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Promising approach to a more effective sunscreen

Chronic exposure to the sun increases the risk of an individual developing skin cancer because UV light from the sun can cause genetic mutations that enable cells in the skin to grow in an uncontrolled manner. Hope for a new sunscreen that can prevent and treat UV light induced skin cancers has been provided by a new study in a mouse model of the disease by Mohammad Athar and colleagues at Columbia University College of Physicians and Surgeons, New York.

Mutations in the p53 gene that result in the production of mutant forms of p53 protein unable to do their job are associated with cancer in various tissues in mice and humans. An agent known as CP-31398 has previously been shown to bind mutant forms of p53 and restore their function. In the study, applying CP-31398 to the skin of mice reduced skin cancer (in terms of the size and number of tumors) induced by exposure to UVB light. In addition, treating mice already carrying UVB light induced skin cancers with CP-31398 reduced tumor growth and volume. These effects were associated with increased p53 function. The clinical relevance of these observations was highlighted by the demonstration that CP-31398 increased p53 function in a human skin cancer cell line expressing a mutant form of p53. However, as Wafik S. El-Deiry from the University of Pennsylvania School of Medicine, Philadelphia, notes in his accompanying commentary, this study raises a number of questions that need to be addressed before these promising findings lead to new sunscreens for the prevention and treatment of UV light induced skin cancer.

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TITLE: CP-31398 restores mutant p53 tumor suppressor function and inhibits UVB-induced skin carcinogenesis in mice

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ACCOMPANYING COMMENTARY

TITLE: Targeting mutant p53 shows promise for sunscreens and skin cancer

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