Endothelin-1 (1-31) (Human) TFA

Cat. No.: HY-P4159A

Molecular Formula: $C_{164}H_{237}F_3N_{38}O_{49}S_5$

Molecular Weight: 3742.18

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: Sealed storage, away from moisture and light, under nitrogen

Powder	-80°C	2 years
	-20°C	1 year

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

BIOLOGICAL ACTIVITY

Description

Endothelin-1 (1-31) (Human) TFA is a potent vasoconstrictor and hypertensive agent. Endothelin-1 (1-31) (Human) TFA 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) TFA induces human mesangial cells proliferation[2].
Endothelin-1 (1-31) (Human) (100 nM; 0-10 min) TFA 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) TFA 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.

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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