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PROTEOMIC PROFILING IDENTIFIES DYSREGULATED PATHWAYS IN SMALL CELL LUNG CANCER AND NOVEL THERAPEUTIC TARGETS INCLUDING PARP1 ................. 798
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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.

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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

For more News and Research Watch, visit Cancer Discovery online at http://CDnews.aacrjournals.org. Online-only News stories include the following:

• TCGA Findings Spotlight Drivers for Colorectal Cancer
• Kyprolis Gains Accelerated Approval for Multiple Myeloma
• Novel Biostatistics May Speed Access to Pediatric Drugs
• Method Yields Single-Cell Transcriptomes
• A Better Way to Grow Tumor-Initiating Cells?
• Ultrasound System Finds Tumor Blood Vessels

Dahlman and colleagues identified a BRAF\textsuperscript{L597R} mutation in an aggressive BRAF\textsuperscript{V600E}-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.

ON THE COVER

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