Clinical Response to a Lapatinib-Based Therapy for a Li-Fraumeni Syndrome Patient with a Novel HER2 V659E 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.

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, F.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.

In The Spotlight
Small RNAs Deliver a Blow to Ovarian Cancer . . . . . . . 1220
A. Kasinski and F.J. Slack
See article, p. 1302

Androgen Receptor Signaling Fuels DNA Repair and Radioresistance in Prostate Cancer . . . . . . . 1222
J. Bartek, M. Mistrik, and J. Bartkova
See article, p. 1245
See article, p. 1254

Tumor-Promoting and -Suppressive Roles of Autophagy in the Same Mouse Model of Braf V600E-Driven Lung Cancer . . . . . . . 1225
S. Chen and J.-L. Guan
See article, p. 1272

Misregulation of Pre-mRNA Alternative Splicing in Cancer . . . . . . . 1228
J. Zhang and J.L. Manley
See article, p. 1272
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.