BMS-536924

Cat. No.: HY-10262

CAS No.: 468740-43-4

Molecular Formula: $C_{25}H_{26}ClN_5O_3$

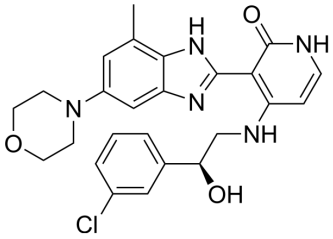
Molecular Weight: 479.96

Target: IGF-1R; Insulin Receptor; Apoptosis

Pathway: Protein Tyrosine Kinase/RTK; Apoptosis

Storage:

Powder	-20°C	3 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (20.84 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0835 mL	10.4175 mL	20.8351 mL
	5 mM	0.4167 mL	2.0835 mL	4.1670 mL
	10 mM	0.2084 mL	1.0418 mL	2.0835 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: ≥ 3.75 mg/mL (7.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.21 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.25 mg/mL (4.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-536924 is an orally active, competitive and selective insulin-like growth factor receptor (IGF-1R) kinase and insulin receptor (IR) inhibitor with IC_{50}s of 100 nM and 73 nM, respectively. BMS-536924 has anti-cancer activity[1][2].

IC_{50} & Target

IC50: 100 nM (IGF-1R) and 73 nM (IR)[1][2]

In Vitro

BMS-536924 inhibits FAK and Lck with IC_{50}s of 150 nM and 341 nM, respectively[1].
BMS-536924 (1 μM; every four days for 12 days) blocks pBabe-MCF10A and CD8-IGF-IR-MCF10A acinar proliferation[2].
BMS-536924 (0.01-1 μM; 24 hours) inhibits growth of CD8-IGF-IR-MCF10A cells and has an IC_{50} of 0.48 μM[2].

BMS-536924 (1 μM; for 4 days) induces apoptosis in CD8-IGF-IR-MCF10A acini[2].
BMS-536924 (0.1-1 μM; for 24 hours) decreases in S-phase cells and causes a G0/G1 block[2].
BMS-536924 (1 μM; 10 min, 1, 8, 24, 48 hours) inhibits IGF-IR signaling in pBabe-MCF10A cells and inhibits phosphorylation of CD8-IGF-IR. BMS-536924 time-dependently inhibits AKT phosphorylation[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	pBabe-MCF10A and CD8-IGF-IR-MCF10A acini
Concentration:	1 μM
Incubation Time:	Every four days for 12 days
Result:	Blocked acinar proliferation.

Cell Viability Assay[2]

Cell Line:	CD8-IGF-IR-MCF10A cells
Concentration:	0.01, 0.1, 1 μM
Incubation Time:	24 hours
Result:	Has an IC50 of 0.48 μM.

Apoptosis Analysis[2]

Cell Line:	CD8-IGF-IR-MCF10A acini
Concentration:	1 μM
Incubation Time:	For 4 days
Result:	Resulted in a dramatic induction of apoptosis.

Cell Cycle Analysis[2]

Cell Line:	CD8-IGF-IR-MCF10A cells and pBabe-MCF10A control cells
Concentration:	0.1, 0.5, 1 μM
Incubation Time:	For 24 hours
Result:	Decreased in S-phase cells and caused a G0/G1 block.

Western Blot Analysis[2]

Cell Line:	CD8-IGF-IR-MCF10A cells
Concentration:	1 μM
Incubation Time:	10 min, 1, 8, 24, 48 hours
Result:	Caused maximal inhibition of phosphorylated IGF-IR at 10 min and retained its ability to inhibit IGF-IR phosphorylation for up to 48 hours.

In Vivo

BMS-536924 (100 mg/kg; gavage; daily; for 35 days) causes regression of xenografts in vivo and an average reduction of 76% tumor volume after 2 weeks[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Athymic nude mice with CD8-IGF-IRMCF10A cells[2]
Dosage:	100 mg/kg
Administration:	Gavage; daily; for 35 days
Result:	Caused an average reduction of 76% tumor volume after 2 weeks.

CUSTOMER VALIDATION

- Cell Discov. 2023 Mar 7;9(1):26.
- Nat Commun. 2023 Jun 15;14(1):3560.
- Gen Comp Endocrinol. 2019 Sep 15;281:83-90.

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REFERENCES

[1]. Wittman M, et al. Discovery of a (1H-benzoimidazol-2-yl)-1H-pyridin-2-one (BMS-536924) inhibitor of insulin-like growth factor I receptor kinase with in vivo antitumor activity. J Med Chem. 2005 Sep 8;48(18):5639-43.

[2]. Litzenburger BC, et al. BMS-536924 reverses IGF-IR-induced transformation of mammary epithelial cells and causes growth inhibition and polarization of MCF7 cells. Clin Cancer Res. 2009 Jan 1;15(1):226-37.

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