.4-0.8) [21]. Similarly, total hours of sun exposure per week had a weak negative association with non-Hodgkin lymphoma risk among individuals living in Nebraska [22]. A major meta-analysis combining data from 10 studies covering 8,243 cases and 9,697 controls further supports these claims; individuals who had the highest levels of recreational sun exposure were found to have a decreased risk of non-Hodgkin lymphoma (pooled OR = 0.76, 95% CI

= 0.63-0.91) [23*]. It is important to note that sun exposure does not necessarily correlate with vitamin D levels, and, in fact, when vitamin D intake was measured, it was found not to be associated with lymphoma risk [22]. Furthermore, vitamin D receptor polymorphisms have been shown not to have a statistically significant association with lymphoma risk [24]. Thus, in the case of non-Hodgkin lymphoma, sun exposure may affect disease risk through a different mechanism than vitamin D (for example, by UV-mediated immunomodulation in the skin), or it may alternatively reflect a confounding variable (such as exercise frequency or baseline health status).

Cardiovascular Disease

There is also an increasing body of evidence associating vitamin D levels with other non-cancer disease states. In a significant report from the NHANES III study, 7186 men and 7902 women were evaluated for both vitamin D status and cardiovascular risk factors [25**]. Compared to subjects in the highest quartile of vitamin D levels (greater than 92 nmol/L), those in the lowest quartile (less than 52 nmol/L) were more likely after adjusting for age, sex, and race to have elevated blood pressure (OR = 1.30, 95% CI = 1.13-1.49), a history of diabetes (OR = 1.73, 95% CI = 1.38-2.16) and an elevated triglyceride level (OR = 1.47, 95% CI = 1.30-1.65). These results reinforce the importance of vitamin D to overall health, but they do not imply that intentional UV exposure is necessary for attaining optimal vitamin D levels. While frequent indoor tanning can be associated with elevated serum vitamin D, non-carcinogenic alternatives such as dietary intake and oral supplements should be preferentially considered [26].

Tanning Trends and Regulation

In 1988, as few as 1% of American adults reported using indoor tanning facilities; by 2007, that number had increased to 27% [27**]. Other reports have found even higher levels of tanning among younger adults, particularly young white women [28]. At the same time that tanning rates are increasing, many tanning devices are employing more powerful UV lamps: a recent study from England found that 83% of tanning beds evaluated exceeded European standards for UV-B radiation levels, and similar findings of increased UV intensity have been reported in Norway [29*,30]. Combined, these results portend a potential looming public health crisis, with tens of millions of individuals putting themselves at increased risk of developing BCC, SCC, and melanoma in the years ahead.

Future prospects

How can this information be utilized optimally relative to the escalating incidence of skin cancers? First, education of the general public regarding the risks of UV exposure, including from indoor tanning devices, is necessary. The ability of UV to induce cutaneous vitamin D production is not a persuasive argument supporting UV-tanning, because of non-UV means to obtain identical (if not more accurate) maintenance of healthy blood vitamin D levels. In one study, 67% of subjects surveyed in 2007 were aware that limiting tanning decreases the risk of developing skin cancer, but that proportion was down from 77% in 1994 [27**]. In light of the indoor tanning industry's current public relations campaign to dissociate tanning from melanoma risk, knowledge of the dangers posed by intentional UV exposure is likely only to decrease unless an effective counter-message is offered by the biomedical community.

Second, physician counseling of patients who engage in intentional tanning should be considered. Several recent studies have found that 12-26% of frequent tanners, particularly adolescents and young adults, show signs of addiction to tanning, which may be related to endogenous opioids that are released as a byproduct of the tanning pathway [31,32*,33*,

8**]. Physicians may therefore need to view tanning similarly to cigarette smoking or alcohol use: as a potentially addictive behavior that can increase the risk of future malignancies.

