Endothelin-1 (1-31) (Human)

Cat. No.:	HY-P4159
CAS No.:	133972-52-8
Molecular Formula:	$C_{162}H_{236}N_{38}O_{47}S_5$
Molecular Weight:	3628.16
Sequence:	Cys-Ser-Cys-Ser-Ser-Leu-Met-Asp-Lys-Glu-Cys-Val-Tyr-Phe-Cys-His-Leu-Asp-Ile-Ile-Trp-Val-Asn-Thr-Pro-Glu-His-Val-Val-Pro-Tyr (Disulfide bridge:Cys1-Cys15;Cys3-Cys11)
Sequence Shortening:	CSCSSLMDKECVYFCHLDIIWVNTPEHVVPY (Disulfide bridge:Cys1-Cys15;Cys3-Cys11)
Target:	ERK
Pathway:	MAPK/ERK Pathway; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Endothelin-1 (1-31) (Human) is a potent vasoconstrictor and hypertensive agent. Endothelin-1 (1-31) (Human) is derived from the selective hydrolysis of big ET-1 by chymase[1].

In Vitro

Endothelin-1 (1-31) (Human) (100 pM-100 nM; 24 h) induces human mesangial cells proliferation[2].
Endothelin-1 (1-31) (Human) (100 nM; 0-10 min) induces ERK activation in human mesangial cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	Human mesangial cells
Concentration:	100 pM-100 nM
Incubation Time:	24 h
Result:	Caused an increase in [^{3}H]-thymidine incorporation into the cells in a concentration-dependent manner.

Western Blot Analysis[2]

Cell Line:	Human mesangial cells
Concentration:	100 nM
Incubation Time:	0, 5, 10, 15 and 30 min
Result:	ERK activities rapidly increased 2.45-fold at 5 min and peaked at 10 min. The activities of both ERKs rapidly declined, returning to the baseline control value 30 min after stimulation.

In Vivo

ET-1 (1-31) (100 nM; single dose) induces contraction in the mouse mesenteric artery. The contraction may be mediated by the ET_A receptor and may be increased by aging. A clear difference exists between males and females in the present chronic

diabetic condition[1].

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Animal Model:	ICR mice, Streptozocin (HY-13753)-induced diabetic model[1]
Dosage:	100 nM
Administration:	In the organ bath, single dose
Result:	In the 1-week control (but not diabetic) group, induced contraction and the contractile response was significantly greater in female mice than in male mice, and there was no significant difference in either male or female mice between the age-matched controls and the diabetic mice. In the 8-weeks group, the contraction was or tended to be increased compared with the corresponding 1-week group in all mice. Although in male mice this contraction was not different between control and diabetic groups, it was significantly greater in diabetic female mice than in the control female mice and in female diabetic mice than in male diabetic mice. The contraction was inhibited by ET_A receptor inhibitor.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2024 Mar;45(3):545-557.

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REFERENCES

[1]. Matsumoto T, et al. Gender differences in vascular reactivity to endothelin-1 (1-31) in mesenteric arteries from diabetic mice. Peptides. 2008 Aug;29(8):1338-46.

[2]. Yoshizumi M, et al. Effect of endothelin-1 (1-31) on human mesangial cell proliferation. Jpn J Pharmacol. 2000 Oct;84(2):146-55.

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