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Produktinformation

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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

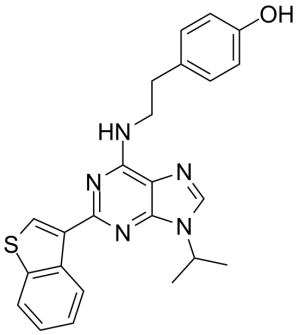
mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Stemregenin 1

Cat. No.:	HY-15001
CAS No.:	1227633-49-9
Molecular Formula:	$C_{24}H_{23}N_5OS$
Molecular Weight:	429.54
Target:	Aryl Hydrocarbon Receptor
Pathway:	Immunology/Inflammation
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 : 62.5 mg/mL (145.50 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3281 mL	11.6404 mL	23.2807 mL
	5 mM	0.4656 mL	2.3281 mL	4.6561 mL
	10 mM	0.2328 mL	1.1640 mL	2.3281 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 (5.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.82 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Stemregenin 1 is a potent aryl hydrocarbon receptor (AhR) antagonist with IC_{50} of 127 nM.

IC_{50} & Target

IC50: 127 nM (AhR)[1]

In Vitro

Stemregenin 1 (SR1) acts by antagonizing the aryl hydrocarbon receptor (AhR). Stemregenin 1 increases the number of $CD34^+$ cells after 5 to 7 days with an EC_{50} of ~120 nM. Stemregenin 1 inhibits photoaffinity ligand (PAL) binding (IC_{50}=40 nM) These results support the conclusion that Stemregenin 1 -induced $CD34^+$ cell expansion is mediated through direct binding and

inhibition of the AhR[1]. An aryl hydrocarbon receptor antagonist, Stemregenin 1 (SR1), robustly promotes ex vivo expansion of human $CD34^+$ cells. Stemregenin 1 treatment accelerates the proliferation of $CD34^+$ cells and decreases the expression levels of VentX[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]

A quantity of 250,000 CB-derived $CD34^+$ cells are cultured with control conditions (DMSO, 0.01%) or StemRegenin 1 (0.75 μM) for 3 weeks. At this point control cultures had expanded 1080-fold and StemRegenin 1 treated cells expanded 2024-fold relative to starting cell numbers. A quantity of 30 to 30,000 uncultured $CD34^+$ CB-derived cells or a fraction of the final culture equivalent to 30 to 10,000 starting cells are transplanted. The cells are injected intravenously via the retro-orbital route into sub-lethally irradiated (300 rads, 200 rads) 6- to 10-week-old NSG mice. Engraftment is performed within 24 h after irradiation. Engraftment is monitored by flow cytometric analysis of blood obtained via retro-orbital sinus or bone marrow using anti-human CD45 and anti-mouse CD45 antibodies. The mice are sacrificed between 13-16 weeks posttransplantation; bone marrow (from both femurs and tibiae), spleen and thymus are collected for analysis. For secondary engraftment, 50% of the bone marrow from each recipient mouse is transplanted into one secondary sub-lethally irradiated NSG mouse. Fifteen weeks after transplantation, bone marrow is harvested from the secondary mice and analyzed by flow cytometry[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv. 2022 Mar 18;8(11):eabf8627.
- J Hazard Mater. 2020 Mar 5;385:121521.
- Theranostics. 2021; 11(18):8797-8812.
- Int J Biol Macromol. 2023 Sep 15;126920.
- Ecotoxicol Environ Saf. 2023 Dec 5:269:115782.

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REFERENCES

[1]. Boitano AE, et al. Aryl Hydrocarbon Receptor Antagonists Promote the Expansion of Human Hematopoietic Stem Cells. Science. 2010 Sep 10;329(5997):1345-8.

[2]. Gao H, et al. Suppression of homeobox transcription factor VentX promotes expansion of human hematopoietic stem/multipotent progenitor cells. J Biol Chem. 2012 Aug 24;287(35):29979-87.

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