The general public currently holds a positive attitude regarding tanning bed use and many people use tanning beds regularly for what they perceive to be cosmetic and psychological benefits despite their awareness of the association between excessive ultraviolet light exposure and its health consequences. Studies have shown that the majority of tanning bed users are female, young, and single. Yet, the direct risks of tanning bed use are well-documented in the literature with examples of exacerbations of lupus erythematosus, porphyria, mid-dermal elastolysis, actinic granulomata, verruca, and lentigines. Of greatest concern following tanning bed use is the development...
of cutaneous malignancies, both melanoma and non-melanoma.\textsuperscript{19,23}

While ultraviolet light under the supervision of a trained physician is used routinely in the management of various skin disorders in dermatology, the indiscriminate use of tanning beds is not generally recommended. Tanning salons differ vastly in bed types used, lamps used, frequency of changing lamps, number of hours on each lamp, spectra of ultraviolet light exposure, cleanliness, and the presence of knowledgeable and professional staff.\textsuperscript{24} Tanning bed lamps progressively lose their intensity and wavelength emission specificity if not regularly calibrated. Thus, these variables underscore the near impossibility of estimating the cumulative ultraviolet exposure a particular patient may receive, especially if he/she is using several different tanning salons over a course of several years.

DFSP arises from gene rearrangements within chromosomes 17 and 22 resulting in the fusion of the collagen type 1α1 gene (COL1A1) to the platelet-derived growth factor (PDGF) β-chain gene (PDGFB). As a result, there is a deregulation of PDGF β-chain expression leading to continuous activation of the PDGF receptor β protein tyrosine kinase, which promotes DFSP cell growth.\textsuperscript{5,10} Interestingly, Sasaki et al analyzed p53 expression in 19 patients with DFSP and suggested that p53 overexpression may be linked to a subgroup with more aggressive clinical behavior.\textsuperscript{8} Similarly, Diaz-Cascajo et al compared the expression of p53 in 12 cases of DFSP and 10 cases of dermatofibromas (DF) and found nuclear accumulation of the p53 protein in 11 cases of DFSP and in no cases of DF.\textsuperscript{25} These authors suggested that mutations of the p53 gene may be involved in the molecular pathogenesis of DFSP but not of DF.

The p53 protein is the product of a tumor suppressor gene that plays an important role in the control of cell proliferation by suppressing proliferation of cells with DNA damage. It is regarded as the “guardian of the genome” as it
protects DNA integrity in response to cytotoxic stress, including ultraviolet radiation, by promoting cellular apoptosis. The p53 gene is the most frequently altered gene in human cancer. The majority of p53 mutations are missense mutations leading to an altered amino acid sequence. In chronically sun exposed skin of Caucasians, proliferations of keratinocytes over expressing mutations of the p53 protein have been demonstrated. These abnormal keratinocytes with a mutated p53 gene have a lower probability of undergoing apoptosis and may very well be forerunners of skin cancer although a clear relationship to skin cancer has yet to be proven.

We report a case of DFSP in a young woman with an excessive history of tanning bed use. While there is clearly no current link between ultraviolet light exposure and increased risk for DFSP, we feel that this case is illustrative especially in light of recent advances in molecular genetics as described above possibly linking DFSP to mutations in p53 and mutations in p53 to skin cancer. While these findings are circumstantial at best, further studies are necessary to examine the risks of ultraviolet light exposure and DFSP.

References


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