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Columns: Editorial

Promise and challenges on the horizon of MET-targeted cancer therapeutics

Yu-Wen Zhang

Abstract

MET is one of the receptor tyrosine kinases (RTKs) whose activities are frequently altered in human cancers, and it is a promising therapeutic target. MET is normally activated by its lone ligand, hepatocyte growth factor (HGF), eliciting its diverse biological activities that are crucial for development and physiology. Alteration of the HGF-MET axis results in inappropriate activation of a cascade of intracellular signaling pathways that contributes to hallmark cancer events including deregulated cell proliferation and survival, angiogenesis, invasion, and metastasis. Aberrant MET activation results from autocrine or paracrine mechanisms due to overexpression of HGF and/or MET or from a ligand-independent mechanism caused by activating mutations or amplification of *MET*. The literature provides compelling evidence for the role of MET signaling in cancer development and progression. The finding that cancer cells often use MET activation to escape therapies targeting other pathways strengthens the argument for MET-targeted therapeutics. Diverse strategies have been explored to

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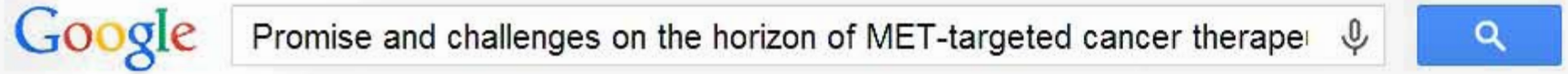

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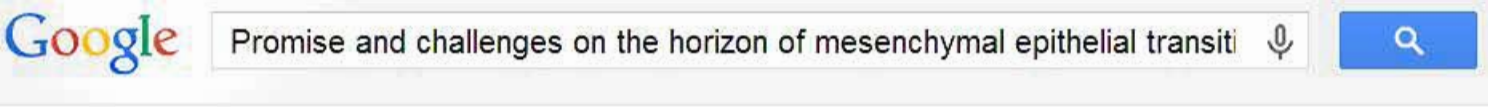

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