The Promise of Combining Inhibition of PI3K and PARP as Cancer Therapy

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Measuring Oncogenic Signaling Pathways in Cancer with PET: An Emerging Paradigm from Studies in Castration-Resistant Prostate Cancer

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Androgen Receptor Signaling in Circulating Tumor Cells as a Marker of Hormonally Responsive Prostate Cancer


Précis: Automated immunofluorescence imaging of circulating tumor cells can noninvasively detect androgen receptor activity in patients with metastatic prostate cancer.

Distinct DNA methylation profiles and gene expression patterns are associated with expression of leukemic fusion proteins in adult B-ALLs with poor outcome.


Précis: Distinct epigenetic mechanisms distinguish TMPRSS2-ERG fusion-positive and -negative prostate cancers.

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The Potential of Circulating Tumor Cells as a Liquid Biopsy to Guide Therapy in Prostate Cancer

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Fingerprinting Acute Leukemia: DNA Methylation Profiling of B-Acute Lymphoblastic Leukemia

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Emphasizing the Provocative

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Genome-wide DNA Methylation Events in TMPRSS2–ERG Fusion-Negative Prostate Cancers Implicate an EZH2-Dependent Mechanism with miR-26a Hypermethylation .......... 1024
Précis: EZH2 overexpression is caused by miR-26a hypermethylation in prostate cancers lacking the TMPRSS2–ERG gene fusion, which have distinct DNA methylation profiles.

PI3K Inhibition Impairs BRCA1/2 Expression and Sensitizes BRCA-Proficient Triple-Negative Breast Cancer to PARP Inhibition ............ 1036
Précis: PI3K suppression represses BRCA1/2 expression and increases the sensitivity of BRCA-wild-type breast cancer cells to PARP inhibitors via ERK activation.

Combining a PI3K Inhibitor with a PARP Inhibitor Provides an Effective Therapy for BRCA1-Related Breast Cancer ............ 1048
Précis: PI3K inhibition synergizes with PARP inhibitors in vivo to decrease the growth of BRCA1-mutant breast tumors, revealing a role for PI3K in the DNA damage response.