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Cancer Prevention Research

January, 2010 • Volume 3 • Number 1

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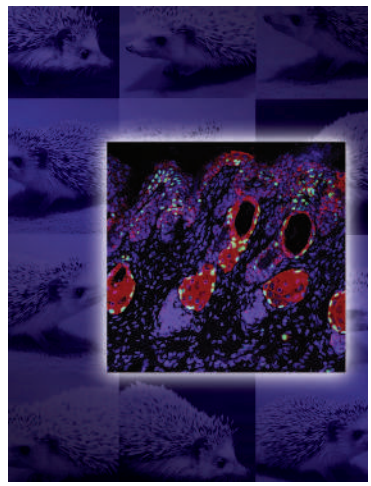
Note: Supplement to *Cancer Prevention Research*, Volume 3, Issue 1 (January 2010)
The abstracts of the 2009 AACR International Conference on Frontiers in Cancer Prevention Research (December 6–9, 2009) are being published as an online-only supplement to the January issue of *Cancer Prevention Research*. The supplement is available at http://cancerpreventionresearch.aacrjournals.org/.

ABOUT THE COVER



The cover shows a tissue section from a murine model of skin basal cell carcinoma (BCC) related to aberrant hedgehog signaling (20X magnification; courtesy of Rehan Villani and Brandon Wainwright). The engineered mouse has a keratin 14 (K14) promoter-driven Cre recombinase transgene (*K14-Cre*) and a conditionally targeted allele of the Patched1 (*Ptch1*) gene (*K14-Cre:Ptch1*$^{lox/lox}$). The *K14-Cre:Ptch1*$^{lox/lox}$ section shows immunofluorescence staining for proliferating cell nuclear antigen (PCNA; *green*), indicating proliferating cells of BCC, which are largely absent from stained sections of control littermates (*Ptch1*$^{lox/lox}$; not shown). Staining also highlights K14 (*red*) in the basal cell population and DAPI counterstained nuclei (in all cell populations; *blue*). Epidermis-specific mutation of *Ptch1* in the mice leads quickly to lesions closely resembling human BCC. Hedgehog signaling is constitutively activated (commonly via inactivated *PTCH1*) in human BCCs. Germline heterozygous mutations of *PTCH1* highly predispose Gorlin syndrome patients to BCC with loss or inactivation of the remaining wild-type allele. See articles by Tang et al. (beginning on page 25), Rudin (beginning on page 1), and Arbiser (beginning on page 4) for more information.

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Cancer Prev Res 2010;3:1-124.