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Zuschläge

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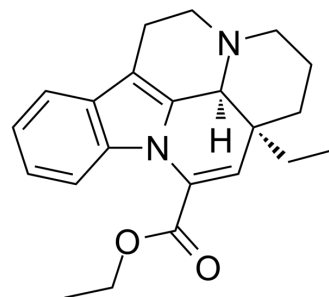
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Vinpocetine

Cat. No.:	HY-13295		
CAS No.:	42971-09-5		
Molecular Formula:	C ₂₂ H ₂₆ N ₂ O ₂		
Molecular Weight:	350.45		
Target:	Sodium Channel; IKK; Phosphodiesterase (PDE)		
Pathway:	Membrane Transporter/Ion Channel; NF-κB; Metabolic Enzyme/Protease		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 14.29 mg/mL (40.78 mM; Need ultrasonic)
DMSO : 6.25 mg/mL (17.83 mM; Need ultrasonic)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		2.8535 mL	14.2674 mL	28.5347 mL
	5 mM		0.5707 mL	2.8535 mL	5.7069 mL
	10 mM		0.2853 mL	1.4267 mL	2.8535 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 0.62 mg/mL (1.77 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 0.62 mg/mL (1.77 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 0.62 mg/mL (1.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vinpocetine (Ethyl apovincamate) is a derivative of the alkaloid Vincamine that blocks voltage-gated Na⁺ channels. The IC₅₀ value of Vinpocetine on direct IKK inhibition in the cell-free system is 17.17 μM. Vinpocetine is a phosphodiesterase (PDE) inhibitor and inhibits NF-κB-dependent inflammatory responses by directly targeting IκB kinase complex (IKK), and has been widely used for the treatment of cerebrovascular disorders^{[1][2][3]}.

IC₅₀ & Target

IKK

	17.17 μ M (IC ₅₀)								
In Vitro	Vinpocetine (5-50 μM; 7 hours; VSMCs, HUVECs, A549 cells and RAW264.7 cells) potently inhibits TNF-α-induced NF-κB-dependent transcriptional activity in a dose-dependent manner with an approximate IC₅₀ value of 25 μM. Vinpocetine do not have a significant effect on cell viability^[1]. Vinpocetine (50 μM; 7 hours; VSMCs, HUVECs, A549 cells and RAW264.7 cells) potently inhibits TNF-α-induced up-regulation of TNF-α, IL-1β, IL-8, MCP-1, VCAM-1, ICAM-1 and MIP-2 transcripts in several cell types^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. RT-PCR^[1] <table> <tr> <td>Cell Line:</td><td>VSMCs, HUVECs, A549 cells and RAW264.7 cells</td></tr> <tr> <td>Concentration:</td><td>50 μM</td></tr> <tr> <td>Incubation Time:</td><td>7 hours</td></tr> <tr> <td>Result:</td><td>Inhibited TNF-α-induced up-regulation of TNF-α, IL-1β, IL-8, MCP-1, VCAM-1, ICAM-1 and MIP-2 transcripts in several cell types.</td></tr> </table>	Cell Line:	VSMCs, HUVECs, A549 cells and RAW264.7 cells	Concentration:	50 μ M	Incubation Time:	7 hours	Result:	Inhibited TNF- α -induced up-regulation of TNF- α , IL-1 β , IL-8, MCP-1, VCAM-1, ICAM-1 and MIP-2 transcripts in several cell types.
Cell Line:	VSMCs, HUVECs, A549 cells and RAW264.7 cells								
Concentration:	50 μ M								
Incubation Time:	7 hours								
Result:	Inhibited TNF- α -induced up-regulation of TNF- α , IL-1 β , IL-8, MCP-1, VCAM-1, ICAM-1 and MIP-2 transcripts in several cell types.								
In Vivo	Vinpocetine (2.5-10 mg/kg; intraperitoneal injection; C57BL/6 mice) potently inhibits TNF-α- or LPS-induced up-regulation of proinflammatory mediators, including TNF-α, IL-1β, and MIP-2, and decreases interstitial infiltration of polymorphonuclear leukocytes in a mouse model of TNF-α- or LPS-induced lung inflammation^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. <table> <tr> <td>Animal Model:</td><td>C57BL/6 mice (8 weeks of age)^[1]</td></tr> <tr> <td>Dosage:</td><td>2.5 mg/kg, 5 mg/kg, and 10 mg/kg</td></tr> <tr> <td>Administration:</td><td>Intraperitoneal injection</td></tr> <tr> <td>Result:</td><td>Inhibited TNF-α- or LPS-induced up-regulation of proinflammatory mediators, including TNF-α, IL-1β, and MIP-2, and decreased interstitial infiltration of polymorphonuclear leukocytes in a mouse model of TNF-α- or LPS-induced lung inflammation.</td></tr> </table>	Animal Model:	C57BL/6 mice (8 weeks of age) ^[1]	Dosage:	2.5 mg/kg, 5 mg/kg, and 10 mg/kg	Administration:	Intraperitoneal injection	Result:	Inhibited TNF- α - or LPS-induced up-regulation of proinflammatory mediators, including TNF- α , IL-1 β , and MIP-2, and decreased interstitial infiltration of polymorphonuclear leukocytes in a mouse model of TNF- α - or LPS-induced lung inflammation.
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Dosage:	2.5 mg/kg, 5 mg/kg, and 10 mg/kg								
Administration:	Intraperitoneal injection								
Result:	Inhibited TNF- α - or LPS-induced up-regulation of proinflammatory mediators, including TNF- α , IL-1 β , and MIP-2, and decreased interstitial infiltration of polymorphonuclear leukocytes in a mouse model of TNF- α - or LPS-induced lung inflammation.								

CUSTOMER VALIDATION

- Nat Commun. 2023 Jun 3;14(1):3224.
- Front Cell Dev Biol. 2021 Feb 4.
- J Cancer Res Clin Oncol. 2023 Mar 31.
- Neuroscience. 2023 Jun 6;S0306-4522(23)00241-5.
- J Investig Med. 2022 May 17;jim-2022-002369.

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REFERENCES

[1]. Kye-Im Jeon et al. Vinpocetine inhibits NF- κ B-dependent inflammation via an IKK-dependent but PDE-independent mechanism PNAS May 25, 2010 vol. 107 no. 21 9795-9800

[2]. Patyar S, et al. Role of vinpocetine in cerebrovascular diseases. Pharmacol Rep. 2011;63(3):618-28.

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

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