Third, political action may be required to expand and enforce regulation of the tanning industry. Twenty states currently have laws regarding indoor tanning, most of which regulate access by minors to tanning facilities [34,35]. However, mechanisms to enforce these regulations are not currently sufficient. In one study conducted in Minnesota and Massachusetts, 15 year-old girls were sent to 200 indoor tanning facilities and attempted to purchase tanning sessions without parental consent, in violation of state law; the girls succeeded in purchasing sessions at 81% of facilities [36]. Given that early exposure to indoor tanning is associated with the greatest increase in risk of developing melanoma, improved regulation of the tanning industry, especially as it concerns youth access, is of vital public health concern. It may also be cost-effective: in a recent analysis in Austraila, strict regulation of tanning access was estimated to save $AU 256,054 in eventual healthcare costs per 100,000 persons [37**]. Importantly “Tanning Salons” sometimes offer customers non-UV tanning options, which represent cosmetic alternatives that are likely to be profoundly safer (many of these cosmetic darkening products may contain sunblocks). The switch to such marketing strategies may profoundly alter the skin cancer risk for clients.

Conclusions

Despite claims by the tanning industry, recent evidence supports an association between indoor tanning and increased risk of skin cancers, including BCC, SCC, and melanoma. While association studies have correlated vitamin D levels with protection against various internal malignancies and other disease states, acquiring vitamin D by cutaneous UV exposure is not necessary, and non-carcinogenic sources, such as dietary supplementation, are to be preferred. Moreover prospective randomized trials are needed (and some are underway) to directly test the role(s) of vitamin D supplements at varied doses relative to a variety of cancer endpoints. Given the high prevalence of indoor and outdoor tanning, especially among younger individuals, it is a public health imperative to educate the general population about associated risks and set in place opportunities to utilize this information to more effectively prevent skin cancer.

Acknowledgments

Disclosure of funding: DEF discloses consulting relationships with Novartis Pharmaceuticals, Magen BioSciences, and Source MDx.

References

1. Indoor Tanning Association. [9/21/08]. http://www.sunlightscam.com
2. Gandini S, Sera F, Cattaruzza MS, et al. Meta-analysis of risk factors for cutaneous melanoma: II Sun exposure. Eur J Cancer 2005;41:45–60. [PubMed: 15617990]
3. Ibrahim SF, Brown MD. Tanning and cutaneous malignancy. Dermatol Surg 2008;34:460–74. [PubMed: 18248469]
4. Ravanat JL, Douki T, Cadet J. Direct and indirect effects of UV radiation on DNA and its components. J Photochem Photobiol B 2001;63:88–102. [PubMed: 11684456]
5. Mouret S, Baudouin C, Charveron M, et al. Cyclobutane pyrimidine dimers are predominant DNA lesions in whole human skin exposed to UVA radiation. Proc Natl Acad Sci USA 2006;103:13765–70. [PubMed: 16954188]
6. Brash DE, Rudolph JA, Simon JA, et al. A role for sunlight in skin cancer: UV-induced p53 mutations in squamous cell carcinoma. Proc Natl Acad Sci USA 1991;88:10124–8. [PubMed: 1946433]

7. Brash DE, Ziegler A, Jonason AS, et al. Sunlight and sunburn in human skin cancer: p53, apoptosis, and tumor promotion. J Investig Dermatol Symp Proc 1996;1:136–42.

** 8. Cui R, Widlund HR, Feige E, et al. Central role of p53 in the suntan response and pathologic hyperpigmentation. Cell 2007;128:853–64. [PubMed: 17350573] By demonstrating the role of p53 as a transcriptional activator of the pro-opiomelanocortin gene, this study provides a molecular mechanism by which UV exposure causes tanning. Because p53 is activated in response to DNA damage, this study also suggests that UV-mediated tanning must occur secondary to DNA damage and therefore carries an inherent carcinogenic risk.

9. D'orazio JA, Nobuhisa T, Cui R, et al. Topical drug rescue strategy and skin protection based on the role of Mc1r in UV-induced tanning. Nature 2006;443:340–4. [PubMed: 16988713]

10. Valverde P, Healy E, Jackson I, et al. Variants of the melanocyte-stimulating hormone receptor gene are associated with red hair and fair skin in humans. Nat Genet 1995;11:328–30. [PubMed: 7581459]

11. 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;94:224–6. [PubMed: 11830612]

* 12. Clough-Gorr KM, Titus-Ernstoff L, Perry AE, et al. Exposure to sunlamps, tanning beds, and melanoma risk. Cancer Causes Control 2008;19:659–69. [PubMed: 18273687] This study found a modest association between tanning bed use and increased melanoma risk, but the relatively short post-exposure period suggests the need for longer-term retrospective analysis.

