Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

AVE 0991 (sodium salt)

Catalog No: tcsc1753

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
306288-04-0

Formula:
$C_{29}H_{31}N_4NaO_5S_2$

Pathway:
GPCR/G Protein

Target:
Angiotensin Receptor

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 55 mg/mL (91.26 mM)

Observed Molecular Weight:
602.7

Product Description

AVE 0991 sodium salt is a nonpeptide and orally active **Ang-(1-7) receptor Mas** agonist. AVE 0991 competes for high-affinity

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

binding of [^{125}I]-Ang-(1-7) to bovine aortic endothelial cell membranes with **IC_{50}** of 21±35 nM.

IC50 & Target: IC50: 21±35 nM (Ang-(1-7) receptor)[1]

In Vitro: AVE 0991 is a nonpeptide compound that evokes effects similar to Ang-(1-7) on the endothelium. AVE 0991 and unlabeled Ang-(1-7) compete for high-affinity binding of [^{125}I]-Ang-(1-7) to bovine aortic endothelial cell membranes with **IC_{50}**s of 21±35 and 220±280 nM, respectively. Peak concentrations of NO and O_2^- release by AVE 0991 sodium salt and Ang-(1-7) (both 10 μM) are not significantly different (NO: 295±20 and 270±25 nM; O_2^-: 18±2 and 20±4 nM). However, the released amount of bioactive NO is ≈5 times higher for AVE 0991 in comparison to Ang-(1-7)[1].

In Vivo: AVE 0991 (0.58 nmol/g) produces a significant decrease of water diuresis in WT mice compared with vehicle-treated animals (0.06±0.03 mL versus 0.27±0.05; n=9 for each group; P2O versus 681.1±165.8 mOsm/KgH_2O in vehicle-treated mice; PMas abolishes the antidiuretic effect of AVE 0991 during water loading (0.37±0.10 mL [n=9] versus 0.27±0.03 mL [n=11] in AVE 0991-treated mice). As observed with C57BL/6 mice, administration of AVE 0991 (0.58 nmol/g) in water-loaded Swiss mice also produces a significant decrease of the urinary volume compared with vehicle-treated animals (0.13±0.05 mL [n=16] versus 0.51±0.04 mL [n=40]; P[2]. One week of treatment with AVE-0991 produces a significant decrease in perfusion pressure (56.55±0.86 vs. 68.73±0.69 mmHg in vehicle-treated rats) and an increase in systolic tension (11.40±0.05 vs. 9.84±0.15 g in vehicle-treated rats), rate of tension rise (+dT/dt; 184.30±0.50 vs. 155.20±1.97 g/s in vehicle-treated rats), rate of tension fall (−dT/dt; 179.60±1.39 vs. 150.80±2.42 g/s in vehicle-treated rats). A slight increase in heart rate (HR) is also observed (220.40±0.71 vs. 214.20±0.74 beats/min in vehicle-treated rats[3].