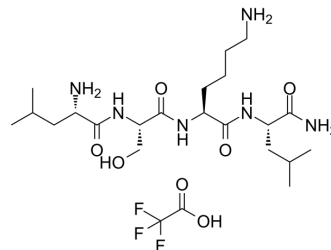


LSKL, Inhibitor of Thrombospondin (TSP-1) (TFA)

Cat. No.:	HY-P0299A
Molecular Formula:	C ₂₃ H ₄₃ F ₃ N ₆ O ₇
Molecular Weight:	572.62
Sequence:	Leu-Ser-Lys-Leu-NH ₂
Sequence Shortening:	LSKL-NH ₂
Target:	TGF-β Receptor
Pathway:	TGF-beta/Smad
Storage:	Sealed storage, away from moisture
	Powder -80°C 2 years
	-20°C 1 year



* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (436.59 mM; Need ultrasonic)
 H₂O : 100 mg/mL (174.64 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.7464 mL	8.7318 mL	17.4636 mL
	5 mM	0.3493 mL	1.7464 mL	3.4927 mL
	10 mM	0.1746 mL	0.8732 mL	1.7464 mL

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

In Vivo

1. Add each solvent one by one: PBS
 Solubility: 100 mg/mL (174.64 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
 Solubility: ≥ 2.08 mg/mL (3.63 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
 Solubility: ≥ 2.08 mg/mL (3.63 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

LSKL, Inhibitor of Thrombospondin (TSP-1) TFA is a latency-associated protein (LAP)-TGFβ derived tetrapeptide and a competitive TGF-β1 antagonist. LSKL, Inhibitor of Thrombospondin (TSP-1) TFA inhibits the binding of TSP-1 to LAP and alleviates renal interstitial fibrosis and hepatic fibrosis. LSKL, Inhibitor of Thrombospondin (TSP-1) TFA suppresses subarachnoid fibrosis via inhibition of TSP-1-mediated TGF-β1 activity, prevents the development of chronic hydrocephalus and improves long-term neurocognitive defects following subarachnoid hemorrhage (SAH). LSKL, Inhibitor of

	Thrombospondin (TSP-1) TFA can readily cross the blood-brain barrier ^{[1][2]} .								
IC₅₀ & Target	TGF-β1 ^[1]								
In Vitro	<p>The KTRF sequence from ADAMTS1 is responsible for the interaction with the LSKL, Inhibitor of Thrombospondin (TSP-1) (LSKL peptide) from the latent form of TGF-β, leading to its activation. There is a stable binding mode between LSKL, Inhibitor of Thrombospondin (TSP-1) and ADAMTS1 KTRF sequence, characterized by 3 salt bridges and 2 hydrogen bonds^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>								
In Vivo	<p>LSKL, Inhibitor of Thrombospondin (TSP-1) (1 mg/kg; intraperitoneal injection; male Sprague-Dawley rats) is protective against subarachnoid fibrosis, attenuates ventriculomegaly and effectively suppresses hydrocephalus. LSKL, Inhibitor of Thrombospondin (TSP-1) treatment inhibits TGF-β1 activity and subsequent Smad2/3 signaling^[1].</p> <p>?LSKL, Inhibitor of Thrombospondin (TSP-1) (30 mg/kg, i.p.) successfully inhibits transforming growth factor (TGF) β-Smad signal activation induced by partial hepatectomy. LSKL, Inhibitor of Thrombospondin (TSP-1) successfully attenuates TGF-β-Smad signal activation by antagonizing TSP-1, but not by reducing TSP-1 protein expression. LSKL, Inhibitor of Thrombospondin (TSP-1) accelerates hepatocyte proliferation after hepatectomy^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>103 male Sprague-Dawley rats (6 weeks of age; 160-180 g) with subarachnoid hemorrhage (SAH)^[1]</td> </tr> <tr> <td>Dosage:</td> <td>1 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection</td> </tr> <tr> <td>Result:</td> <td>Was protective against subarachnoid fibrosis, attenuated ventriculomegaly and effectively suppressed hydrocephalus.</td> </tr> </table>	Animal Model:	103 male Sprague-Dawley rats (6 weeks of age; 160-180 g) with subarachnoid hemorrhage (SAH) ^[1]	Dosage:	1 mg/kg	Administration:	Intraperitoneal injection	Result:	Was protective against subarachnoid fibrosis, attenuated ventriculomegaly and effectively suppressed hydrocephalus.
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Administration:	Intraperitoneal injection								
Result:	Was protective against subarachnoid fibrosis, attenuated ventriculomegaly and effectively suppressed hydrocephalus.								

CUSTOMER VALIDATION

- J Exp Clin Cancer Res. 2022 Aug 26;41(1):259.
- Cell Death Dis. 2022 Jul 30;13(7):663.
- Environ Pollut. 2020 Apr;259:113915.
- Front Immunol. 2022 Mar 18;13:853894.
- Front Cell Dev Biol. 18 February 2021.

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REFERENCES

- [1]. Liao F, et al. LSKL peptide alleviates subarachnoid fibrosis and hydrocephalus by inhibiting TSP1-mediated TGF-β1 signaling activity following subarachnoid hemorrhage in rats. *Exp Ther Med*. 2016 Oct;12(4):2537-2543. Epub 2016 Aug 31.
- [2]. Laurent MA, et al. In silico characterization of the interaction between LSKL peptide, a LAP-TGF-beta derived peptide, and ADAMTS1. *Comput Biol Chem*. 2016 Apr;61:155-61.
- [3]. Kuroki H, et al. Effect of LSKL peptide on thrombospondin 1-mediated transforming growth factor β signal activation and liver regeneration after hepatectomy in an experimental model. *Br J Surg*. 2015 Jun;102(7):813-25.

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

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