

Endothelin-1 (1-31) (Human) acetate

Cat. No.:	HY-P4159B
Molecular Formula:	C ₁₆₄ H ₂₄₀ N ₃₈ O ₄₉ S ₅
Molecular Weight:	3688.21
Sequence:	Cys-Ser-Cys-Ser-Ser-Leu-Met-Asp-Lys-Glu-Cys-Val-Tyr-Phe-Cys-His-Leu-Asp-Ile-Ile-Tr p-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) acetate is a potent vasoconstrictor and hypertensive agent. Endothelin-1 (1-31) (Human) acetate is derived from the selective hydrolysis of big ET-1 by chymase ^[1] .																
In Vitro	<p>Endothelin-1 (1-31) (Human) (100 pM-100 nM; 24 h) acetate induces human mesangial cells proliferation^[2]. Endothelin-1 (1-31) (Human) (100 nM; 0-10 min) acetate induces ERK activation in human mesangial cells^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Proliferation Assay^[2]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Human mesangial cells</td> </tr> <tr> <td>Concentration:</td> <td>100 pM-100 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>Caused an increase in [³H]-thymidine incorporation into the cells in a concentration-dependent manner.</td> </tr> </table> <p>Western Blot Analysis^[2]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Human mesangial cells</td> </tr> <tr> <td>Concentration:</td> <td>100 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>0, 5, 10, 15 and 30 min</td> </tr> <tr> <td>Result:</td> <td>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.</td> </tr> </table>	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.	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.
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In Vivo	<p>ET-1 (1-31) (100 nM; single dose) acetate 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>																

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.

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

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