Clinical Response to a Lapatinib-Based Therapy for a Li-Fraumeni Syndrome Patient with a Novel HER2V659E Mutation ............... 1238
Précis: Tumors of a patient with a germline TP53 mutation were found to harbor alterations in either EGFR or HER2 and were responsive to targeted therapy with lapatinib.

Androgen Receptor Signaling Regulates DNA Repair in Prostate Cancers ............... 1245
Précis: Antiandrogen therapy suppresses androgen receptor–mediated induction of DNA repair genes, resulting in increased DNA damage and enhanced radiosensitivity of prostate cancer cells.
See commentary, p. 1222

A Hormone–DNA Repair Circuit Governs the Response to Genotoxic Insult ............... 1254
J.F. Goodwin, M.J. Schiewer, J.L. Dean, R.S. Schrecengost, R. de Leeuw, S. Han, T. Ma, R.B. Den, A.P. Dicker, I.Y. Feng, and K.E. Knudsen
Précis: Androgen receptor activation in response to DNA damage promotes double-strand break repair via DNAPKcs and confers resistance to genotoxic insult in advanced prostate cancer.
See commentary, p. 1222
Strohecker and colleagues found that deletion of the essential autophagy gene \( \text{Atg7} \) initially induced oxidative stress and accelerated the formation of \( \text{Braf}^{\text{V600E}} \)-driven lung tumors but eventually slowed tumor growth and prolonged survival. \( \text{Atg7} \) deficiency led to an accumulation of morphologically and functionally defective mitochondria in \( \text{Braf}^{\text{V600E}} \)-driven lung tumors and rendered tumor cells dependent on exogenously supplied glutamine for survival. \( \text{Braf}^{\text{V600E}} \)-driven tumors may therefore become addicted to autophagy to sustain cell survival and proper mitochondrial function through the clearance of damaged organelles and recycling of metabolites for biosynthesis, and may thus be sensitive to autophagy inhibitors. For details, please see the article by Strohecker and colleagues on page 1272.

**ON THE COVER**

Strohecker and colleagues found that deletion of the essential autophagy gene \( \text{Atg7} \) initially induced oxidative stress and accelerated the formation of \( \text{Braf}^{\text{V600E}} \)-driven lung tumors but eventually slowed tumor growth and prolonged survival. \( \text{Atg7} \) deficiency led to an accumulation of morphologically and functionally defective mitochondria in \( \text{Braf}^{\text{V600E}} \)-driven lung tumors and rendered tumor cells dependent on exogenously supplied glutamine for survival. \( \text{Braf}^{\text{V600E}} \)-driven tumors may therefore become addicted to autophagy to sustain cell survival and proper mitochondrial function through the clearance of damaged organelles and recycling of metabolites for biosynthesis, and may thus be sensitive to autophagy inhibitors. For details, please see the article by Strohecker and colleagues on page 1272.

**Autophagy Sustains Mitochondrial Glutamine Metabolism and Growth of \( \text{Braf}^{\text{V600E}} \)-Driven Lung Tumors**


Précis: Autophagy ablation suppresses the growth of \( \text{Braf}^{\text{V600E}} \)-driven lung tumors by limiting glutamine availability and impairing mitochondrial function.

See commentary, p. 1225

**Targeting the Wnt Pathway in Synovial Sarcoma Models**


Précis: Constitutive activation of WNT/\( \beta \)-catenin signaling by the SYT-SSX oncogene is required for the initiation and progression of synovial sarcoma.
CANCER DISCOVERY

3 (11)


Updated version  Access the most recent version of this article at:
http://cancerdiscovery.aacrjournals.org/content/3/11

E-mail alerts  Sign up to receive free email-alerts related to this article or journal.
Reprints and Subscriptions  To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.
Permissions  To request permission to re-use all or part of this article, contact the AACR Publications Department at permissions@aacr.org.