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**Androgen Receptor Interaction with Mediator Complex Is Enhanced in Castration-Resistant Prostate Cancer by CDK7 Phosphorylation of MED1** ... 1490
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Altered RNA Processing in Cancer Pathogenesis and Therapy... 1493
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### RESEARCH BRIEF
Infiltrating Myeloid Cells Drive Osteosarcoma Progression via GRM4 Regulation of IL23 ....... 1511

**Précis:** Glutamate metabotropic receptor 4 regulates the expression of the inflammatory cytokine IL23 in mice, leading to a restriction of osteosarcoma development that can be therapeutically targeted.

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### RESEARCH ARTICLES
Antitumor T-cell Homeostatic Activation Is Uncoupled from Homeostatic Inhibition by Checkpoint Blockade ........... 1520

**Précis:** Combination treatment with PD-1 and CLTA4 inhibitors both before and after bone-marrow transplant into lymphodepleted recipient mice increased antitumor efficacy over immune-checkpoint blockade or bone-marrow transplant alone in a γ-dependent manner.

See commentary, p. 1487

CDK7 Inhibition Suppresses Castration-Resistant Prostate Cancer through MED1 Inactivation ............... 1538

**Précis:** Phosphorylation of MED1 by the cyclin-dependent kinase CDK7 leads to an interaction between MED1 and AR, and CDK7 inhibition causes tumor regression in a mouse model of castration-resistant prostate cancer.

See commentary, p. 1490
Targeting Glioblastoma Stem Cells through Disruption of the Circadian Clock. 1556
Précis: The core circadian proteins BMAL1 and CLOCK are essential to the maintenance of glioblastoma stem cells, and genetic or pharmacologic inhibition of BMAL1 or CLOCK increased survival in a mouse model of glioblastoma.

ADAMDEC1 Maintains a Growth Factor Signaling Loop in Cancer Stem Cells. 1574
Précis: A positive feedback loop involving the disintegrin metalloproteinase ADAMDEC1 maintains the stemness of recurrence-driving glioblastoma stem cells.

An LKB1–SIK Axis Suppresses Lung Tumor Growth and Controls Differentiation. 1590
Précis: An in vivo CRISPR screen of LKB1 targets identified SIK1 and SIK3 as mediators of the tumor-suppressive effects of LKB1 in the lung.

The AMPK-Related Kinases SIK1 and SIK3 Mediate Key Tumor-Suppressive Effects of LKB1 in NSCLC. 1606
Précis: Lack of activation of the AMPK-related kinases SIK1 and SIK3 may be responsible for the tumorigenic effect of LKB1 mutations in non–small cell lung cancer.

Corrections
Correction: NF2 Loss Promotes Oncogenic RAS-Induced Thyroid Cancers via YAP-Dependent Transactivation of RAS Proteins and Sensitizes Them to MEK Inhibition. 1628
Correction: Cell-Cycle Reprogramming for PI3K Inhibition Overrides a Relapse-Specific C481S BTK Mutation Revealed by Longitudinal Functional Genomics in Mantle Cell Lymphoma. 1629

ON THE COVER Murray and colleagues as well as Hollstein and colleagues showed that the tumorigenic effects of disruption of the kinase LKB1, frequently mutated in lung cancer, are mediated by its downstream kinases SIK1 and SIK3. SIK1 and SIK3 acted synergistically, with their combined disruption leading to a tumor burden and average tumor size comparable to those of LKB1-deficient tumors in mouse lung cancer models. Histologic and molecular analyses revealed further overlap between the combined SIK1 and SIK3 mutants and LKB1 mutants. This pair of studies provides clarification of the mechanisms underlying the effects of LKB1 loss and provides a basis for further investigation and therapeutic targeting of the LKB1–SIK signaling axis in lung cancer. For details, please see the article by Murray and colleagues on page 1590 and the article by Hollstein and colleagues on page 1606.
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