H-Leu-Ser-Lys-Leu-OH

Cat. No.:	HY-P3971	
CAS No.:	162559-45-7	$\begin{array}{c} \begin{array}{c} & NH_2 \\ & NH_2 \\ & H_2 \\ & H_2$
Molecular Formula:	C ₂₁ H ₄₁ N ₅ O ₆	
Molecular Weight:	459.58	
Sequence Shortening:	LSKL	
Target:	TGF-beta/Smad	
Pathway:	Stem Cell/Wnt; TGF-beta/Smad	
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.	

Description	H-Leu-Ser-Lys-Leu-OH (LSYL) is a latency-associated peptide at the amino terminus of LAP, with inhibitory effect on TGF-β1 activation. H-Leu-Ser-Lys-Leu-OH, binding with <u>KRFK</u> (HY-P3970), can block the signal transduction of TGF-β1, and prevent the progression of hepatic damage and fibrosis ^[1] .	
IC ₅₀ & Target	TGF-β1 ^[1]	
In Vitro	H-Leu-Ser-Lys-Leu-OH (LSKL) also blocks the activation of TGF-b1 by TSP-1, and decreases the activation of TGF-b1 by the KRFK peptide ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
In Vivo	H-Leu-Ser-Lys-Leu-OH (LSKL) (100 μg/0.5 mL; i.p.; daily for 4 weeks, accompanied with DMN) protects dimethylnitrosamine (DMN)-treated (10 mg/kg; i.p.; 3 consecutive days a week for 4 weeks) rats from liver atrophy. H-Leu-Ser-Lys-Leu-OH also prevents the progression of hepatic damage and fibrosis ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	

REFERENCES

[1]. Kondou H, et al. A blocking peptide for transforming growth factor-beta1 activation prevents hepatic fibrosis in vivo. J Hepatol. 2003 Nov;39(5):742-8.



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