* 13. Ting W, Schultz K, Cac NN, et al. Tanning bed exposure increases the risk of malignant melanoma. Int J Dermatol 2007:1253–7. [PubMed: 18173518] In this study, ever-use, age at first use, and duration per session of tanning bed use were evaluated with regard to melanoma risk. All subtypes of use were found to be positively associated with melanoma risk, and risk was highest for individuals who began using tanning beds prior to age 45.

** 14. IARC International Agency for Research on Cancer Working Group on artificial ultraviolet light and skin cancer. The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers: a systematic review. Int J Cancer 2007;120:1116–22. [PubMed: 17131335] This major meta-analysis combined data from 19 prior studies to assess the relationship of indoor tanning to melanoma and non-melanoma skin cancer risk. Ever-use of a tanning bed was associated with an increased risk of melanoma, and use prior to age 35 increased the risk of melanoma by 75%. This study provides the most extensive evidence to date of the risk of melanoma associated with indoor tanning, especially as it pertains to youth tanning.

* 15. Halliday GM, Lyons JG. Inflammatory doses of UV may not be necessary for skin carcinogenesis. Photochem Photobiol 2008;84:272–83. [PubMed: 18353168] In a review of *in vitro*, *ex vivo*, and *in vivo* data, the authors summarize evidence that skin tumors can form after exposure to ultraviolet radiation that is below the threshold needed to induce inflammation. These findings are significant because they suggest that tanners are at risk of skin cancer even if they do not suffer sunburns.

16. Giovannucci E. The epidemiology of vitamin D and cancer incidence and mortality: a review (United States). Cancer Causes Control 2005;16:83–95. [PubMed: 15868450]

* 17. Freedman DM, Looker AC, Chang SC, Graubard BI. Prospective study of serum vitamin D and cancer mortality in the United States. J Natl Cancer Inst 2007;99:1594–602. [PubMed: 17971526] This extensive prospective analysis examined both overall cancer mortality and specific cancer mortalities in relation to serum vitamin D levels. While overall cancer mortality was not related to vitamin D status, colorectal cancer mortality was significantly decreased among individuals with elevated serum vitamin D.

* 18. Wu K, Feskanich D, Fuchs CS, et al. A nested case-control study of plasma 25-hydroxyvitamin D concentrations and risk of colorectal cancer. J Natl Cancer Inst 2007;99:1120–9. [PubMed: 17623801] This analysis looked specifically at colorectal cancer risk and its relationship to vitamin D status. In combining data from two large cohorts, the authors found a decreased risk of developing colon cancer in men and women with higher serum vitamin D levels.

* 19. Ahn J, Peters U, Albanes D, et al. Serum vitamin D concentration and prostate cancer risk: a nested case-control study. J Natl Cancer Inst 2008;100:796–804. [PubMed: 18505967] This study found an unexpected association between higher pre-diagnosis serum vitamin D levels and increased aggressiveness of subsequent prostate cancer. There was no association between

vitamin D status and prostate cancer incidence. The findings suggest a need for further examination of how vitamin D might influence the progression of prostate cancer.

* 20. Li H, Stampfer MJ, Hollis JBW, et al. A prospective study of plasma vitamin D metabolites, vitamin D receptor polymorphisms, and prostate cancer. PLoS Med 2007;4:e103. [PubMed: 17388667] In addition to analyzing the relationship between pre-diagnosis serum vitamin D levels and prostate cancer, this study also accounted for a polymorphism in the vitamin D receptor. The combination of low vitamin D levels and loss-of-function polymorphism was associated with increased disease risk and disease aggressiveness, suggesting that the entire vitamin D axis warrants examination in future studies.

21. Weihkopf T, Becker N, Nieters A, et al. Sun exposure and malignant lymphoma: a population-based case-control study in Germany. Int J Cancer 2007;120:2445–51. [PubMed: 17311289]

22. Soni LK, Hou L, Gapstur SM, et al. Sun exposure and non-Hodgkin lymphoma: a population-based, case-control study. Eur J Cancer 2007;43:2388–95. [PubMed: 17686627]

* 23. Kricker A, Armstrong BK, Hughes AM, et al. Personal sun exposure and risk of non Hodgkin lymphoma: a pooled analysis from the Interlymph Consortium. Int J Cancer 2008;122:144–54. [PubMed: 17708556] In a meta-analysis of 10 studies examining sun exposure history and non-Hodgkin lymphoma risk, the authors found that increased recreational sun exposure – but not necessarily total sun exposure – was associated with decreased risk of non-Hodgkin lymphoma.

