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Highlighted research articles

NEWS IN BRIEF
Important news stories affecting the community

NEWS IN DEPTH
Q&A: Louis Staudt on Genomics Initiatives
Moving Ahead with Personalized Mouse Models

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

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

VIEWS
Small RNAs Deliver a Blow to Ovarian Cancer
Androgen Receptor Signaling Fuels DNA Repair and Radioresistance in Prostate Cancer
Tumor-Promoting and -Suppressive Roles of Autophagy in the Same Mouse Model of BrafV600E-Driven Lung Cancer

RESEARCH ARTICLES
Clinical Response to a Lapatinib-Based Therapy for a Li-Fraumeni Syndrome Patient with a Novel HER2V659E Mutation

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

Précis: Antiandrogen therapy suppresses androgen receptor–mediated induction of DNA repair genes, resulting in increased DNA damage and enhanced radiosensitivity of prostate cancer cells.

A Hormone–DNA Repair Circuit Governs the Response to Genotoxic Insult

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.
Strohecker and colleagues found that deletion of the essential autophagy gene Atg7 initially induced oxidative stress and accelerated the formation of BrafV600E-driven lung tumors but eventually slowed tumor growth and prolonged survival. Atg7 deficiency led to an accumulation of morphologically and functionally defective mitochondria in BrafV600E-driven lung tumors and rendered tumor cells dependent on exogenously supplied glutamine for survival. BrafV600E-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.

Précis: Autophagy ablation suppresses the growth of BrafV600E-driven lung tumors by limiting glutamine availability and impairing mitochondrial function.

See commentary, p. 1225

ON THE COVER

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