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Précis: Combined inhibition of RAF and EGFR may be necessary to effectively suppress MAPK signaling in BRAF-mutant colorectal cancers.

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B. Benassi, R. Flavin, L. Marchionni, S. Zanata, Y. Pan, D. Chowdhury, M. Marani, S. Strana, P. Muti, G. Blandino, and M. Loda
Précis: Overexpression of USP2a activates MYC and promotes prostate cancer growth and invasiveness via downregulation of miR-34b/c.
Akt/PKB-Mediated Phosphorylation of Twist1 Promotes Tumor Metastasis via Mediating Cross-Talk between PI3K/Akt and TGF-β Signaling Axes


Précis: Phosphorylation of TWIST1 by AKT promotes EMT and metastasis via TGF-β2 transcriptional regulation and PI3K/AKT feedback activation.

nab-Paclitaxel Potentiates Gemcitabine Activity by Reducing Cytidine Deaminase Levels in a Mouse Model of Pancreatic Cancer


Précis: Combined nab-paclitaxel and gemcitabine therapy leads to synergistic antitumor effects due to decreased gemcitabine metabolism.

Suppression of Tumor Invasion and Metastasis by Concurrent Inhibition of c-Met and VEGF Signaling in Pancreatic Neuroendocrine Tumors


Précis: Combined inhibition of VEGF and c-MET reduces the tumor invasiveness and metastasis observed after inhibition of VEGF alone and decreases tumor growth and angiogenesis.

For more News and Research Watch, visit Cancer Discovery online at www.AACR.org/CDnews. Online-only News stories include the following:

- Tracking Down Tumor-Targeting Bacteria
- Can Chemotherapy Cause Cancer Relapse?
- Antiangiogenic Drugs Increase Xenograft Aggressiveness
- Mutations, Tissue Type Both Influence Cancer Metabolism

ON THE COVER Frese and colleagues utilized a genetically engineered mouse model of pancreatic ductal adenocarcinoma (PDA) to better understand the mechanistic basis for the clinical observation that nab-paclitaxel, a water-soluble, albumin-bound form of paclitaxel, elicits synergistic antitumor activity when combined with gemcitabine, a nucleoside analogue that is the current standard of care for PDA. Combination treatment with nab-paclitaxel increases intratumoral gemcitabine levels by creating an oxidative environment within the tumor that promotes degradation of cytidine deaminase, the primary gemcitabine metabolizing enzyme. For details, please see the article by Frese and colleagues on page 260.