DPH

Cat. No.: HY-12070

CAS No.: 484049-04-9

Molecular Formula: $C_{18}H_{13}FN_4O_2$

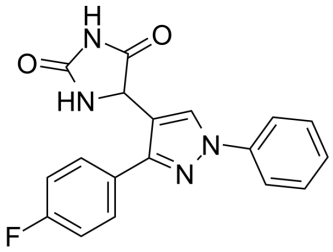
Molecular Weight: 336.32

Target: Bcr-Abl

Pathway: Protein Tyrosine Kinase/RTK

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 : 50 mg/mL (148.67 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9734 mL	14.8668 mL	29.7336 mL
	5 mM	0.5947 mL	2.9734 mL	5.9467 mL
	10 mM	0.2973 mL	1.4867 mL	2.9734 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 (7.43 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.43 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.43 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DPH is a potent cell permeable c-Abl activator, which displays potent enzymatic and cellular activity in stimulating c-Abl activation.

In Vitro

DPH binds to the myristoyl binding site and prevents the formation of the bent conformation of the αI helix through steric hindrance, a mode of action distinct from the previously identified allosteric c-Abl inhibitor, GNF-2, that also binds to the myristoyl binding site. DPH represents the first cell-permeable, small-molecule tool compound for c-Abl activation[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv. 2021 May 7;7(19):eabe5171.
- Toxicol Sci. 2018 Sep 1;165(1):232-243.
- Toxicol Lett. 2022 May 18;363:11-26.
- Mol Cell Neurosci. 2017 Dec;85:226-234.

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REFERENCES

[1]. Yang J, et al. Discovery and characterization of a cell-permeable, small-molecule c-Abl kinase activator that binds to the myristoyl binding site. Chem Biol. 2011 Feb 25;18(2):177-86

[2]. Shapiro LP, et al. Corticosteroid-induced dendrite loss and behavioral deficiencies can be blocked by activation of Abl2/Arg kinase. Mol Cell Neurosci. 2017 Oct 26;85:226-234.

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