

MOLECULAR CANCER RESEARCH

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SIGNAL TRANSDUCTION AND FUNCTIONAL IMAGING

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TUMOR MICROENVIRONMENT AND IMMUNOBIOLOGY

- 1571** **Cytidine Deaminase APOBEC3A Regulates PD-L1 Expression in Cancer Cells in a JNK/c-JUN-Dependent Manner**
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- 1583** **Exosomal CD47 Plays an Essential Role in Immune Evasion in Ovarian Cancer**
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- 1596** **Oncogenic *Kras*^{G12D} Activation in the Nonhematopoietic Bone Marrow Microenvironment Causes Myelodysplastic Syndrome in Mice**
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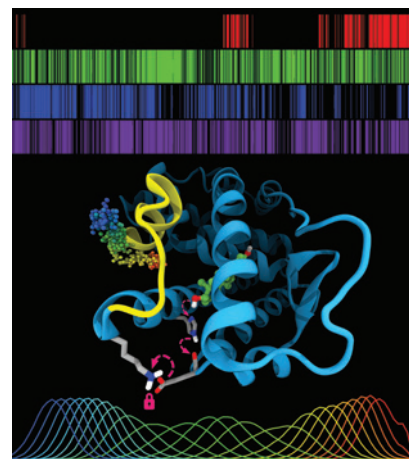
CORRECTION

- 1609** **Correction: Dephosphorylation of the Proneural Transcription Factor ASCL1 Re-Engages a Latent Post-Mitotic Differentiation Program in Neuroblastoma**

ABOUT THE COVER

Estrogen receptor alpha (ER α) is a ligand-activated transcription factor that is therapeutically targetable with endocrine therapies in approximately 70% of breast cancers. Ligand binding initiates an extended hydrogen bonding network (pink arrows) terminating in an ionic lock that allows helix 12 (H12; yellow) to fold into the active conformation capable of binding coactivating proteins that initiate transcriptional cascades. In their study on page 1559, Mayne and colleagues perform free energy calculations of clinically relevant mutations in the helix 11-12 loop of ER α to determine their effect on H12 conformation and receptor activation. The colored balls and associated histograms characterize the dynamics of the loop preceding H12. Each of the point mutations changes the energy landscape of ER α in a manner that no longer requires ligand binding to form the ionic lock and adopt the active conformation, thereby driving therapy resistance and metastatic disease.

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