Knockdown of PAK4 or PAK1 Inhibits the Proliferation of Mutant KRAS Colon Cancer Cells Independently of RAF/MEK/ERK and PI3K/AKT Signaling
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Crizotinib-Resistant NPM-ALK Mutants Confer Differential Sensitivity to Unrelated Alk Inhibitors
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The ATPase Activity of Reptin Is Required for Its Effects on Tumor Cell Growth and Viability in Hepatocellular Carcinoma
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Synergistic Effect of Olaparib with Combination of Cisplatin on PTEN-Deficient Lung Cancer Cells
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CPEB1 Regulates the Expression of MTDH/AEG-1 and Glioblastoma Cell Migration
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MYC-Induced Epigenetic Activation of GATA4 in Lung Adenocarcinoma
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CREB-Binding Protein Regulates Ku70 Acetylation in Response to Ionization Radiation in Neuroblastoma
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miRNA-145 Targets v-ets Erythroblastosis Virus E26 Oncogene Homolog 1 to Suppress the Invasion, Metastasis, and Angiogenesis of Gastric Cancer Cells
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CLT1 Targets Bladder Cancer through Integrin α5β1 and CLIC3
Lynn M. Knowles, James Zewe, Gunjan Malik, Anil V. Parwani, Jeffrey R. Gingrich, and Jan Pilch
ABOUT THE COVER

Crizotinib is a selective ALK inhibitor, recently approved for the treatment of ALK+ non-small cell lung cancer and currently in clinical trials for other ALK-related malignancies. By using a human cell-based screening approach, the appearance of two point mutations able to confer crizotinib resistance was observed. One of these mutations is sensitive to two structurally unrelated ALK inhibitors, NVP-TAE 684 and the clinically relevant AP26113, while the second one is resistant to all drugs. For further details, please see Ceccon and colleagues on page 122 in this issue.