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ABOUT THE COVER

Because of its relative latency in disease onset and progression, prostate cancer should be an ideal target for chemoprevention strategies. However, chemoprevention trials for prostate cancer (PCa) by androgen receptor or androgen synthesis inhibition have proven ineffective. It is known that the epigenome becomes corrupted early during PCa initiation, making the epigenome a prime candidate target for PCa prevention. Employing pre-clinical human and mouse models of PCa in a study beginning on page 979, Burkhart and colleagues demonstrate that inhibition of the enhancer of zeste-2 (Ezh2) catalytic activity augmented progression of PCa by induction of tumor cell senescence, raising the overall question regarding Ezh2 inhibition as a potential chemo (epi) prevention strategy for the prevention of prostate cancer. The micrograph on the cover depicts induction of β-galactosidase staining (senescence) following genetic inhibition of Ezh2 catalytic activity in a mouse prostate gland that over-expresses cMYC.
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