IN THIS ISSUE
Highlighted research articles 377

NEWS IN BRIEF
Important news stories affecting the community 380

NEWS IN DEPTH
Q&A: Michael Pellini on Cancer Diagnostics 382
The States of Research 383
Cancer Stem Cells in the Crosshairs 384

RESEARCH WATCH
Selected highlights of recent articles of exceptional significance from the cancer literature 385

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

IN THE SPOTLIGHT
Making Sense of MEK1 Mutations in Intrinsic and Acquired BRAF Inhibitor Resistance 390
K.H.T. Paraíso and K.S.M. Smalley
Commentary on Shi et al., p. 414

Beta-Testing of PI3-Kinase Inhibitors: Is Beta Better? 393
P.R. Shepherd and W.A. Denny
Commentary on Ni et al., p. 425

CIRCULATING ENDOTHELIAL PROGENITORS AND TUMOR RESISTANCE TO VASCULAR-TARGETING THERAPIES 395
M. De Palma and S. Nucera
Commentary on Taylor et al., p. 434

RESEARCH BRIEFS

PREEXISTING MEK1 EXON 3 MUTATIONS IN V600E/BRAF MELANOMAS DO NOT CONFERR RESISTANCE TO BRAF INHIBITORS 414
Précis: Mutation of the downstream BRAF effector MEK1 is not a mechanism of innate resistance to targeted BRAF inhibitors.

FUNCTIONAL CHARACTERIZATION OF AN ISOFORM-SELECTIVE INHIBITOR OF PI3K-P110β AS A POTENTIAL ANTICANCER AGENT 425
Précis: A selective small-molecule inhibitor of the p110β isoform of PI3K is effective in a subset of PTEN-deficient tumor cell lines and xenografts.

OCUPY EGFR 398
J.H. Park and M.A. Lemmon
Commentary on Barkovich et al., p. 450 and Vivanco et al., p. 458

IN FOCUS

THE CBIO CANCER GENOMICS PORTAL: AN OPEN PLATFORM FOR EXPLORING MULTIDIMENSIONAL CANCER GENOMICS DATA 401

REVIEW
EMERGING EPIGENETIC TARGETS AND THERAPIES IN CANCER MEDICINE 405
R. Popovic and J.D. Licht

PUBLICATIONS

Cancer Stem Cells in the States of Research
M. De Palma and S. Nucera
Targeting Therapies for Resistance to Vascular-Progenitors and Tumor Circulating Endothelial Cells
P.R. Shepherd and W.A. Denny
Inhibitors: Is Beta Better?
Beta-Testing of PI3-Kinase
Commentary on Shi et al., p. 414
MEK1
For the of exceptional significance... •Reprints
Important articles... •Articles
Highlighted •Contents
MAY 2012 • VOLUME 2 • NUMBER 5
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- Annotated Cell-Line Resources Speed Discovery
- Phase II Trial for Lymphoma Gives Promising Early Results
- Targeted Combo Effective for Refractory Ewing Sarcoma
- Novel PI3K Inhibitors Enter Human Studies
- An EMPaCT on Minority Recruitment
- MEK Inhibition Aids in Serous Ovarian Cancer

ON THE COVER

Vivanco and colleagues demonstrated that glioma-specific EGFR extracellular domain mutants were more sensitive to type II EGFR inhibitors (e.g., lapatinib) that stabilize an inactive kinase conformation than type I EGFR inhibitors (e.g., erlotinib) that target the active kinase conformation more commonly found in EGFR-mutant lung cancers. In a related article, Barkovich and colleagues found that the rapid release of erlotinib by glioma-specific EGFR mutants rendered them less sensitive to erlotinib than lung cancer–derived EGFR mutants. Together, these studies provide explanations for the limited success of first-generation EGFR inhibitors in treatment of EGFR-mutant gliomas and suggest alternative EGFR inhibition strategies may work best in these tumors. For details, please see the article by Vivanco and colleagues on page 458 and the article by Barkovich and colleagues on page 450.