Product Data Sheet

Product Name: Astressin 2B
Cat. No.: GC15720

Chemical Properties

Cas. No.	681260-70-8		
SMILES	CCCC[C@@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](CC(C)C)(/N=C(O)/[C@](/N=C(O)/[C@@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/C)([H])CC(O)=O)([H])CC(C)C)([H])CO)([H])CC1=CC=CC=C1)([H])CC2=CN=CN2)C)([H])CC(C)C)([H])CCCNC(N)=N)([H])CCCCN)(		
Formula	$C_{183}H_{307}N_{49}O_{53}$	M.Wt	4041.69
Solubility	Soluble to 1 mg/ml in Water	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Astressin 2B		

Protocol

Cell experiment [1]:

Cell lines	A431 epidermoid cells
Preparation Method	cells were pretreated overnight with Astressin 2b (1 μM). Non-adherent cells were removed by washing three times with PBS, and cell adhesion was estimated by a colorimetric cell proliferation assay.

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Reaction Conditions	Astressin 2b (1 μM) overnight
Applications	Ach-mediated effects on cell adhesion are blocked by astressin 2b, a CRF2 antagonist, suggesting that Ach action depends partly on CRF2 signaling.

Animal experiment [2]:

Animal models	Male Sprague-Dawley rats (240-280 g)
Preparation Method	The animals were administered indomethacin and killed 24 h later under deep ether anesthesia. Ucn I (a CRFR1 and CRFR2 agonist: 2 20 μg/kg) or astressin (a nonselective CRFR antagonist: 50 μg/kg) was given i.v. 10 min before the administration of indomethacin. NBI-27914 or astressin 2B (60 μg/kg) was given i.v. 10 min before the administration of Ucn I or indomethacin.
Dosage form	60 μg/kg astressin 2B

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Applications	Astressin 2B also exacerbated the intestinal ulcerogenic response induced by indomethacin. Urocortin I prevented indomethacin-induced intestinal lesions, together with the suppression of bacterial invasion and an increase in mucosal MPO activity and iNOS expression. Urocortin I suppressed the hypermotility response to indomethacin, and this effect was also abrogated by astressin-2B.

References:

[1]. Pelissier-Rota M, Chartier NT,et,al. A crosstalk between muscarinic and CRF2 receptors regulates cellular adhesion properties of human colon cancer cells. Biochim Biophys Acta Mol Cell Res. 2017 Jul;1864(7):1246-1259. doi: 10.1016/j.bbamcr.2017.04.008. Epub 2017 Apr 18. PMID: 28432022.
[2]. Kubo Y, Kumano A, et,al. Urocortin prevents indomethacin-induced small intestinal lesions in rats through activation of CRF2 receptors. Dig Dis Sci. 2010 Jun;55(6):1570-80. doi: 10.1007/s10620-009-0930-1. Epub 2009 Aug 26. PMID: 19707872.

Background

Astressin 2B is selective and potent antagonist of corticotropin-releasing factor receptor 2 (CRF2) (IC50 values are 1.3 and > 500 nM for CRF2 and CRF1 respectively) that

antagonizes CRF2-mediated inhibition of gastric emptying.

Ach-mediated effects on cell adhesion are blocked by astressin 2b, a CRF2 antagonist, suggesting that Ach action depends partly on CRF2 signaling[4].

Astressin-2B also exacerbated the intestinal ulcerogenic response induced by indomethacin. Urocortin I prevented indomethacin-induced intestinal lesions, together with the suppression of bacterial invasion and an increase in mucosal MPO activity and iNOS expression. Urocortin I suppressed the hypermotility response to indomethacin, and this effect was also abrogated by astressin-2B[2]. In mcie, Central administration of Ucn1 increased significantly the number of entries into the chamber of the unknown male, without changing the time of interaction and this effect was blocked by astressin2B[1]. NmU, a potent endogenous anorectic, serves as a catabolic signaling molecule in the brain. For an investigation of the possible role of receptors in mediating hyperthermia, the animals were treated with astressin 2B, a CRH2 receptor antagonist. NmU increased the colon temperature, maximal action being observed at 2-3h[3].

References:
[1]. Bagosi Z, Karasz G, et,al. The effects of CRF and urocortins on the sociability of mice. Brain Res. 2017 May 15;1663:114-122. doi: 10.1016/j.brainres.2017.03.003. Epub 2017 Mar 14. PMID: 28315311.
[2]. Kubo Y, Kumano A, et,al. Urocortin prevents indomethacin-induced small intestinal lesions in rats through activation of CRF2 receptors. Dig Dis Sci. 2010 Jun;55(6):1570-80. doi: 10.1007/s10620-009-0930-1. Epub 2009 Aug 26. PMID: 19707872.
[3]. Telegdy G, Adamik A. Mediators involved in the hyperthermic action of neuromedin U in rats. Regul Pept. 2014 Jun-Aug;192-193:24-9. doi: 10.1016/j.regpep.2014.07.004. Epub 2014 Aug 7. PMID: 25108055.
[4]. Pelissier-Rota M, Chartier NT, et,al. A crosstalk between muscarinic and CRF2 receptors regulates cellular adhesion properties of human colon cancer cells. Biochim Biophys Acta Mol Cell Res. 2017 Jul;1864(7):1246-1259. doi: 10.1016/j.bbamcr.2017.04.008. Epub 2017 Apr 18. PMID: 28432022.