Targeting MET for glioma therapy.
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Abstract
Glioblastoma multiforme is the most common and most lethal of all primary brain tumors. Even with the standard therapy, life expectancy is still poor, with an average survival of approximately 14 months following initial diagnosis. Hence, there is an urgent need for novel treatment strategies that inhibit proliferation and angiogenesis in high-grade gliomas. One such strategy consists of inhibiting receptor tyrosine kinases, including MET and/or its ligand hepatocyte growth factor (HGF). Because of their widespread involvement in human cancer, HGF and MET have emerged as promising therapeutic targets, and some inhibitory agents that target them have already entered clinical trials. In this paper, the authors highlight recent evidence implicating HGF/MET pathway deregulation in glioblastoma multiforme, discuss therapeutic approaches to inhibit HGF/MET signaling, and summarize ongoing clinical trials targeting this pathway.

KEYWORDS: GBM = glioblastoma multiforme; GSC = glioma stem cell; HGF = hepatocyte growth factor; MET; RTK = receptor tyrosine kinase; glioblastoma multiforme; hepatocyte growth factor; mAb = monoclonal antibody

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