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Precis: This study of global DNA methylation in the most deadly form of brain cancer reveals a simple prognostic marker, with potential implications for treatment.

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Parathyroid Hormone–Related Protein Drives a CD11b+Gr1+ Cell–Mediated Positive Feedback Loop to Support Prostate Cancer Growth
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Precis: A cancer cell-secreted bone regulatory factor promoting hypercalcemia has a pivotal role in recruiting a class of immune-suppressor cells that drive tumor angiogenesis and progression.

MOLECULAR AND CELLULAR PATHOBIOLOGY

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Genetic and Pharmacologic Inhibition of mTORC1 Promotes EMT by a TGF-β–Independent Mechanism
Ivan Mikaelian, Mouhannad Malek, Rudy Gadet, Jean Viallet, Amandine Garcia, Anais Girard-Gagnepain, Cedric Hesling, Germain Gillet, Philippe Gonzalez, Ruth Rimokh, and Marc Billaud

Precis: This important study raises concerns about using mTORC1 inhibitors for clinical management of cancer, given that they not only impair tumor immunity but also even promote EMT in epithelial cells, perhaps explaining the progressive pulmonary fibrosis associated with therapeutic use of mTOR inhibitors.
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Personalizing the Treatment of Pediatric Medulloblastoma: Polo-like Kinase 1 as a Molecular Target in High-Risk Children

Crizotinib Inhibits Metabolic Inactivation of Gemcitabine in c-Met–driven Pancreatic Carcinoma
Amir Avan, Viola Caretti, Niccola Funel, Elena Galvani, Mina Maftouh, Richard J. Honeywell, Tonny Lagerweij, Olaf van Tellingen, Daniela Campani, Dieter Fuchs, Henk M. Verheul, Gerrit-Jan Schuurhuis, Ugo Boggi, Godfrieds J. Peters, Thomas Wurdinger, and Elisa Giovannetti

Chk1 Targeting Reactivates PP2A Tumor Suppresser Activity in Cancer Cells

Cetuximab Response of Lung Cancer–Derived EGF Receptor Mutants Is Associated with Asymmetric Dimerization

Taccalonolide Binding to Tubulin Imparts Microtubule Stability and Potent In Vivo Activity

Small-Molecule Intramimics of Formin Autoinhibition: A New Strategy to Target the Cytoskeletal Remodeling Machinery in Cancer Cells

Requirements for Aurora-A in Tissue Regeneration and Tumor Development in Adult Mammals
Ignacio Pérez de Castro, Cristina Aguirre-Portolés, Gonzalo Fernández-Miranda, Marta Cañamero, Dale O. Cowley, Terry Van Dyke, and Marcos Malumbres

Precise: These findings suggest repositioning inhibitors of a critical mitotic kinase, currently in clinical testing, to treat a deadly pediatric tumor.

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RNAi-Mediated Silencing of Myc Transcription Inhibits Stem-like Cell Maintenance and Tumorigenicity in Prostate Cancer
Gianluca Civenni, Anastasia Malek, Domenico Albino, Ramon Garcia-Escudero, Sara Napoli, Stefano Di Marco, Sandra Pinton, Manuela Sarti, Giuseppina M. Carbone, and Carlo V. Catapano
Précis: This important study offers a preclinical proof of concept to target Myc function in cancer stem-like cells as a general strategy to attack most if not all human cancers.

MyoD Is a Tumor Suppressor Gene in Medulloblastoma
Joyoti Dey, Adrian M. Dubuc, Kyle D. Pedro, Derek Thirstrup, Brig Mecham, Paul A. Northcott, Xiaochong Wu, David Shih, Stephen J. Tapscott, Michael LeBlanc, Michael D. Taylor, and James M. Olson
Précis: A central muscle differentiation factor is for the first time shown to be expressed during development of the cerebellum and to function there as a tumor suppressor.

ABOUT THE COVER
Diaphanous-related formins create new and/or stabilize microfilament and microtubule structures that support polarized cell adhesion, migration, and division. GTP-bound Rho proteins activate these formins by direct binding. The molecular mechanism of Rho activation is through steric disruption of intramolecular interactions between Dia-inhibitory (DID) and Dia-autoregulatory (DAD) domains. Screening for compounds that block DID-DAD binding led to the discovery of intramimics, which are small molecules that interfere with autoinhibition, resulting in activation of cellular formins. Using immunofluorescence to detect detyrosinated microtubules (a trait of stabilized microtubules), this image illustrates microtubules stabilized by intramimic exposure. For details on the mechanism and pharmacologic impairment of tumor growth, see article by Lash and colleagues on page 6793.

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