IN THIS ISSUE
Highlighted research articles..................193

NEWS IN BRIEF
Important news stories affecting the community..................196

NEWS IN DEPTH
Q&A: Alan Auerbach on Small and Speedy Biotechs ............198

Venture Capital Arms Flex Their Muscle ..................199

Breast Cancer Screening Goes Personalized ..................200

RESEARCH WATCH
Selected highlights of recent articles of exceptional significance from the cancer literature..................201

ONLINE
For more News and Research Watch, visit Cancer Discovery online at www.AACR.org/CDnews.

VIEWS
In The Spotlight

USP2a Activation of MYC in Prostate Cancer ..................206
W.G. Nelson, A.M. De Marzo, and S. Yegnasubramanian
Commentary on Benassi et al., p. 236

Drug Interactions: The Importance of Looking Inside Cancer Cells ..................208
J.W. Clark
Commentary on Frese et al., p. 260

RESEARCH ARTICLES

Anti-VEGF Therapy Revived by c-Met Inhibition, But Is c-Met the Answer? ..................211
K.D. Lynn and R.A. Breken
Commentary on Sennino et al., p. 270

REVIEW
Circumventing Cancer Drug Resistance in the Era of Personalized Medicine ..................214
L.A. Garraway and P.A. Jänne

RESEARCH BRIEF
EGFR-Mediated Reactivation of MAPK Signaling Contributes to Insensitivity of BRAF-Mutant Colorectal Cancers to RAF Inhibition with Vemurafenib........227
Précis: Combined inhibition of RAF and EGFR may be necessary to effectively suppress MAPK signaling in BRAF-mutant colorectal cancers.

MYC Is Activated by USP2a-Mediated Modulation of MicroRNAs in Prostate Cancer ..................236
Précis: Overexpression of USP2a activates MYC and promotes prostate cancer growth and invasiveness via downregulation of miR-34b/c.
Akt/PKB-Mediated Phosphorylation of Twist1 Promotes Tumor Metastasis via Mediating Cross-Talk between PI3K/Akt and TGF-β Signaling Axes 248
Précis: Phosphorylation of TWIST1 by AKT promotes EMT and metastasis via TGF-β2 transcriptional regulation and PI3K/AKT feedback activation.

nab-Paclitaxel Potentiates Gemcitabine Activity by Reducing Cytidine Deaminase Levels in a Mouse Model of Pancreatic Cancer 260
Précis: Combined nab-paclitaxel and gemcitabine therapy leads to synergistic antitumor effects due to decreased gemcitabine metabolism.

Suppression of Tumor Invasion and Metastasis by Concurrent Inhibition of c-Met and VEGF Signaling in Pancreatic Neuroendocrine Tumors 270
Précis: Combined inhibition of VEGF and c-MET reduces the tumor invasiveness and metastasis observed after inhibition of VEGF alone and decreases tumor growth and angiogenesis.

For more News and Research Watch, visit Cancer Discovery online at www.AACR.org/CDnews. Online-only News stories include the following:

• Tracking Down Tumor-Targeting Bacteria
• Antiangiogenic Drugs Increase Xenograft Aggressiveness
• Can Chemotherapy Cause Cancer Relapse?
• Mutations, Tissue Type Both Influence Cancer Metabolism

On the Cover
Frese and colleagues utilized a genetically engineered mouse model of pancreatic ductal adenocarcinoma (PDA) to better understand the mechanistic basis for the clinical observation that nab-paclitaxel, a water-soluble, albumin-bound form of paclitaxel, elicits synergistic antitumor activity when combined with gemcitabine, a nucleoside analogue that is the current standard of care for PDA. Combination treatment with nab-paclitaxel increases intratumoral gemcitabine levels by creating an oxidative environment within the tumor that promotes degradation of cytidine deaminase, the primary gemcitabine metabolizing enzyme. For details, please see the article by Frese and colleagues on page 260.
## CANCER DISCOVERY

### 2 (3)

*Cancer Discovery* 2012;2:OF5-287.

<table>
<thead>
<tr>
<th>Updated version</th>
<th>Access the most recent version of this article at: <a href="http://cancerdiscovery.aacrjournals.org/content/2/3">http://cancerdiscovery.aacrjournals.org/content/2/3</a></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>E-mail alerts</th>
<th>Sign up to receive free email-alerts related to this article or journal.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reprints and Subscriptions</td>
<td>To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at <a href="mailto:pubs@aacr.org">pubs@aacr.org</a>.</td>
</tr>
<tr>
<td>Permissions</td>
<td>To request permission to re-use all or part of this article, contact the AACR Publications Department at <a href="mailto:permissions@aacr.org">permissions@aacr.org</a>.</td>
</tr>
</tbody>
</table>