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**RESEARCH ARTICLES**

Genomic Complexity Profiling Reveals That HORMAD1 Overexpression Contributes to Homologous Recombination Deficiency in Triple-Negative Breast Cancers


Précis: The meiotic protein HORMAD1 is aberrantly expressed in a subset of triple-negative breast cancers and drives allelic imbalance by suppressing RAD51-dependent homologous recombination.

Suppression of Early Hematogenous Dissemination of Human Breast Cancer Cells to Bone Marrow by Retinoic Acid–Induced 2


Précis: Retinoic acid–induced 2 (RAI2) maintains the differentiation of luminal breast epithelial cells and is a suppressor of early occurring bone metastasis in ERα-positive breast cancer.

See commentary, p. 466

In The Spotlight

RAI2: Linking Retinoic Acid Signaling with Metastasis Suppression

M. Esposito and Y. Kang

See article, p. 506

Tumor Twitter: Cellular Communication in the Breast Cancer Stem Cell Niche

M.D. Brooks and M.S. Wicha

See article, p. 520

Understanding the MIG6–EGFR Signaling Axis in Lung Tumorigenesis

E. Izumchenko and D. Sidransky

See article, p. 534

**REVIEWS**

The BCL2 Family: Key Mediators of the Apoptotic Response to Targeted Anticancer Therapeutics

A.N. Hata, J.A. Engelman, and A.C. Faber

See commentary, p. 469

Intratumoral Heterogeneity in a Trp53-Null Mouse Model of Human Breast Cancer


Précis: Paracrine WNT signaling between subpopulations of tumor cells promotes tumor-initiating cell self-renewal and tumorigenicity in a Trp53-null mouse model of breast cancer.

See commentary, p. 520
Loss of MIG6 Accelerates Initiation and Progression of Mutant Epidermal Growth Factor Receptor–Driven Lung Adenocarcinoma .................................. 534
Précis: MIG6 functions as a tumor suppressor in mutant EGFR–driven lung cancer, and its inhibitory function may be reversed by mutant EGFR–mediated hyperphosphorylation.
See commentary, p. 472

Suppression of CHK1 by ETS Family Members Promotes DNA Damage Response Bypass and Tumorigenesis ......................... 550
Précis: ETS factors transcriptionally repress CHK1, resulting in accumulation of DNA damage, accelerated prostate cancer progression, and enhanced sensitivity of prostate cancer cells to etoposide.

Using a mouse model of breast cancer driven by Trp53 loss, Zhang and colleagues investigated the functional interaction between CD29hiCD24hi tumor-initiating cells (TIC) and a mesenchymal-like CD29hiCD24lo tumor cell subpopulation. These CD29hiCD24lo cells promoted the self-renewal capacity of TICs via secretion of soluble factors including WNT2 and chemokine (C-X-C motif) ligand 12 (CXCL12). Depletion of WNT2 and CXCL12 in CD29hiCD24lo niche cells or of the corresponding receptors in TICs impaired the ability of CD29hiCD24lo cells to promote TIC self-renewal in vitro. Furthermore, in limiting dilution transplantation assays, CD29hiCD24lo niche cells enhanced the tumor-initiating potential of TICs, which was decreased by knockdown of WNT2 in the CD29hiCD24lo subpopulation. These data highlight the importance of paracrine crosstalk between different tumor cell subpopulations in promoting tumor initiation. For details, please see the article by Zhang and colleagues on page 520.
CANCER DISCOVERY

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