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STK11/LKB1 Mutations and PD-1 Inhibitor Resistance in KRAS-Mutant Lung Adenocarcinoma .................. 822
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Précis: High-throughput enhancer profiling identifies metastasis-associated enhancers that are activated to drive CXCR4 expression and metastatic colonization in clear cell renal cell carcinoma.

PTEN Deficiency and AMPK Activation Promote Nutrient Scavenging and Anabolism in Prostate Cancer Cells .... 866
Précis: AMPK-dependent macropinocytosis of necrotic cell debris enhances the growth and survival of PTEN-deficient prostate cancer cells.

ON THE COVER
Skoulidis, Goldberg, Greenawalt, and colleagues linked STK11 mutations to PD-1 inhibitor resistance in KRAS-mutant lung cancer. Patients with lung cancer harboring co-occurring STK11 and KRAS alterations had a lower response rate to PD-1/PD-L1 blockade than patients with co-occurring KRAS and TP53 alterations or KRAS mutations alone. STK11 alterations were enriched in PD-L1-negative tumors with an intermediate to high tumor mutation burden. However, STK11 alterations were also associated with primary resistance to PD-1 blockade in patients with PD-L1-positive tumors. STK11 deletion induced de novo resistance to PD-1 inhibition in a mouse model of KRAS-mutant lung adenocarcinoma. These results demonstrate that STK11 alterations confer primary resistance to PD-1/PD-L1 blockade and suggest that genomic profiling may identify patients likely to benefit from PD-1 blockade. For details, please see the article by Skoulidis, Goldberg, Greenawalt, and colleagues on page 822.
CANCER DISCOVERY

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