Acalabrutinib

Cat. No.: HY-17600

CAS No.: 1420477-60-6

Molecular Formula: $C_{26}H_{23}N_7O_2$

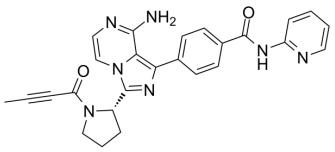
Molecular Weight: 465.51

Target: Btk

Pathway: Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (214.82 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1482 mL	10.7409 mL	21.4818 mL
5 mM	0.4296 mL	2.1482 mL	4.2964 mL
10 mM	0.2148 mL	1.0741 mL	2.1482 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: ≥ 2.08 mg/mL (4.47 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.47 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.47 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Acalabrutinib (ACP-196) is an orally active, irreversible, and highly selective second-generation BTK inhibitor. Acalabrutinib binds covalently to Cys481 in the ATP-binding pocket of BTK. Acalabrutinib demonstrates potent on-target effects and efficacy in mouse models of chronic lymphocytic leukemia (CLL)[1][2].

IC_{50} & Target

IC50: 3 nM (BTK in CD69 B cell)[2]

In Vitro

Acalabrutinib (ACP-196) inhibits tyrosine phosphorylation of downstream targets of ERK, IKB, and AKT, in the in vitro

	signaling assay on primary human CLL cells. In the human CLL NSG xenograft model, Acalabrutinib demonstrates on-target effects including decreased phosphorylation of PLCγ2, ERK and significant inhibition of CLL cell proliferation[1]. Acalabrutinib inhibits purified BTK with an IC_{50} of 3 nM and an EC_{50} of 8 nM in a human whole-blood CD69 B cell activation assay. Acalabrutinib has improved target specificity over ibrutinib with 323-, 94-, 19-, and 9-fold selectivity over the other TEC kinase family members (ITK, TXK, BMX, and TEC , respectively) and no activity against EGFR[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Acalabrutinib (100 mg twice per day) assessed for thrombus formation at injured arterioles of the mice, exhibits more selective for inhibiting BTK and has virtually no inhibition of platelet activity[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2020 Sep 14;5(1):200.
- J Med Chem. 2019 Jul 25;62(14):6561-6574.
- Cancers (Basel). 2020 Dec 11;12(12):3731.
- Stem Cell Reports. 2019 May 14;12(5):996-1006.
- Pharmacol Res. 2020 Jan;151:104512.

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REFERENCES

[1]. Herman SE, et al. The Bruton's tyrosine kinase (BTK) inhibitor acalabrutinib demonstrates potent on-target effects and efficacy in two mouse models of chronic lymphocytic leukemia. Clin Cancer Res. 2016 Nov 30

[2]. Wu J, et al. Acalabrutinib (ACP-196): a selective second-generation BTK inhibitor. J Hematol Oncol. 2016 Mar 9;9:21

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