

Product Data Sheet

Product Name: Urantide
Cat. No.: GC50138

Chemical Properties

Cas. No. 669089-53-6

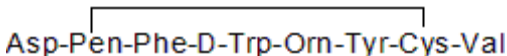
SMILES CC(C)[C@@H](NC(=O)[C@@H]1CSCC(C)(C)[C@@H](NC(=O)[C@@H](N)CC(O)=O)C(=O)N[C@@H](Cc2ccccc2)C(=O)N[C@@H](Cc2c[nH]c3ccccc23)C(=O)N[C@@H](CCCN)C(=O)N[C@@H](Cc2ccc(O)cc2)C(=O)N1)C(O)=O

Formula C₅₁H₆₆N₁₀O₁₂S₂ M.Wt 1075.26

Solubility Soluble in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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 

Protocol

Cell experiment [1]:

Cell lines Rat vascular smooth muscle cells

Rat vascular smooth muscle cells were grown in DMEM medium with 10% (v/v) fetal bovine serum (FBS), 100µg/ml streptomycin, and 100U/ml penicillin at 37°C in 5% CO₂/atmosphere. Cells were seeded at a density of 1×10⁵ cells/ml

Preparation Method in the 96-well plates and allowed to adhere in a 5% CO₂ incubator at 37°C overnight. The cells were treated with different concentrations of Urantide (0, 0.001, 0.01, 0.1, and 1µM) in the presence of 0.01µM UT for 48h. Cell viability was evaluated.

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

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Reaction
Conditions 0, 0.001, 0.01, 0.1, and 1µM; 24h

Applications Urantide treatment significantly reduced UT-induced cell proliferation of rat vascular smooth muscle cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Wistar rats

Preparation Method Wistar rats (3-month-old; 180-200g) were housed singly in a standard environment with food and water ad libitum. The rats were randomly assigned to one of the two groups: the early-treatment group (n=30) and the late-treatment group (n=30). Then, rats in each group were divided into three subgroups: control group, MCT group, and Urantide group (n=10 per subgroup). A PAH rat model was induced by the subcutaneous injection of 50mg/kg monocrotaline in the monocrotaline and Urantide groups. One week after PAH model construction, the surviving rats (some rats died of pulmonary edema or pulmonary hemorrhage within 1 week of monocrotaline injection) were injected intraperitoneally with either Urantide (10µg/kg/day) or an equal amount of normal saline (NS) (n=8) for 3 weeks. The normal control rats (n=10) received an equal volume of 0.9% saline for 3 weeks. Ultrasonic cardiogram was monitored at week 4.

Dosage form 10µg/kg/day for 3 weeks; i.p.

Applications Urantide treatment significantly improved the structure and function of right ventricle in rats with PAH.

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References:

- [1] Zhao J, Zhang S F, Shi Y, et al. Effects of urotensin II and its specific receptor antagonist urantide on rat vascular smooth muscle cells[J]. Bosnian Journal of Basic Medical Sciences, 2013, 13(2): 78.
- [2] Wang Y, Tian W, Xiu C, et al. Urantide improves the structure and function of right ventricle as determined by echocardiography in monocrotaline-induced pulmonary hypertension rat model[J]. Clinical rheumatology, 2019, 38(1): 29-35.

Background

Urantide is a selective and competitive urotensin-II (UT) receptor antagonist, with the pK_B value of 8.32 [1]. Urantide can down-regulate the expression levels of UT and GPR14 in the liver and reduce the phosphorylation levels of Erk1/2 and JNK to prevent hepatic steatosis [2]. Urantide has been widely used to regulate the contraction and calcium release in the thoracic aorta of rats[3].

In vitro, Urantide (1 μ M) treatment for 48 hours significantly inhibited the urotensin II-stimulated proliferation of rat vascular smooth muscle cells (VSMCs) and induced cell cycle arrest[4]. Treatment with 1 μ M Urantide for 48 hours significantly reduced the expression of p-JAK2 and p-STAT3 proteins and the phenotypic transformation in rat VSMCs [5].

In vivo, Urantide treatment via intraperitoneal injection at a dose of 30 μ g/kg three times within 2 weeks significantly alleviated myocardial injury in rats with atherosclerosis[6]. Intraperitoneal injection of 10 μ g/kg/day dose of Urantide for 3 weeks significantly alleviated the increase in pulmonary arterial hypertension (PAH) induced by monocrotaline in rats and improved the structure and function of the right ventricle[7].

References:

- [1] Carotenuto A, Auriemma L, Merlino F, et al. Lead optimization of P5U and urantide: discovery of novel potent ligands at the urotensin-II receptor[J]. Journal of Medicinal Chemistry, 2014, 57(14): 5965-5974.
- [2] Cui H, Lin Y, Xie L, et al. Urantide decreases hepatic steatosis in rats with experimental atherosclerosis via the MAPK/Erk/JNK pathway[J]. Molecular Medicine Reports, 2021, 23(4): 1-10.

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- [3] Camarda V, Song W, Marzola E, et al. Urantide mimics urotensin-II induced calcium release in cells expressing recombinant UT receptors[J]. European journal of pharmacology, 2004, 498(1-3): 83-86.
- [4] Zhao J, Zhang S F, Shi Y, et al. Effects of urotensin II and its specific receptor antagonist urantide on rat vascular smooth muscle cells[J]. Bosnian Journal of Basic Medical Sciences, 2013, 13(2): 78.
- [5] Wang T, Xie L, Bi H, et al. Urantide alleviates the symptoms of atherosclerotic rats in vivo and in vitro models through the JAK2/STAT3 signaling pathway[J]. European Journal of Pharmacology, 2021, 902: 174037.
- [6] Zhao J, Miao G, Wang T, et al. Urantide attenuates myocardial damage in atherosclerotic rats by regulating the MAPK signalling pathway[J]. Life sciences, 2020, 262: 118551.
- [7] Wang Y, Tian W, Xiu C, et al. Urantide improves the structure and function of right ventricle as determined by echocardiography in monocrotaline-induced pulmonary hypertension rat model[J]. Clinical rheumatology, 2019, 38(1): 29-35.

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