Fasentin

Cat. No.: HY-101849

CAS No.: 392721-37-8

Molecular Formula: $C_{11}H_9ClF_3NO_2$

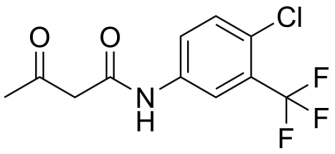
Molecular Weight: 279.64

Target: GLUT; TNF Receptor; Apoptosis

Pathway: Membrane Transporter/Ion Channel; Apoptosis

Storage:

Pure form	-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 (357.60 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5760 mL	17.8801 mL	35.7603 mL
	5 mM	0.7152 mL	3.5760 mL	7.1521 mL
	10 mM	0.3576 mL	1.7880 mL	3.5760 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 (8.94 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (8.94 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fasentin, a potent glucose uptake inhibitor, inhibits GLUT-1/GLUT-4 transporters. Fasentin preferentially inhibits GLUT4 (IC_{50}=68 μM) over GLUT1. Fasentin is a death receptor stimuli (FAS) sensitizer and sensitizes cells to FAS-induced cell death. Fasentin is also a tumor necrosis factor (TNF) apoptosis-inducing ligand sensitizer. Fasentin blocks glucose uptake in cancer cell lines and has anti-angiogenic activity[1][2][3].

IC_{50} & Target		
	GLUT4	GLUT1
	68 μM (IC_{50})	

In Vitro

Fasentin (0.1-1000 μM; 72 hours) inhibits endothelial, tumour and fibroblast cell growth without inducing cell death[1].
Fasentin (25-100 μM; 16-24 hours) induces a cell cycle arrest in G0/G1 phase and reduces the cell number in S phase in a dose-dependent manner[1].
Fasentin (50 μM; 16 hours) alters expression of genes associated with glucose deprivation such as AspSyn and PCK-2[2].
Fasentin (15, 30, 80 μM; pretreatment 1 hour) induces glucose deprivation, partially blocks glucose uptake in PPC-1, DU145, and U937 cells[2].
Fasentin (100 μM; 16 hours) does not affect the migratory capability of endothelial cells[1].
Fasentin (25-100 μM; 16 hours) lowers levels of phospho-ERK in HMECs, indicating a partial inhibition on the ERK signalling pathway, even though the effect is not statistically significant. Fasentin does not inhibit the tyrosine kinase activity of VEGFR2[1].
Fasentin interacts with a unique site in the intracellular channel of GLUT1[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Three types of endothelial cells ECs (HMEC, human microvascular endothelial cells; HUVEC, human umbilical vein endothelial cells; and BAEC, bovine aortic endothelial cells), three human tumour cell lines (MDA-MB-231 and MCF7 breast carcinoma cells, and HeLa cervix adenocarcinoma cells), and human gingival fibroblasts (HGF)
Concentration:	0.1, 1, 10, 100, 1000 μM
Incubation Time:	72 hours
Result:	Inhibited endothelial, tumour and fibroblast cell growth (IC_{50}=26.3-111.2 μM) without inducing cell death.

Cell Cycle Analysis[1]

Cell Line:	HMECs
Concentration:	25, 50, 100 μM
Incubation Time:	16, 24 hours
Result:	Induced a cell cycle arrest in G0/G1 phase and reduced the cell number in S phase in a dose-dependent manner. Did not increase the subG1 population.

RT-PCR[2]

Cell Line:	PPC-1 cells[2]
Concentration:	50 μM
Incubation Time:	16 hours
Result:	Altered expression of genes associated with glucose deprivation such as AspSyn and PCK-2 not FLIP mRNA expression.

CUSTOMER VALIDATION

- Cell Metab. 2022 Nov 11;S1550-4131(22)00490-9.
- Cell Death Dis. 2022 Mar 11;13(3):229.
- Mbio. 2023 Oct 5:e0211023.

- ACS Chem Biol. 2023 Apr 28.

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REFERENCES

[1]. Mª Carmen Ocaña, et al. Fasentin diminishes endothelial cell proliferation, differentiation and invasion in a glucose metabolism-independent manner. Sci Rep. 2020 Apr 9;10(1):6132.

[2]. Tabitha E Wood, et al. A novel inhibitor of glucose uptake sensitizes cells to FAS-induced cell death. Mol Cancer Ther. 2008 Nov;7(11):3546-55.

[3]. Qin Wu, et al. GLUT1 inhibition blocks growth of RB1-positive triple negative breast cancer. Nat Commun. 2020 Aug 21;11(1):4205.

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

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