24. Purdue MP, Hartge P, Davis S, et al. Sun exposure, vitamin D receptor gene polymorphisms and risk of non-Hodgkin lymphoma. Cancer Causes Control 2007;18:989–99. [PubMed: 17653830]

** 25. Martins D, Wolf M, Pan D, et al. Prevalence of cardiovascular risk factors and the serum levels of 25-hydroxyvitamin D in the United States. Arch Intern Med 2007;167:1159–65. [PubMed: 17563024] Over 15,000 adults were enrolled in this study, which found that elevated serum vitamin D levels were associated with a decreased risk of several highly prevalent diseases, including hypertension, diabetes mellitus, and obesity. Given the major public health burden associated with these diseases, the results of this study suggest that vitamin D supplementation may have an important role in preventative medicine.

26. Thieden E, Jorgensen HL, Jorgensen NR, et al. Sunbed radiation provokes cutaneous vitamin D synthesis in humans: a randomized controlled trial. Photobiol. 2008 epub ahead of print.

** 27. Robinson JK, Kim J, Rosenbaum S, Ortiz S. Indoor tanning knowledge, attitudes, and behavior among young adults from 1988-2007. Arch Dermatol 2008;144:484–8. [PubMed: 18427042] This study tracks trends in indoor tanning use, reasons for tanning, and knowledge of tanning risks over a 20-year period. The data demonstrate a massive increase in the rate of indoor tanning, along with a developing deficit of risk awareness.

28. Heckman CJ, Coups EJ, Manne SL. Prevalence and correlates of indoor tanning among US adults. J Am Acad Dermatol 2008;58:769–80. [PubMed: 18328594]

* 29. Oliver H, Ferguson J, Moseley H. Quantitative risk assessment of sunbeds: impact of new high power lamps. Br J Dermatol 2007;157:350–6. [PubMed: 17650177] This British study found an increase in the intensity of indoor tanning lamps over the past decade and revealed that a majority of tanning facilities surveyed used lamps that exceeded European UV radiation standards.

30. Nilsen LTN, Hannevik M, Aalerud TN, et al. Trends in UV irradiance of tanning devices in Norway: 1983-2005. Photochem Photobiol 2008;84:1100–8. [PubMed: 18399922]

31. Zeller S, Lazovich D, Forster J, Widome R. Do adolescent indoor tanners exhibit dependency? J Am Acad Dermatol 2006;54:589–96. [PubMed: 16546579]

* 32. Poorsattar SP, Hornung RL. UV light abuse and high-risk tanning behavior among undergraduate college students. J Am Acad Dermatol 2007;56:375–9. [PubMed: 17257709] In a study of college students, the authors found that more than 10% satisfied the criteria for a substance-related disorder with regard to UV tanning.

* 33. Heckman CJ, Egleston BL, Wilson DB, Ingersoll KS. A preliminary investigation of the predictors of tanning dependence. Am J Health Behav 2008;32:451–64. [PubMed: 18241130] In this report, more than a quarter of college students interviewed met criteria for tanning dependence; the magnitude of this finding suggests that tanning addiction is a significant concern in certain populations.

34. McLaughlin JA, Francis SO, Burkhardt DL, Dellavalle RP. Indoor UV tanning youth access laws: update 2007. Arch Dermatol 2007;143:529–32. [PubMed: 17438188]

35. Woodruff SI, Pichon LC, Hoerster KD, et al. Measuring the stringency of states' indoor tanning regulations: instrument development and outcomes. J Am Acad Dermatol 2007;56:774–80. [PubMed: 17276543]

36. Forster JL, Lazovich D, Hickle A, et al. Compliance with restrictions on sale of indoor tanning sessions to youth in Minnesota and Massachusetts. J Am Acad Dermatol 2006;55:962–7. [PubMed: 17097392]

** 37. Hirst N, Gordon L, Gies P, Green AC. Estimation of avoidable skin cancers and cost-savings to government associated with regulation of the solarium industry in Australia. Health Policy. 2008 epub ahead of print. The authors constructed a lifetime decision-analytic model based on a meta-analysis of health risks associated with indoor tanning, and they used this model to estimate the savings that would result from increased stringency of tanning access laws. Based on this model, improved tanning regulations would produce a significant reduction in government healthcare expenditure, suggesting that such regulations may ultimately be cost-effective.

