Paracrine Signaling between Carcinoma Cells and Mesenchymal Stem Cells Generates Cancer Stem Cell Niche via Epithelial–Mesenchymal Transition... 775
K. Räsänen and M. Herlyn
Commentary on Li et al., p. 840

Investigating Metformin for Cancer Prevention and Treatment: The End of the Beginning... 778
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BRAF L597 Mutations in Melanoma Are Associated with Sensitivity to MEK Inhibitors... 791
Précis: Non-V600E BRAF mutations that are sensitive to MEK inhibition occur in 8% of "BRAF–wild-type" melanomas.

Proteomic Profiling Identifies Dysregulated Pathways in Small Cell Lung Cancer and Novel Therapeutic Targets Including PARP1... 798
Précis: Small cell lung cancer cells express significantly higher levels of PARP1 than non-small cell lung cancer cells and are highly sensitive to PARP inhibition.
DDX5 Regulates DNA Replication and Is Required for Cell Proliferation in a Subset of Breast Cancer Cells 812
A. Mazurek, W. Luo, A. Krasnitz, J. Hicks, R.S. Powers, and B. Stillman
Précis: DDX5 amplification frequently occurs in breast cancer and promotes cell proliferation by controlling transcription of DNA replication genes.

CD36 Repression Activates a Multicellular Stromal Program Shared by High Mammographic Density and Tumor Tissues 826
Précis: Decreased CD36 expression in mammary stromal cells promotes a pro-oncogenic microenvironment and enhances breast cancer risk.

Cancer-Stimulated Mesenchymal Stem Cells Create a Carcinoma Stem Cell Niche via Prostaglandin E₂ Signaling 840
H-J. Li, F. Reinhardt, H.R. Herschman, and R.A. Weinberg
Précis: Bidirectional signaling between tumor cells and associated mesenchymal stem cells promotes EMT and enhances cancer stem cell formation.

Correction
Correction: Gene Signatures Associated with Mouse Postnatal Hindbrain Neural Stem Cells and Medulloblastoma Cancer Stem Cells Identify Novel Molecular Mediators and Predict Human Medulloblastoma Molecular Classification 856
Dahlman and colleagues identified a BRAF<sup>L597R</sup> mutation in an aggressive BRAF<sup>V600E</sup>-negative melanoma, and found that as many as 8% of melanomas classified clinically as "BRAF wild type" may actually harbor other less common BRAF exon 15 mutations. Importantly, these mutants led to increased MEK/ERK signaling that was readily suppressed by MEK inhibitors, suggesting that patients with these less common BRAF mutations may also benefit from MEK inhibitor therapy. Indeed, one such patient with metastatic melanoma enrolled in a phase I trial of an allosteric MEK inhibitor experienced a sustained partial response, indicating that expanded BRAF mutational testing may benefit additional patients. For details, please see the article by Dahlman and colleagues on page 791.