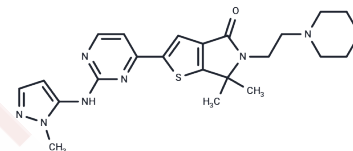


Temuterkib

Chemical Properties

CAS No. :	1951483-29-6
Formula:	C ₂₂ H ₂₇ N ₇ O ₂ S
Molecular Weight:	453.56
Storage:	Powder: -20°C for 3 years In solvent: -80°C for 1 year Actual storage temperature shall be subject to the COA.



Biological Description

Description	Temuterkib (LY3214996) is a potent and selective, orally available inhibitor of the extracellular signal-regulated kinase (ERK) 1 and 2, with potential antineoplastic activity.
Targets(IC50)	ERK
In vitro	Temuterkib is a highly selective inhibitor of ERK1 and ERK2, with IC ₅₀ of 5 nM for both enzymes in biochemical assays. Temuterkib potently inhibits cellular phospho-RSK1 in BRAF and RAS mutant cancer cell lines. In an unbiased tumor cell panel sensitivity profiling for inhibition of cell proliferation, tumor cells with MAPK pathway alterations including BRAF, NRAS or KRAS mutation are generally sensitivity to Temuterkib[1].
In vivo	In tumor xenograft models, LY3214996 effectively inhibits the PD biomarker phospho-p90RSK1 and its PD effects, correlating with compound exposure and antitumor activity. It demonstrates comparable or superior efficacy to existing ERK inhibitors in BRAF or RAS mutant cell lines and xenografts. Oral administration of LY3214996 notably suppresses tumor growth across various cancer xenografts, including BRAF or NRAS mutant melanoma, and BRAF or KRAS mutant colorectal, lung, and pancreatic cancers, showing good tolerance. It offers a promising therapeutic strategy for cancers linked to MAPK pathway alterations. Remarkably, LY3214996 also counters Vemurafenib-resistant A375 melanoma xenografts, highlighting its potential in treating melanoma patients unresponsive to BRAF therapy. Furthermore, LY3214996 can be used alongside investigational and approved treatments, especially in KRAS mutant cases. Its combination with the CDK4/6 inhibitor abemaciclib exhibits significant tumor growth inhibition or regression in diverse cancer models, including KRAS mutant colorectal and non-small cell lung cancers, showcasing its broad therapeutic potential[1].

Solubility Information

Solubility	DMSO: 7.36 mg/mL (16.23 mM),Sonication is recommended. H ₂ O: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 1 mg/mL (2.2 mM),Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and</i>

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In vivo Formulation	<i>used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.2048 mL	11.0239 mL	22.0478 mL
5 mM	0.441 mL	2.2048 mL	4.4096 mL
10 mM	0.2205 mL	1.1024 mL	2.2048 mL
50 mM	0.0441 mL	0.2205 mL	0.441 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Shripad V. Bhagwat, et al. Abstract 4973: Discovery of LY3214996, a selective and novel ERK1/2 inhibitor with potent antitumor activities in cancer models with MAPK pathway alterations. Cancer Research. July 2017.

Li X, Qin X, Tian J, et al. Liquiritin protects PC12 cells from corticosterone-induced neurotoxicity via regulation of metabolic disorders, attenuation ERK1/2-NF- κ B pathway, activation Nrf2-Keap1 pathway, and inhibition mitochondrial apoptosis pathway. Food and Chemical Toxicology. 2020: 111801

Li X, Qin X, Tian J, et al. Liquiritin protects PC12 cells from corticosterone-induced neurotoxicity via regulation of metabolic disorders, attenuation ERK1/2-NF- κ B pathway, activation Nrf2-Keap1 pathway, and inhibition mitochondrial apoptosis pathway[J]. Food and Chemical Toxicology. 2020: 111801.

Chen S J, Huang Y, Yu F, et al. BMAL1/p53 mediating bronchial epithelial cell autophagy contributes to PM2.5-aggravated asthma. Cell Communication and Signaling. 2023, 21(1): 39.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

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