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1265  The Key Role of Calmodulin in KRAS-Driven Adenocarcinomas
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CELL DEATH AND SURVIVAL

1274  VEGFR-1 Pseudogene Expression and Regulatory Function in Human Colorectal Cancer Cells
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1283  Ca2⁺-Activated IK K⁺ Channel Blockade Radiosensitizes Glioblastoma Cells
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1296  Suppression of Reserve MCM Complexes Chemosensitizes to Gemcitabine and 5-Fluorouracil
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GENOMICS

1306  Patient Mutation Directed shRNA Screen Uncovers Novel Bladder Tumor Growth Suppressor
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1316  E2F4 Program Is Predictive of Progression and Intravesical Immunotherapy Efficacy in Bladder Cancer
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ONCOGENES AND TUMOR SUPPRESSORS

1325  Biochemical and Structural Analysis of Common Cancer-Associated KRAS Mutations
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1336  PKCε Is an Essential Mediator of Prostate Cancer Bone Metastasis
Alvaro Gutierrez-Uzquiza, Cynthia Lopez-Haber, Danielle L. Jerimag, Alessandro Fatatis, and Marcelo G. Kazanietz

SIGNAL TRANSDUCTION

1347  miR-181a Targets RGS16 to Promote Chondrosarcoma Growth, Angiogenesis, and Metastasis
Xiaojuan Sun, Cherie Charbonneau, Lei Wei, Qian Chen, and Richard M. Terek
ABOUT THE COVER

The 1.1 Å x-ray crystal structure of oncogenic KRAS G13D, reported by Hunter and colleagues (p.1325), reveals a destabilizing interaction between the aspartate side-chain and GDP. The calculated electrostatic potential surface map highlights the negative charge (red) introduced by the aspartate, which disrupts the normally positively charged (blue) nucleotide binding pocket and repels adjacent negatively charged phosphates in GDP, explaining the enhanced rate of nucleotide exchange observed for this mutant.