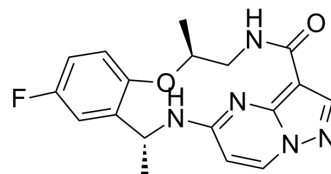


Repotrectinib

Cat. No.:	HY-103022	
CAS No.:	1802220-02-5	
Molecular Formula:	C ₁₈ H ₁₈ FN ₅ O ₂	
Molecular Weight:	355.37	
Target:	ROS Kinase; Trk Receptor; Anaplastic lymphoma kinase (ALK)	
Pathway:	Protein Tyrosine Kinase/RTK; Neuronal Signaling	
Storage:	Powder	-20°C 3 years 4°C 2 years
	In solvent	-80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 25 mg/mL (70.35 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.8140 mL	14.0698 mL	28.1397 mL
		5 mM	0.5628 mL	2.8140 mL	5.6279 mL
10 mM		0.2814 mL	1.4070 mL	2.8140 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (7.03 mM); Clear solution 2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (7.03 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Repotrectinib (TPX-0005) is a potent ROS1 (IC ₅₀ =0.07 nM) and TRK (IC ₅₀ =0.83/0.05/0.1 nM for TRKA/B/C) inhibitor. Repotrectinib potently inhibits WT ALK (IC ₅₀ =1.01 nM). Repotrectinib has anti-cancer activity ^{[1][2]} .
IC₅₀ & Target	TrkA
In Vitro	Repotrectinib (TPX-0005) inhibits mutant ALKs including ALK G1202R (IC ₅₀ =1.26 nM) and ALK L1196M (IC ₅₀ =1.08 nM). Repotrectinib also inhibits a variety of other kinases, including JAK2, LYN, Src, and FAK (IC ₅₀ =1.04, 1.66, 5.3, and 6.96 nM, respectively) ^[1] . Repotrectinib effectively overcomes this primary resistance (IC ₅₀ =100 nM in cell proliferation assay) with strong inhibition of the phosphorylation of EML4-ALK (IC ₅₀ =13 nM) and the SRC substrate paxillin (IC ₅₀ =107 nM). Repotrectinib inhibits H2228

cell migration in a wound healing assay with similar activity to saracatinib^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Repotrectinib (TPX-0005) effectively inhibits tumor growth in vivo in ALK WT and ALK G1202R xenografts^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancer Discov. 2023 Mar 1;13(3):598-615.
- J Med Chem. 2024 Oct 3.
- Sci Rep. 2024 Sep 27;14(1):22191.
- bioRxiv. 2024 Feb 1.

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REFERENCES

[1]. Dayong Zhai, et al. Abstract 2132: The novel, rationally-designed, ALK/SRC inhibitor TPX-0005 overcomes multiple acquired resistance mechanisms to current ALK inhibitors. Cancer Research. July 2016

[2]. Karachaliou N, et al. Common Co-activation of AXL and CDCP1 in EGFR-mutation-positive Non-smallcell Lung Cancer Associated With Poor Prognosis. EBioMedicine. 2018 Mar;29:112-127.

Caution: Product has not been fully validated for medical applications. For research use only.

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