# In This Issue

Highlighted research articles

# Research Articles

**MAX Inactivation in Small Cell Lung Cancer Disrupts MYC-SWI/SNF Programs and Is Synthetic Lethal with BRG1**


**Précis:** MAX is a tumor suppressor recurrently altered in SCLC that is regulated by the SWI/SNF protein BRG1, and BRG1 depletion is selectively toxic in MAX-deficient cells.

See commentary, p. 273

**An In Vivo Functional Screen Identifies ST6GalNAc2 Sialyltransferase as a Breast Cancer Metastasis Suppressor**


**Précis:** Modification of O-glycans by ST6GalNAc2 suppresses galectin-3 binding and metastatic seeding of breast cancer cells in the lung and determines responsiveness to galectin-3 inhibition.

See commentary, p. 275

**RapidCaP, a Novel GEM Model for Metastatic Prostate Cancer Analysis and Therapy, Reveals Myc as a Driver of Pten-Mutant Metastasis**


**Précis:** RapidCaP is a GEM model of prostate cancer that exhibits features typical of human disease and identifies MYC as a spontaneous metastatic driver.

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**In the Spotlight**

**MYC, MAX, and Small Cell Lung Cancer**

C.M. Rudin and J.T. Poirier

See article, p. 292

**Sticking to Sugars at the Metastatic Site: Sialyltransferase ST6GalNAc2 Acts as a Breast Cancer Metastasis Suppressor**

C.M. Ferrer and M.J. Reginato

See article, p. 304

**Forecast: Rough Seas for Leukemia**

D.M. Hockenbery

See article, p. 362

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**Review**

Improving the Efficacy of Chemoradiation with Targeted Agents

M.A. Morgan, L.A. Parsels, J. Maybaum, and T.S. Lawrence

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**Online**

For more News and Research Watch, visit Cancer Discovery online at http://CDnews.aacrjournals.org.
Romero and colleagues identified recurrent inactivating deletions within MYC-associated factor X (MAX) in small cell lung cancer (SCLC) cell lines and tumors that were mutually exclusive with MYC amplification, inactivation of MAX dimerization protein (MGA), and mutations in BRG1 (also known as SMARCA4), which encodes an ATPase subunit of the SWI/SNF chromatin remodeling complex, suggesting that these genetic alterations drive SCLC through disruption of a common pathway. Indeed, BRG1 directly regulated MAX expression, and MAX upregulated MYC target genes in a BRG1-dependent manner. Moreover, BRG1 depletion selectively inhibited the growth of MAX-deficient SCLC cells, suggesting that a synthetic lethal relationship exists between these two proteins and raising the possibility that BRG1 may be a therapeutic target in SCLC. For details, please see the article by Romero and colleagues on page 292.