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Sun Damage Occurs Even After Sunset

Skin cancer normally occurs when ultraviolet (UV) radiation from the sun or from tanning beds damages DNA in skin cells, triggering mutations that lead to such cancers as melanoma. The mechanism by which this occurs was understood to take place during exposure to UV light. Researchers at Yale have recently discovered that this damage continues even when we're out of the sun and that melanin, the pigment that gives skin its color, may be both protective and harmful.

Dermatologists have long known that blondes and redheads are more sensitive to sunburn and skin cancer. Melanin helps block UV, so this was thought

Sun damage as seen with ultraviolet photography.

to be attributable to their light skin. Yet there are light-skinned, dark-haired people in countries near the equator who don't have a high incidence of skin cancer. Furthermore, scientists at Yale and elsewhere found that when skin cells or mice were irradiated with UV, there was more cell death in cells that contained melanin.

When UV light hits the skin, it causes a type of DNA damage known as a cyclobutane pyrimidine dimer (CPD), in which two adjacent bases attach, causing a bend that makes it difficult for the cell

to copy its DNA correctly. During UV exposure, many CPDs are instantly created in skin cells, but the DNA is able to repair itself by removing most of the CPDs and replacing them with normal DNA. In a study published in the journal *Science*, Douglas E. Brash, PhD, Clinical Professor of Therapeutic Radiology and Dermatology, and a member of Yale Cancer Center's Radiobiology and Radiotherapy Research Program, and his colleagues found that melanocytes, the cells that form melanin, continued to generate CPDs for several hours after

UV exposure ended. Interestingly, cells without melanin generated CPDs only during UV exposure.

"We've been underestimating the amount of UV damage that the sun or sun beds are causing because we measure it immediately after exposure," said Dr. Brash. In fact, over half of a person's DNA damage arises in the car on the way home from the beach. Furthermore, while melanin does act as a shield, it is also associated with skin cell damage.

what other diseases might involve similar chemistry.

Even though we now know that melanin has a dark side, it is nonetheless evolution's best solution to absorbing harmful UV radiation. "The imperfect melanin strategy does serve to spread CPDs out over time, which may be better than occurring all at once during sun exposure and possibly overwhelming the DNA repair system," said Dr. Brash. It also creates an opportunity. The delayed

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When researchers delved into the details of this process, the results were surprising. Sanjay Premi, PhD, associate research scientist in the Brash laboratory, discovered that UV light activated two enzymes that combined to excite an electron in a fragment of melanin. The energy generated from this process was transferred to DNA in the dark, creating the same DNA damage that occurs in the presence of sunlight. Chemically induced electron excitation – known as chemiexcitation – was previously seen only in plants and lower organisms such as jellyfish and fireflies, where it generates softly glowing light. In humans, however, it is less benign, raising the question of

pathway should be interceptable at several points, creating an opening for an "evening-after" sunscreen that might prevent the enzyme activation or divert the energy from the excited electron into heat before it can damage DNA.

"This finding doesn't really change the habits of UV exposure," said Dr. Brash. "I tell people to enjoy the sun but don't lie on the beach between 10 and 2, and wear a hat." Sunscreens are useful, as long as they block both UVB and UVA, the two types of harmful radiation in sunlight. But once a new generation of sunscreens is developed, a new habit would need to be added: Putting on a different sunscreen when you go inside. ☺