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David L. McCormick, Jonathan M. Phillips, Thomas L. Horn, William D. Johnson, Vernon E. Steele, and Ronald A. Lubet
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 (Ptc1) gene (K14-Cre:Ptc1flx/lox). The K14-Cre:Ptc1flx/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 [Ptc1flx/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 Ptc1 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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