Cancer Prevention Research

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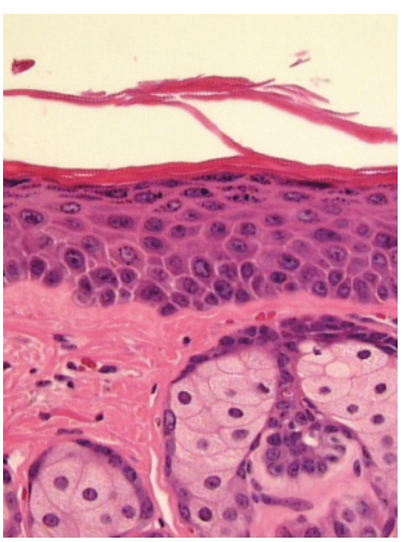
ABOUT THE COVER

An urgent need exists for the development of more efficacious molecular strategies targeting non-melanoma skin cancer (NMSC), the most common malignancy worldwide. Inflammatory signaling downstream of Toll-like receptor 4 (TLR4) has been implicated in several forms of tumorigenesis, yet its role in solar UV-induced skin carcinogenesis remains undefined. Recently, we have shown TLR4 contributes to UV-induced stress and inflammatory signaling in keratinocytes, and that the specific TLR4 antagonist resatorvid (TAK-242) blocks acute UV-induced AP-1 and NF-κB activity. In the current study, topical resatorvid has been employed for skin photochemoprevention, suppressing tumor area and multiplicity in SKH-1 mice exposed to solar simulated light (SSL). In the cover figure, we highlight the inhibition of UV-induced epidermal immune cell infiltration in chronically exposed mouse epidermis (background), and the potentiation of apoptosis mediated by resatorvid treatment in skin tumors (inset). SKH-1 mice were exposed to SSL three times weekly for 15 weeks, during which time they were topically treated with either vehicle (acetone) or resatorvid 1 hour prior to SSL. Some mice were then sacrificed and chronically exposed skin was examined for epidermal immune infiltration using H&E stained tissue (400x). Skin treated with SSL + vehicle displayed significant infiltration of lymphocytic cells (not shown), while skin treated with SSL + resatorvid lack infiltrates, as displayed in the background image. See the article by Blohm-Mangone et al. beginning on page 265 for more information.



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