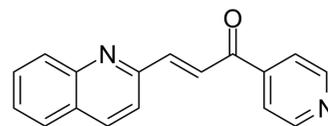


PFK-015

Cat. No.:	HY-12204		
CAS No.:	4382-63-2		
Molecular Formula:	C ₁₇ H ₁₂ N ₂ O		
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)

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2 mg/mL (7.68 mM); Clear solution
- 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
- 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 IC₅₀ value of 110 nM and inhibits PFKFB3 activity in cancer cells with an IC₅₀ 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]}.

IC₅₀ & 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 μ M) increases HIF-1 α mediated PD-L1 transcriptional activity^[1].
PFK-015 induces the expression of tumor PD-L1 via the phos-PFKFB3/HIF-1 α axis^[1].
PFK-015 potently inhibits recombinant PFKFB3 with an IC₅₀ value of 110 nM and inhibits PFKFB3 activity in cancer cells with an IC₅₀ 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 impedes ESCC tumor growth in immunodeficient in vivo models^[1].
PFK-015 (0-12.5 μ M, 48 h) induces tumor PD-L1 expression^[1].
PFK-015 (0-12.5 μ M, 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.

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

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