Astressin

Cat. No.: HY-P0257

CAS No.: 170809-51-5

Molecular Formula: $C_{161}H_{269}N_{49}O_{42}$

Molecular Weight: 3563.16

DPhe-HLLREVLE-Nle-ARAEQLAQ-cyclo(EAHK)-NRKL-Nle-EII-NH_2

Sequence Shortening: DPhe-HLLREVLE-Nle-ARAEQLAQ-cyclo(EAHK)-NRKL-Nle-EII-NH2

Target: Others

Pathway: Others

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)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 18.18 mg/mL (5.10 mM; ultrasonic and adjust pH to 4 with HCl)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2806 mL	1.4032 mL	2.8065 mL
	5 mM	0.0561 mL	0.2806 mL	0.5613 mL
	10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

Astressin is a potent corticotropin releasing factor (CRF) antagonist.

In Vitro

Astressin has low affinity for the CRF binding protein and high affinity (K_i=2 nM) for the cloned pituitary receptor. Astressin shows high affinity for cloned human CRF-RA1 stably expressed in CHO cells and high potency to inhibit ACTH secretion[1].

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

In Vivo

Astressin is significantly more potent than any previously tested antagonist in reducing hypophyseal corticotropin (ACTH) secretion in stressed or adrenalectomized rats. Low doses of astressin (30 μg and 100 μg per kg) administered i.v. still produce a significant decrease in ACTH levels at 45 and 90 min, respectively[1]. Astressin significantly reverses the anxiogenic-like response induced by both social stress and ICV rat/humanCRF (r/hCRF) on the elevated plus-maze, but fails to block the effects of r/hCRF-induced locomotor activity in a familiar environment[2]. Intracerebroventricular infusion of the peptide both 30 min before and 10 min after seizures decreases damage in some hippocampal cell fields by as much as 84%, a magnitude of protection greater than reported for other CRF antagonists against other models of necrotic neuronal injury.

Astressin protects even if administered only 10 min following excitotoxin exposure[3].

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PROTOCOL

Animal Administration [1]

Rats: Rat diet is supplemented with oranges, and their water contained 0.9% NaCl. They are equipped with indwelling jugular cannulae 48 h prior to the i.v. injection of either vehicle or astressin. Astressin is first diluted in sterile, distilled, apyrogenic water, and the pH is adjusted to 7.0. Further dilutions are made in 0.04 M phosphate buffer, pH 7.4, containing 0.1% bovine serum albumin and 0.01% ascorbic acid. Blood samples are obtained immediately before treatment, as well as 15-120 min later. Decanted plasma samples are frozen until assayed for ACTH concentrations[1].

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

CUSTOMER VALIDATION

- SSRN. 2023 Jul 18.

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REFERENCES

[1]. Gulyas J, et al. Potent, structurally constrained agonists and competitive antagonists of corticotropin-releasing factor. Proc Natl Acad Sci U S A. 1995 Nov 7;92(23):10575-9.

[2]. Spina MG, et al. Behavioral effects of central administration of the novel CRF antagonist astressin in rats. Neuropsychopharmacology. 2000 Mar;22(3):230-9.

[3]. Maecker H, et al. Astressin, a novel and potent CRF antagonist, is neuroprotective in the hippocampus when administered after a seizure. Brain Res. 1997 Jan 2;744(1):166-70.

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