Senktide

Cat. No.: HY-P0187
CAS No.: 106128-89-6
Molecular Formula: C₄₀H₅₅N₇O₁₁S
Molecular Weight: 841.97
Target: Neurokinin Receptor
Pathway: GPCR/G Protein; Neuronal Signaling
Storage: Powder
-80°C 2 years
-20°C 1 year
In solvent
-80°C 6 months
-20°C 1 month

Solvent & Solubility

In Vitro
10 mM in DMSO

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>1.1877 mL</td>
<td>5.9385 mL</td>
<td>11.8769 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.2375 mL</td>
<td>1.1877 mL</td>
<td>2.3754 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1188 mL</td>
<td>0.5938 mL</td>
<td>1.1877 mL</td>
</tr>
</tbody>
</table>

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

BIOLICAL ACTIVITY

Description
Senktide is a tachykinin NK₃ receptor agonist. Sequence: Suc-Asp-(Me-Phe)-Phe-Gly-Leu-Met-NH₂.

IC₅₀ & Target

NK₃ receptor[1]

In Vitro
The selective NK₃ receptor agonist Senktide excites 24 of 31 dopaminergic neurons in the substantia nigra pars compacta in a concentration-dependent manner. The effective concentration range is between 3 to 3000 nm. The mean EC₅₀ for Senktide is 41.2±9 nm (n=5)[2].

In Vivo
I.c.v. injection of Senktide causes a dose-dependent increase in total distance traveled (F₆,7₂=6.344, P<0.001). This increase reaches statistical significance compared to the vehicle-treated group at 0.06 nmol and higher. The Senktide-induced increase in locomotor activity brought about by 0.1 nmol of Senktide is significantly and dose-dependently decreased by the tachykinin NK₃ receptor antagonists talnetant at 30 mg/kg and SB222200 at 30 mg/kg, but not by osanetant, when tested in parallel in a single experiment (F₇,7₈=10.32, P<0.001), although a non-significant reduction is observed. However, when tested using another vehicle (Vitamin E and glycofurol), osanetant does decrease activity.
significantly compare to Senktide-treated gerbils ($F_{2,30}=10.10$, $P<0.001$)[1].

## PROTOCOL

### Cell Assay [2]

Experiments are performed on brain slices (300 μM thick) from 150 g male Wistar rats and extracellular recordings are made by conventional techniques. Drugs (including Senktide) are applied by bath perfusion and removal is achieved simply by returning to the control drug-free solution. Extracellular electrodes are filled with aCSF and have resistances of 5 to 14 MΩ. For intracellular recordings, electrodes are filled with 1 M potassium acetate and have d.c. resistances of 70 to 110 MΩ. Neurons are considered to be dopaminergic if they have a characteristic waveform, slow firing rate (~5 Hz) and inhibitory response to dopamine[2].

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

### Animal Administration [1]

For the Senktide dose-response curve, gerbils are first allowed to habituate to the test area for 30 min. Animals ($n=10$ to 12 per drug treatment group) are anesthetized with isoflurane, a small incision is made in the skin over bregma, and an injection of Senktide at 0.01, 0.03, 0.06, 0.1, 0.3 or 0.6 nmol in 5 μL of vehicle is placed i.c.v. using a syringe with a 4.5 mm long needle. Wounds are clipped shut, and animals allowed to awaken from anesthesia, then placed directly into the locomotor activity boxes and recording commenced. For testing of the NK$_1$ receptor antagonist aprepitant (1, 3 or 10 mg/kg p.o.), gerbils are first treated with aprepitant or vehicle (0.9% NaCl with 0.3% Tween80) and returned to the home cage for 90 min. Animals are then placed in the open field for 30 min of habituation. During the last 5 min of habituation, 0.03 nmol Senktide is injected i.c.v[1].

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## REFERENCES
