In This Issue

Highlighted research articles ........................................... 259

News in Brief

Important news stories affecting the community .................. 262

News in Depth

Q&A: Howard Koh on Smoking Cessation and Policy ........... 265

Trial Offers New Model for Drug Development .................. 266

Research Watch

Selected highlights of recent articles of exceptional significance from the cancer literature .......................... 268

Online

For more News and Research Watch, visit Cancer Discovery online at http://CDnews.aacrjournals.org.

Views

In The Spotlight

MYC, MAX, and Small Cell Lung Cancer ....................... 273
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 .... 275
C.M. Ferrer and M.J. Reginato
See article, p. 304

Forecast: Rough Seas for Leukemia ................................. 278
D.M. Hockenberg
See article, p. 362

Review

Improving the Efficacy of Chemoradiation with Targeted Agents .................. 280
M.A. Morgan, L.A. Parsels, J. Maybaum, and T.S. Lawrence

Research Articles

MAX Inactivation in Small Cell Lung Cancer Disrupts MYC–SWI/SNF Programs and Is Synthetic Lethal with BRG1 .............. 292
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 ............... 304
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 ............. 318
Précis: RapidCap is a GEM model of prostate cancer that exhibits features typical of human disease and identifies MYC as a spontaneous metastatic driver.
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.

Rapid Induction of Apoptosis by PI3K Inhibitors Is Dependent upon Their Transient Inhibition of RAS–ERK Signaling .......................... 334

Précis: Unlike AKT inhibitors, PI3K inhibitors rapidly induce cancer cell death because they also inhibit activation of wild-type RAS and downstream RAF–MEK–ERK signaling.

Evolution of DNA Methylation Is Linked to Genetic Aberrations in Chronic Lymphocytic Leukemia .... 348

Précis: Intratumor methylation heterogeneity coevolves with genetic alterations and is associated with high-risk, poor prognostic markers in a subset of CLL tumors.

Selective BCL-2 Inhibition by ABT-199 Causes On-Target Cell Death in Acute Myeloid Leukemia ....................... 362

Précis: ABT-199 shows on-target activity against AML cells that correlates with BCL-2 expression and can be predicted by mitochondrial BH3 profiling.

See commentary, p. 278
CANCER DISCOVERY

4 (3)

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