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### RESEARCH BRIEFS
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- PI3′-Kinase Inhibition Forestalls the Onset of MEK1/2 Inhibitor Resistance in BRAF-Mutated Melanoma ................. 143
  - M.M. Deuker, V. Marsh Durban, W.A. Phillips, and M. McMahon
  - Précis: Treatment with PI3K inhibitors enhances the depth of response to MEK1/2 inhibition and delays the development of drug-resistant tumors in BRAF-mutated melanoma mouse models.

- Linking Tumor Mutations to Drug Responses via a Quantitative Chemical–Genetic Interaction Map .......................... 154
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Aberrant Glycosylation Promotes Lung Cancer Metastasis through Adhesion to Galectins in the Metastatic Niche ...................... 168
N.E. Reticker-Flynn and S.N. Bhatia
Précis: Changes in glycosyltransferase activity in lung cancer cells enhance surface presentation of the carbohydrate ligand T-antigen and potentiate metastasis via increased binding to tumor-mobilized galectin-3+ leukocytes.
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NOTCH Decoys That Selectively Block DLL/NOTCH or JAG/NOTCH Disrupt Angiogenesis by Unique Mechanisms to Inhibit Tumor Growth ................. 182
Précis: Specific inhibition of JAG- or DLL-mediated NOTCH signaling using synthetic NOTCH1 decoys inhibits tumor angiogenesis via distinct mechanisms.
See commentary, p. 112

Promotion of Colorectal Cancer Invasion and Metastasis through Activation of NOTCH-DAB1–ABL–RHOGEF Protein TRIO .................. 198
M. Sonoshita, Y. Itatani, F. Kakizaki, K. Sakimura, T. Terashima, Y. Katsuyama, Y. Sakai, and M.M. Taketo
Précis: NOTCH-DAB1 signaling promotes colon cancer cell invasion and progression by stimulating ABL-mediated phosphorylation of the RHOGEF TRIO at tyrosine residue 2681.
See commentary, p. 115

Reticker-Flynn and Bhatia found that tumor-derived IL6 induced CD11b+-galectin-3+ leukocyte mobilization from the bone marrow in a mouse model of lung adenocarcinoma. Metastatic cell lines and human non–small cell lung cancer samples exhibited increased surface presentation of the galectin-3 ligand, Thomsen-Friedenreich Antigen (T-Antigen). Elevated T-Antigen surface presentation was mediated by altered expression of the glycosyltransferases C2GnT2 and St6GalNAc4, which prevented T-Antigen glycan elongation. Restoration of T-Antigen glycan chain elongation decreased T-Antigen presentation, reduced tumor-cell galectin-3 binding, and inhibited experimental metastases in vivo. These results indicate that aberrant glycosyltransferase activities play a critical role in the early metastatic niche to promote metastatic progression of lung tumors. For details, please see the article by Reticker-Flynn and Bhatia on page 168.
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