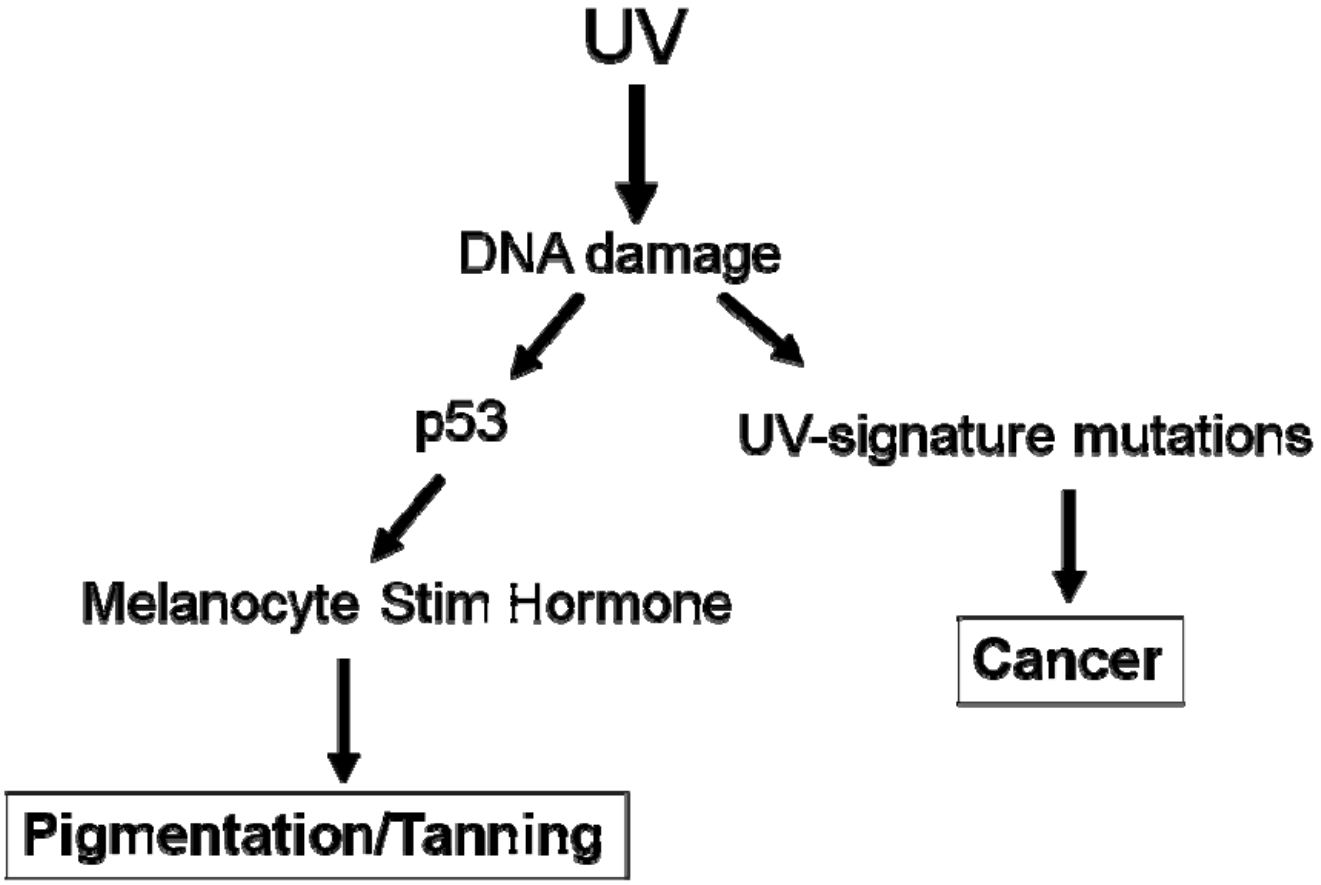

Figure 1.
Schematic of molecular steps in the tanning and UV-carcinogenesis pathways. DNA damage appears to be a common/proximal intermediate in both pathways.

