Lixisenatide

Cat. No.: HY-P0119

CAS No.: 320367-13-3

Molecular Formula: $C_{215}H_{347}N_{61}O_{65}S$

Molecular Weight: 4858.49

HGEGTFTSDLSKQMEEEAVRLFIEWLKNGGPSSGAPPSKKKKKK-NH_2

Sequence: His-Gly-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Leu-Ser-Lys-Gln-Met-Glu-Glu-Glu-Ala-Val-Arg-Leu-Phe-Ile-Glu-Trp-Leu-Lys-Asn-Gly-Gly-Pro-Ser-Ser-Gly-Ala-Pro-Pro-Ser-Lys-Lys-Lys-Lys-Lys-Lys-NH2

Sequence Shortening: HGEGTFTSDLSKQMEEEAVRLFIEWLKNGGPSSGAPPSKKKKKK-NH2

Target: GCGR; MEK; Akt; MMP; JNK

Pathway: GPCR/G Protein; MAPK/ERK Pathway; PI3K/Akt/mTOR; Metabolic Enzyme/Protease

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

H_2O : 100 mg/mL (20.58 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2058 mL	1.0291 mL	2.0583 mL
	5 mM	0.0412 mL	0.2058 mL	0.4117 mL
	10 mM	0.0206 mL	0.1029 mL	0.2058 mL

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

BIOLOGICAL ACTIVITY

Description

Lixisenatide is a GLP-1 receptor agonist. Lixisenatide inhibits the inflammatory response through down regulation of proinflammatory cytokines, and blocks of cellular signaling pathways. Lixisenatide decreases atheroma plaque size and instability in $Apoe^{-/-}$ $Irs^{2+/-}$ mice by reprogramming macrophages towards an M2 phenotype, which leads to reduced inflammation[2][3][5].

IC_{50} & Target

MEK1	MEK2	MMP13	MMP-1
MMP-3			

In Vitro

Lixisenatide (100 μM, 24 h) inhibits the Aβ25-35-induced cytotoxicity on cultured hippocampal cells. [1].

Lixisenatide (100 μM, 24 h) relieves the Aβ25-35-induced suppression of the Akt-MEK1/2 signaling pathway on cultured

hippocampal cells [1].
Lixisenatide (10-20 μM, 48 h) ameliorates IL-1β-induced oxidative stress, mitochondrial dysfunction, and apoptosis in fibroblast-like synoviocytes (FLSs) [3].
Lixisenatide (10-20 μM, 48 h) reduces IL-1β-induced expression of MMPs and inhibits activation of proinflammatory pathways by IL-1β in FLSs[3].
Lixisenatide (10-20 μM, 6 h) reduced oxygen-glucose deprivation/reperfusion (OGD/R)-induced generation of ROS in HUVECs [5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Hippocampal cells
Concentration:	100 μM
Incubation Time:	24 h
Result:	Reversed Aβ25-35-triggered cytotoxicity on hippocampal cell cultures.

Western Blot Analysis[3]

Cell Line:	FLSs
Concentration:	10 μM, 20 μM
Incubation Time:	48 h
Result:	Significantly reduced expression of MMP-1, MMP-3, and MMP-13 at both the mRNA and protein levels in a dose-dependent manner.

In Vivo

Lixisenatide (10 μg/kg, Subcutaneous injection, once a day for a month) diminishes the atherosclerosis burden and produces more stable plaques [2].
Lixisenatide (10 μg/kg, Subcutaneous injection, once a day for a month) decreases systemic inflammation inatherogenic-diet-fed $Apoe^{-/-}$ $Irs^{2+/-}$ mice[2].
Lixisenatide (1 nmol/kg, Intraperitoneal injection, once a day for 14 days) afford renoprotective effects on experimental early diabetic nephropathy in a low dose[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	$Apoe^{-/-}$ $Irs^{2+/-}$ mice [2]
Dosage:	10 μg/kg
Administration:	Subcutaneous injection (s.c.)
Result:	Exhibited smaller atheromas in the aortic arch region. Reduced the lesion size in cross-sections of hearts.

Animal Model:	Diabetic rats [4]
Dosage:	1 nmol/kg
Administration:	Intraperitoneal injection (i.p.)
Result:	Showed a significant amelioration on the elevated renal parameters. Showed significant mitigation in renal MDA and total NOx^- (by 50.3 and 79.9%, respectively) and 43.9% elevation in renal total antioxidant capacity.

	Averted the observed increments in iNOS and COX-2 expressions in the renal tissues of the diabetic group. Decreased the level of TGF-β protein expression.

CUSTOMER VALIDATION

- J Mol Neurosci. 2020 Feb 10.

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REFERENCES

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