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1135 Vasohibin-2 Expressed in Human Serous Ovarian Adenocarcinoma Accelerates Tumor Growth by Promoting Angiogenesis
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1147 Inhibition of the Hedgehog Pathway Targets the Tumor-Associated Stroma in Pancreatic Cancer
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1158 Cancer-Associated Fibroblasts Induce Matrix Metalloproteinase–Mediated Cetuximab Resistance in Head and Neck Squamous Cell Carcinoma Cells
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CELL CYCLE, CELL DEATH, AND SENESCENCE

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1178 Group 1 p21-Activated Kinases (PAKs) Promote Tumor Cell Proliferation and Survival through the AKT1 and Raf–MAPK Pathways
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1189 The p38 MAPK–MK2 Axis Regulates E2F1 and FOXM1 Expression after Epirubicin Treatment
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1203 S-Nitrosylation of EGFR and Src Activates an Oncogenic Signaling Network in Human Basal-Like Breast Cancer
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1216 Hepatocyte Growth Factor Enhances Alternative Splicing of the Kruppel-like Factor 6 (KLF6) Tumor Suppressor to Promote Growth through SRSF1
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1228 KRASG12D- and BRAFV600E-Induced Transformation of Murine Pancreatic Epithelial Cells Requires MEK/ERK-Stimulated IGF1R Signaling
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ABOUT THE COVER

Group I p21-activated kinases (PAKs) regulate cell survival, proliferation and motility, all factors that contribute to tumorigenesis. The tumor suppressor NF2 negatively regulates group I PAKs, and mutation or loss of NF2 leads to subsequent PAK activation. Using immunohistochemistry, PAK was found to be phosphorylated/activated in asbestos-induced malignant mesotheliomas from Nf2-deficient mice. Inhibition of group I PAKs in patient-derived mesothelioma cell lines was sufficient to inhibit tumor cell proliferation and viability via inactivation of the AKT and Raf-MAPK pathways, suggesting that PAKs represent novel targets for therapeutic intervention in NF2-deficient malignancies. For details, see article by Menges and colleagues on page 1178.