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Molecular Cancer Research
Defining the Molecular Basis of Malignancy and Progression
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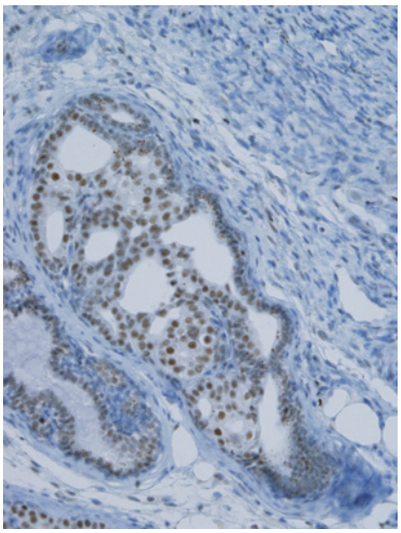
ABOUT THE COVER



In prostate epithelial cells, MED1 serves as a key transcriptional coactivator for the androgen receptor and other signal-activated transcription factors. MED1 can be phosphorylated by ERK and AKT, kinases that are commonly hyperactivated in prostate cancers as a function of tumor progression. MED1 phosphorylation significantly stabilizes its nuclear half-life and stimulates its transcriptional coactivator activity. Jin and colleagues used immunohistochemistry to characterize MED1 levels in an *Nkx3.1:Pten*-mutant mouse model of prostate cancer that recapitulates the human disease. Importantly, *Nkx3.1:Pten*-mutant prostate cancers are genetically programmed to hyperactivate ERK and AKT signaling in parallel with cancer progression. The cover shows MED1 overexpression in a *Nkx3.1:Pten*-mutant prostate adenocarcinoma that was resistant to castration. For additional results and details, please see the article by Jin and colleagues on page 736 of this issue.

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