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Insight into Photocarcinogenesis

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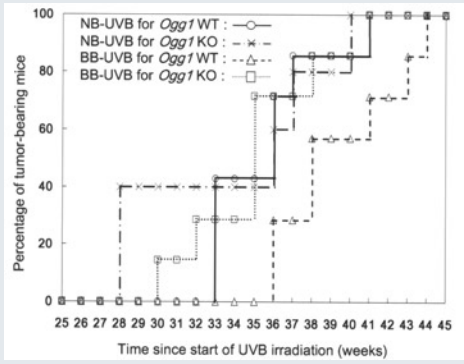

Advances in phototherapy have led to significant improvement in the care of patients with a variety of skin disorders, ranging from psoriasis to vitiligo. The use of narrow-band ultraviolet radiation B (NB-UVB), with peak fluence at 311 nm, has been one such advance. Superior in efficacy to broad-band UVB (BB-UVB), it combines added efficacy similar to that of photochemotherapy (PUVA), with less inconvenience (van Weelden *et al*., 1988; Coven *et al*., 1997; Gathers *et al*., 2002). However, experimental data suggest that the use of NB-UVB is not without cost. Several investigative studies have determined that NB-UVB possesses greater potential for skin carcinogenesis (Flindt-Hansen *et al*., 1991; Wulf *et al*., 1994; Gibbs *et al*., 1995) than BB-UVB, already a known carcinogen (Kraemer, 1997).

In a follow-up to recent work linking BB-UVB with oxidative DNA damage and tumor formation (Kunisada *et al*., 2005), Kunisada *et al*. pursue the question of photocarcinogenesis by NB-UVB in greater detail in this issue (Kunisada *et al*., 2007). The investigators employed comparable (in terms of MED) doses of NB-UVB and BB-UVB in three susceptible mouse strains, examining tumor formation and three types of DNA damage. In two of the three strains of mice, they found a higher number of malignant tumors following exposure to NB-UVB. This was associated with increased cyclobutane pyrimidine dimer formation and decreased formation of 6-4 photoproducts and 8-oxoguanine. Only in *Ogg1* knockout mice, which lack the gene that codes for 8-oxoG–DNA glycosylase, a repair enzyme, did BB-UVB exposure lead to similar numbers of malignant tumors. These results suggest that photocarcinogenesis is greater with NB-UVB and that it is related to cyclobutane pyrimidine dimer formation. Through the following questions we will delve into this article in greater detail. For brief answers, refer to http://network.nature.com/group/jidclub.

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QUESTIONS

1. What are the major findings of the study?
2. What is the benefit of using different strains of mice in this study?
3. Why would NB-UVB be more carcinogenic than BB-UVB, considering that BB-UVB encompasses a wider spectrum of wavelengths?
4. Was the choice of dose (energy) important in the outcome of the study?
5. Because sarcomas may develop in mice after UV exposure, does UV play a role in sarcoma development in humans?
6. What are the clinical implications of this article?

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