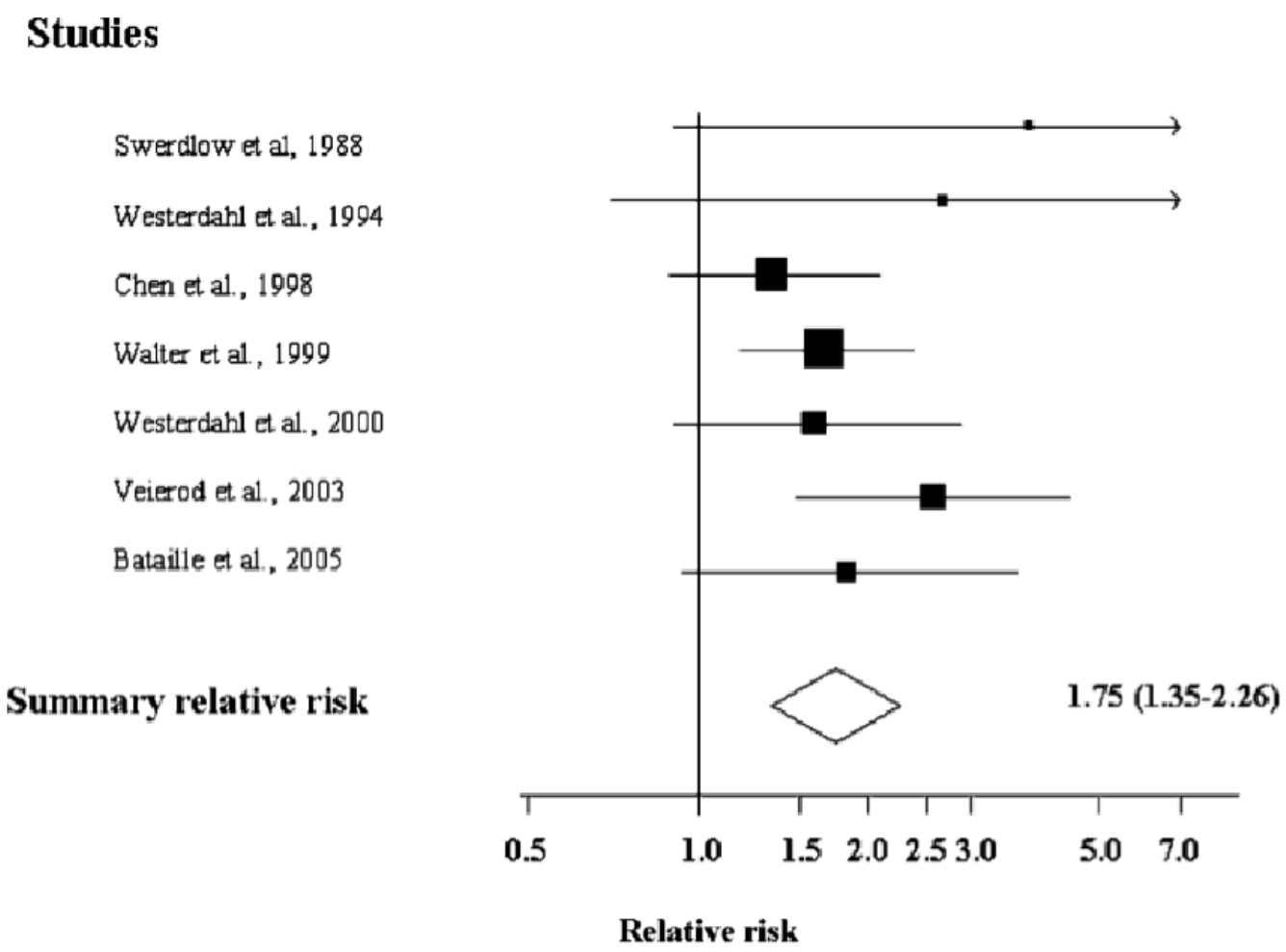

Figure 2.
Relative risk of melanoma associated with early first exposure to indoor tanning: estimates of 7 studies and summary estimate. Reprinted with permission [14**].