Clinics in Dermatology (2016) **34**, 640–648

Clinics in Dermatology

# Tanning beds: Impact on health, and recent regulations

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**Abstract** As the use of indoor tanning beds gained popularity in the decades after their appearance in the market in the early 1970s, concerns arose regarding their use. Clinical research has revealed an association between indoor tanning and several health risks, including the subsequent occurrence of melanoma and non-melanoma skin cancers, the development of psychologic dependence, and a tendency toward other high-risk health behaviors. In the face of mounting evidence, legislation has been passed, which includes the restriction of access to tanning beds by minors in 42 states and the District of Columbia, and the recent re-classification by the Food and Drug Administration, which now categorizes tanning beds as class II devices and worthy of restrictions and oversight. Early evidence suggests that these labors are resulting in cultural change, although continued efforts are necessary to limit further exposure and better inform the public of the dangers associated with indoor tanning use.


## Introduction

Since the introduction of ultraviolet (UV) tanning devices to the general public in the 1970s, the popularity of indoor tanning has grown immensely.[1] In an average day in the United States, it has been estimated that more than one million people will use an indoor tanning device.[2,3] This correlates with the approximately 35% of US adults, 59% of US university students, and 17% of US adolescents, who endorse prior exposure to indoor tanning.[4] The tanning industry's revenue was estimated to be $3 billion in 2014, and the density of tanning salons in 116 cities in 2009 exceeded the average number of Starbucks and McDonald's restaurants.[5,6] The prevalence of this practice has raised concern among the scientific community as UV radiation has long been implicated in the pathogenesis of cutaneous malignancy. We review the molecular, cellular, psychologic, and cultural implications of tanning as currently represented in the literature. Emphasis is placed upon recent legislation and the importance of continued restrictions and regulations.

## Molecular and cellular effects of tanning

Pigmentary change, secondary to UV radiation, occurs in two separate stages: (1) immediate and persistent pigment darkening, and (2) a delayed tanning response.[7] Immediate pigment darkening occurs within minutes of exposure to both UVA and visible light, resolves within 10–20 minutes, and is followed by the appearance of persistent pigment darkening, which is usually less intense and lasts for approximately 2 hours. Both immediate pigment darkening and persistent pigment darkening result from the oxidation of preformed melanin and the redistribution of melanosomes; thus, there is no new melanin synthesis at this stage. In contrast, delayed

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http://dx.doi.org/10.1016/j.clindermatol.2016.05.016
0738-081X/

tanning, which occurs 3–5 days after exposure, is associated with an increase in melanocyte tyrosinase activity resulting in synthesis of new melanin. Both UVA and UVB radiation can induce the delayed response; however, UVA-induced delayed tanning is 2–3 orders of magnitude less efficient (ie, needing higher doses) and requires oxygen at the time of irradiation.[7,8] There are compelling data to suggest that DNA damage secondary to UV radiation serves as a stimulus for pigmentation via upregulation of tyrosinase mRNA and protein levels.[7] Damage to keratinocyte DNA also activates p53, which binds and upregulates transcription of proopiomelanocortin and subsequently α-melanocyte–stimulating hormone. This factor then signals cutaneous melanocytes via the melanocortin 1 receptor to synthesize melanin resulting in cutaneous pigmentation.[9] As DNA damage serves as an intermediate for both tanning and skin carcinogenesis, a "safe tan" secondary to UV radiation is impossible to attain.[2,10]

Modern tanning beds are very effective at delivering UV radiation with an average erythema-effective irradiance of .33 W/m$^2$ (as determined by a study conducted in Switzerland of commercially available sunbeds in 2002). This corresponds to a UV index of 13, which is notably elevated in comparison to the UV index of noontime summer sun at intermediate latitudes of 8.5.[11] Although tanning beds are a polychromatic source of UV radiation, the emission is largely within the UVA range (320–400 nm) reaching values 10–15 times higher than that of mid-day sun.[11] The UVB range (290–320 nm) contains the major wavelengths responsible for inducing erythema, though most tanning lamps emit <10% within this range.[12,13]

