Tanning beds could provide a greater risk than originally thought: new study

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(Medical Xpress) -- A new study published in the *Journal of Investigative Dermatology* has found that, despite previous information, the UVA radiation used in tanning beds may cause more damage to the skin that was originally thought.

Previously it was believed that UVA, which penetrates the deeper layers of the skin, did not play a large role in the aging and wrinkling process, or in skin cancer, as it did not affect the out layer of skin, or the epidermis.

Co-author Antony R. Young from St. John's Institute of Dermatology and the team of researchers exposed the buttocks of 12 volunteers to both UVA1 and UVB rays. The UVA1 rays penetrated and caused damage to the basal layer of the skin and induced lesions known as thymine dimers. UVB also caused these lesions in a greater number but they were on the surface of the skin and not as deep as the UVA1 damage. The doses of UV exposure used in the study were comparable to a sunburn.

The release of this study comes at the same time that the FDA is considering a ban on tanning beds for children and teens under the age of 18. Governor Jerry Brown from California signed a ban on October 9, 2011 which prohibits the use of tanning beds by minors under the age of 18 in California.

Of course this new information is not making the tanning bed industry

happy. John Overstreet, the executive director of the Indoor Tanning Bed Association, points out that tanning beds emit the same ratio of UV waves that the sun does and that the risk of outdoor sun exposure and tanning bed use is the same. He points out that there is no scientific evidence that shows a non-burning exposure to the sun or tanning beds is linked to the cause of cancer.

This new study does also point out the damaging effects of the sun and the deeper penetrating effects of the UVA rays. It points to the need for labeling changes in broad-spectrum sun products to show that they protect against both UVA and UVB rays.

More information: UVA1 Induces Cyclobutane Pyrimidine Dimers but Not 6-4 Photoproducts in Human Skin In Vivo, *Journal of Investigative Dermatology*, (6 October 2011) doi:10.1038/jid.2011.283

Abstract
UVB readily induces cyclobutane pyrimidine dimers, mainly thymine dimers (TTs), and pyrimidine (6-4) pyrimidone photoproducts (6-4PPs) in DNA. These lesions result in “UVB signature mutations” found in skin cancers. We have investigated the induction of TTs and 6-4PPs in human skin in vivo by broadband UVA1, and have compared this with comparable erythemal doses of monochromatic UVB (300 nm). In vitro and ex vivo studies have shown the production of TTs, without 6-4PPs, by UVA1. We show that UVA1 induces TTs, without 6-4PPs, in the epidermis of healthy volunteers in vivo, whereas UVB induced both photoproducts. UVB induced more TTs than UVA1 for the same level of erythema. The level of UVA1-induced TTs increased with epidermal depth in contrast to a decrease that was seen with UVB. UVA1- and UVB-induced TTs were repaired in epidermal cells at a similar rate. The mechanism by which UVA1 induces TTs is unknown, but a lack of intra-individual correlation between our subjects’ UVB and UVA1 minimal erythema doses implies that UVA1 and UVB erythema occur by

different mechanisms. Our data suggest that UVA1 may be more carcinogenic than has previously been thought.