SZABO SCANDIC

Part of Europa Biosite

Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

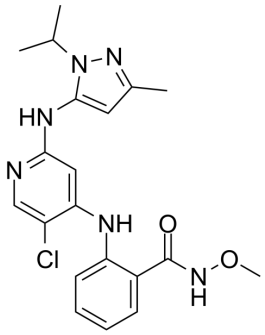
mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

GSK2256098

Cat. No.:	HY-100498
CAS No.:	1224887-10-8
Molecular Formula:	$C_{20}H_{23}ClN_6O_2$
Molecular Weight:	414.89
Target:	FAK; Apoptosis
Pathway:	Protein Tyrosine Kinase/RTK; Apoptosis
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 : ≥ 30 mg/mL (72.31 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4103 mL	12.0514 mL	24.1028 mL
5 mM	0.4821 mL	2.4103 mL	4.8206 mL
10 mM	0.2410 mL	1.2051 mL	2.4103 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 (6.03 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.03 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GSK2256098 is a selective FAK kinase inhibitor, which inhibits growth and survival of pancreatic ductal adenocarcinoma cells.

IC_{50} & Target

FAK[1]

In Vitro

GSK2256098 is a thousand fold more selective for FAK compared to the nearest FAK family member, Pyk2. GSK2256098 inhibits FAK activity through targeting the phosphorylation site of FAK, tyrosine (Y) 397. GSK2256098 inhibits FAK activity or Y397 phosphorylation in cancer cell lines, OVCAR8 (ovary), U87MG (brain), and A549 (lung), at IC_{50}s of 15, 8.5 and 12 nM, respectively. The responses of 6 PDAC cell lines in regards to FAK Y397 phosphorylation or activity to GSK2256098

	treatments (0.1–10 μM) ranged from low (less than 20% inhibition) to high (more than 90% inhibition). The least and most sensitive cell lines (PANC-1 and L3.6P1) are selected for further analysis. GSK2256098 inhibition of FAK Y397 phosphorylation correlated with decreased levels of phosphorylated Akt and ERK in L3.6P1 cells. GSK2256098 decreases cell viability, anchorage-independent growth, and motility in a dose dependent manner[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	FAK is well-known to play an important role in angiogenesis, proliferation, and apoptosis, so the tumor samples harvested from the therapy experiments are examined. Evaluating CD31, significantly lower microvessel densities in tumors from mice treated with GSK2256098 and Paclitaxel is observed than in tumors from mice in the vehicle control group ($P<0.05$). This is consistent across both models, but Ishikawa tumors had the lowest microvessel density. All tumor models in mice treated with GSK2256098 exhibit less proliferation via Ki67 than control. Ishikawa tumors have the lowest Ki67 expression in response to therapy. Ishikawa tumors have higher apoptotic indices than Hec1A tumors after treatment with GSK2256098. Significant rates of apoptosis are seen in all models that had been treated with combination GSK2256098 and Paclitaxel[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	PDAC cells are cultured on the wells of a 96-well plate. Ten microliters of MTS is added to the wells (total value: 100 μL). After the plate is kept in a 37°C incubator for 10-30 min, the absorbance at 450 nm wave length of reacted MTS is determined on a microplate reader. The Sigma plot program is used to calculate IC_{50} of GSK2256098 on cell viability. PDAC cells are cultured on a 6-well plate. When cell confluence reached about 70% in regular medium, the cells are incubated in the medium containing 0.1-10 μM GSK2256098 for 48 or 72 hr. At the end of treatments, cells are re-seeded and kept for 9 d Then, the cells are stained using Clonogenic Reagent, and the blue colonies are counted[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Female 8- to 12-week-old athymic nude mice are used. For therapeutic experiments, 4×10^6 Ishikawa or Hec1A cells are inoculated into the uterine horn. Following tumor cell injection, the mice are randomized (n=10 mice per group) according to the following groups: 1) 100 μL of a vehicle control (oral, daily); 2) 75 mg/kg GSK2256098 in 100 μL of vehicle (oral, daily); 3) 2.5 mg/kg Paclitaxel in 200 μL of PBS (intraperitoneal, weekly); and 4) GSK2256098 and Paclitaxel (doses and frequencies given above). Therapy is initiated 10-14 days after tumor injection. The mice are monitored for adverse effects and sacrificed using cervical dislocation four to six weeks after initiation of treatment. At the completion of each experiment, each mouse's weight, aggregate tumor weight, location, and number of tumor nodules are recorded for each treatment group. Tumor samples are processed for further analysis via preservation in optimal cutting temperature medium for frozen section analysis as well as fixed in formalin for paraffin-embedded section analysis. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- JCI Insight. 2020 Feb 13;5(3):e133232.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Zhang J, et al. A small molecule FAK kinase inhibitor, GSK2256098, inhibits growth and survival of pancreatic ductal adenocarcinoma cells. Cell Cycle. 2014;13(19):3143-9.

[2]. Thanapprapasr D, et al. PTEN Expression as a Predictor of Response to Focal Adhesion Kinase Inhibition in Uterine Cancer. Mol Cancer Ther. 2015 Jun;14(6):1466-75.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA