B. Kocher and D. Piwnica-Worms

Response to Cabozantinib in Patients with RET Fusion-Positive Lung Adenocarcinomas


See commentary, p. 604

Précis: Preliminary data from a prospective phase II trial shows cabozantinib elicits prolonged partial responses and disease stabilization in non-small cell lung cancers harboring RET fusions.


See commentary, p. 607

Précis: FGFR gene fusions that encode for active kinases are present in multiple cancer types and confer enhanced sensitivity to FGFR inhibitors.


A New Alpha in Line Between KRAS and NF-κB Activation?

C. Pak and S. Miyamoto

See article, p. 690

Précis: SDH-deficient tumors of various lineages are characterized by a divergent DNA hypermethylation profile comparable to that of other Krebs cycle-defective tumors.
Amplification of the MET Receptor Drives Resistance to Anti-EGFR Therapies in Colorectal Cancer  

Précis: MET amplification underlies acquired resistance to cetuximab or panitumumab in colorectal cancers that have not developed secondary KRAS mutations.

Canonical Wnt/β-catenin Signaling Drives Human Schwann Cell Transformation, Progression, and Tumor Maintenance

Précis: WNT pathway activation induces oncogenic properties in Schwann cells and promotes growth of malignant peripheral nerve sheath tumors.

GSK-3α Promotes Oncogenic KRAS Function in Pancreatic Cancer via TAK1–TAB Stabilization and Regulation of Noncanonical NF-κB
D. Bang, W. Wilson, M. Ryan, J.J. Yeh, and A.S. Baldwin

Précis: GSK3α but not GSK3β enhances pancreatic cell growth downstream of mutant KRAS via coordinate activation of both canonical and noncanonical NF-κB signaling.

Killian and colleagues found that gastrointestinal stromal tumors (GIST) with mutations in succinate dehydrogenase (SDH) complex genes exhibited a distinct methylation signature relative to the profile of KIT-mutant tumors and normal reference tissues. This methyl-divergent profile was distinguished by increased global DNA hyper-methylation, particularly at DNase hypersensitive sites, and was also present in other SDH-mutant tumor lineages, including paragangioma and pheochromocytoma, supporting the oncogenotype dependence of this signature. In addition, a similarly perturbed methylation profile was detected in gliomas harboring mutations in another Krebs cycle enzyme, isocitrate dehydrogenase (IDH). These findings identify a strong association between the mitochondrial Krebs cycle and cancer epigenomic reprogramming. For details, please see the article by Killian and colleagues on page 648.

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