Drug-mediated inhibition of MET activation

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**Introduction**

Reactome is an open-source, open-access, manually curated, and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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**Literature references**


Reactome database release: 79

This document contains 1 pathway and 2 reactions (see Table of Contents)
Drug-mediated inhibition of MET activation

Stable identifier: R-HSA-9734091

MET receptor tyrosine kinase (RTK) is a proto-oncogene that is frequently aberrantly activated in cancer through gene amplification and/or activating mutations that result in hypersensitivity to HGF stimulation or HGF-independent activation. Oncogenic MET activation can occur as a primary mechanism of malignant transformation or be selected secondarily, as a mechanism of resistance to therapeutics that target related RTKs, such as EGFR. MET targeted anti-cancer therapeutics, either recombinant monoclonal antibodies (MAbs) or small tyrosine kinase inhibitors (TKIs), have shown promise as a first-line agents for the treatment of solid tumors with primary MET activation or as second-line agents for the treatment of solid tumors with acquired MET-mediated resistance to other RTK-targeted therapies (reviewed in Comoglio et al. 2018).

Literature references

Editions

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Anti-MET recombinant therapeutic antibodies bind MET

Location: Drug-mediated inhibition of MET activation

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