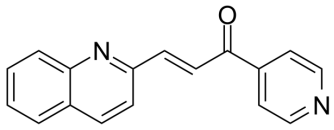
PFK-015

Cat. No.:	HY-12204
CAS No.:	4382-63-2
Molecular Formula:	$C_{17}H_{12}N_2O$
Molecular Weight:	260.29
Target:	Autophagy
Pathway:	Autophagy
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 : 25.2 mg/mL (96.82 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8419 mL	19.2093 mL	38.4187 mL
	5 mM	0.7684 mL	3.8419 mL	7.6837 mL
	10 mM	0.3842 mL	1.9209 mL	3.8419 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 mg/mL (7.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2 mg/mL (7.68 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (7.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PFK-015, a derivative of 3PO, is a specific PFKFB3 inhibitor. PFK-015 inhibits recombinant PFKFB3 with an IC_{50} value of 110 nM and inhibits PFKFB3 activity in cancer cells with an IC_{50} value of 20 nM. PFK-015 can be used for the research of multiple cancers such as lung cancer, stomach cancer, colon cancer and esophageal squamous cell carcinoma (ESCC)[1][2].

IC_{50} & Target

IC50: 110 nM (recombinant PFKFB3); IC50: 20 nM (PFKFB3 activity in cancer cells)[2]

In Vitro

PFK-015 inhibits tumor growth in a dose-dependent manner in esophageal cancer cell line in vitro[1].

	PFK-015 (0-5 μM) increases HIF-1α mediated PD-L1 transcriptional activity[1]. PFK-015 induces the expression of tumor PD-L1 via the phos-PFKFB3/HIF-1a axis[1]. PFK-015 potently inhibits recombinant PFKFB3 with an IC_{50} value of 110 nM and inhibits PFKFB3 activity in cancer cells with an IC_{50} value of 20 nM[2]. PFK-015 inhibits cancer cell proliferation in a panel of 17 cancer cell lines and suppresses glucose uptake in cancer cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PFK-015 impeds ESCC tumor growth in immunodeficient in vivo models[1]. PFK-015 (0-12.5 μM, 48 h) induces tumor PD-L1 expression[1]. PFK-015 (0-12.5 μM, 48 h) can cause a downregulation of immune activity against tumor cells mediated by $CD8^+$ T cells[1]. PFK-015 enhances the efficacy of ESCC by enhancing $CD8^+$ T-cell activity combining PD-1 mAb in immunocompetent mouse models such as C57BL/6 and hu-PBMC-NOG[1]. PFK-015 (iv, 5 mg/kg) has a satisfactory half-life, exposure, tissue distribution and reasonable clearance[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Sep 1;7(1):303.
- Mol Cell Endocrinol. 2023 Oct 9:112083.

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REFERENCES

[1]. Jia Bo Zheng, et al. Glucose metabolism inhibitor PFK-015 combined with immune checkpoint inhibitor is an effective treatment regimen in cancer. Oncoimmunology. 2022 May 25;11(1):2079182.

[2]. Brian Clem, et al. Characterization of a novel small molecule antagonist of 6-phosphofructo-2-kinase that suppresses glucose metabolism and tumor growth. ORAL PRESENTATIONS - PROFFERED ABSTRACTS| APRIL 15 2011.

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