Tanning proponents have argued that, by largely restricting exposure to the UVA range, artificial sources of UV light are a safer alternative to sun exposure.[12] This postulation, although accepted by many consumers, is simply false, as both UVA and UVB exposure produces DNA damage. The predominant mechanism for UVA-induced damage is via the generation of reactive oxygen species, and UVB, through a direct effect on DNA to induce the formation of cyclobutane pyrimidine dimers (CPDs) and other photoproducts. Although UVA exposure was once presumed to contribute to skin cancer pathogenesis only through oxidative stress, as the wavelengths are poorly absorbed by cellular DNA, this assumption has been challenged by recent evidence. By coupling high-performance liquid chromatography and mass spectrometry, investigators were able to determine the type and yield of cellular damage after exposure. They observed that CPDs—believed to be the signature mutation of UVB-induced DNA damage—were the predominant product in both human skin explants and cultured keratinocytes after exposure to UVA alone.[14,15] They have hypothesized that, instead of direct energy transfer, photosensitization was responsible for the observed changes. Another study, utilizing a mouse model and human melanocytes *in vitro*, also found CPD formation to be possible after discontinuation of UVA radiation with dimers detected up to 3 hours after the completion of exposure. This occurrence is due to the interaction of reactive oxygen and nitrogen species with melanin, which generates energy that results in "dark CPD" formation (ie, the production of CPDs in the absence of continued UV radiation).[16]

Although a smaller proportion of tanning bed emission (<1-9.5%) has been described within the UVB range, there is a high degree of variability between devices.[12,13] A study conducted at tanning facilities in North Carolina found an average erythemally weighted UVB output of .35 W/m$^2$. Compared with the equivalent solar noon UVB dose in Washington, DC, of .18 W/m$^2$, such exposure can also be clinically relevant.[12] *In vivo* tanning salon exposures, ranging from a single nonerythemogenic dose to 10 successive exposures for 2 weeks, have been shown to increase CPDs and alter p53 protein expression within epidermal keratinocytes.[17]

Even though DNA damage is crucial to understanding the health risks associated with tanning, this is simply one facet to the molecular changes that occur after exposure to UV radiation. Both UVA and UVB induce cutaneous immunosuppression, inhibit antigen presenting cells, and alter the expression of key cell signaling molecules and adhesion proteins.[18–20] A study exploring the effects of tanning beds specifically showed localized suppression of contact hypersensitivity and sensitization with an increase in circulating T suppressor cells after exposure.[21] Cumulative doses of UVA have also been associated with alterations in the normal cutaneous architecture, including epidermal hyperplasia, the presence of a persistent dermal infiltrate, and the deposition of lysozyme—found to be increased in actinically damaged skin—on elastic fibers.[19]

## Indoor tanning and the risk of cutaneous malignancy

### Nonmelanoma skin cancer

Given the strength of the molecular evidence implicating tanning bed use in carcinogenesis, it is of no surprise that several epidemiologic studies have revealed a positive link between artificial UV light sources and nonmelanoma skin cancer. As basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are collectively the most common form of human malignancy, this awareness should prompt an appropriate health policy response.[22] A case–control study was conducted in 2002 utilizing 863 subjects and 540 controls to characterize further the risk of nonmelanoma skin cancer and artificial tanning. This study found that prior use of a tanning device more than doubled the risk of developing an SCC (odds ratio [OR] = 2.5, 95% confidence interval [CI] = 1.7-3.8) and resulted in a 50% increased risk of developing a BCC (OR = 1.5, 95% CI = 1.1-2.1).[22] Adjustment for history of sunburns, sunbathing behavior, and sun exposure did not alter the results. The subject's age at the initial tanning experience was similarly significant, as the odds ratio for SCC and BCC were found to be increased by 20% and 10%, respectively, for each decade younger the subject was at that time.[22] These findings were corroborated by a 2007 systematic review conducted by the