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Zuschläge

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SZABO-SCANDIC Handels GmbH

Quellenstraße 110, A-1100 Wien

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

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

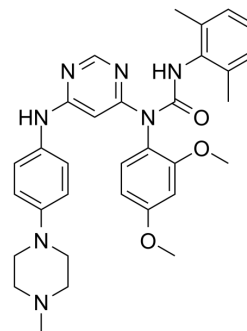
mail@szabo-scandic.com

www.szabo-scandic.com

[linkedin.com/company/szaboscandic](https://www.linkedin.com/company/szaboscandic) 

HG-9-91-01

Cat. No.:	HY-15776
CAS No.:	1456858-58-4
Molecular Formula:	C ₃₂ H ₃₇ N ₇ O ₃
Molecular Weight:	567.68
Target:	Salt-inducible Kinase (SIK)
Pathway:	Immunology/Inflammation
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 150 mg/mL (264.23 mM)

* "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		1.7616 mL	8.8078 mL	17.6156 mL
	5 mM		0.3523 mL	1.7616 mL	3.5231 mL
	10 mM		0.1762 mL	0.8808 mL	1.7616 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (4.40 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.08 mg/mL (3.66 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.08 mg/mL (3.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HG-9-91-01 is a potent and highly selective salt-inducible kinase (SIK) inhibitor with IC₅₀s of 0.92 nM, 6.6 nM and 9.6 nM for SIK1, SIK2 and SIK3 respectively.

IC₅₀ & Target

IC₅₀: 0.92/6.6/9.6 nM (SIK1/2/3)^[1]

In Vitro

HG-9-91-01 inhibits a number of protein tyrosine kinases that possess a threonine residue at the gatekeeper site, such as Src

family members (Src, Lck, and Yes), BTK, and the FGF and Ephrin receptors^[1]. HG-9-91-01 demonstrates a strong correlation between the potency of SIK2 inhibition and enhanced IL-10 production. In agreement with these reports, pretreating BMDCs with HG-9-91-01, a recently described inhibitor of SIK1-3, along with several other kinases, results in concentration-dependent potentiation of zymosan-induced IL-10 production with an EC₅₀ ~200 nM and a maximum effect similar to that observed with PGE₂^[2]. HG-9-91-01 has more than a 100-fold greater potency against SIKs than AMPK (IC₅₀=4.5 μM) in a cell-free assay. HG-9-91-01 treatment dose dependently increased mRNA expression of Pck1 and G6pc and that effect is similar in cells treated with 4 μM HG-9-91-01. Consistent with this observation, there is also a dose-dependent increase in glucose production following HG-9-91-01 treatment^[3].

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

PROTOCOL

Cell Assay ^[2]

Bone marrow is harvested from femurs and tibias of C57BL/6 mice. Bone-marrow-derived dendritic cells (BMDCs) are differentiated DMEM. Cultures are differentiated for 7 d and routinely analyzed for >90% CD11c (allophycocyanin (APC) anti-CD11c clone HL3) positivity by flow cytometry before use in experiments. Lentiviral transduction of bone marrow cultures is conducted by addition of 293T culture supernatants containing lentiviral particles encoding the CREB-dependent luciferase reporter construct or CRT3 targeting or control shRNAs 1 d postisolation. Stable integration of lentiviral shRNA constructs is selected by addition of puromycin (3 μg/mL) on day 4 posttransduction. After 2 d, stably transduced BMDCs are released from selection and used in subsequent assays. Unless otherwise indicated, cells are treated for 2 d with PGE₂ (5 μM) or HG-9-91-01 (0.5 μM) or an equivalent concentration of DMSO (≤0.5%) and then stimulated for 18 h with LPS (100 ng/mL), R848 (10 μg/mL), or Zymosan (4 μg/mL)^[2].

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

CUSTOMER VALIDATION

- Nat Cancer. 2023 May;4(5):754-773.
- Nature Metabolism. 2021 May;3(5):682-700.
- Mol Cell. 2023 May 2;S1097-2765(23)00288-5.
- Cell Death Dis. 2022 Feb 25;13(2):188.
- Cell Death Dis. 2020 Jan 13;11(1):25.

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REFERENCES

[1]. Clark K, et al. Phosphorylation of CRT3 by the salt-inducible kinases controls the interconversion of classically activated and regulatory macrophages. Proc Natl Acad Sci U S A. 2012 Oct 16;109(42):16986-91.

[2]. Sundberg TB, et al. Small-molecule screening identifies inhibition of salt-inducible kinases as a therapeutic strategy to enhance immunoregulatory functions of dendritic cells. Proc Natl Acad Sci U S A. 2014 Aug 26;111(34):12468-73.

[3]. Patel K, et al. The LKB1-salt-inducible kinase pathway functions as a key gluconeogenic suppressor in the liver. Nat Commun. 2014 Aug 4;5:4535.

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

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