Alniditan

Cat. No.: HY-101698
CAS No.: 152317-89-0
Molecular Formula: C₁₇H₂₆N₄O
Molecular Weight: 302.41
Target: 5-HT Receptor
Pathway: GPCR/G Protein; Neuronal Signaling
Storage: Please store the product under the recommended conditions in the COA.

Solvent & Solubility

<table>
<thead>
<tr>
<th>Solvent &amp; Solubility</th>
<th>In Vitro</th>
<th>10 mM in DMSO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparing Stock Solutions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concentration</td>
<td>Mass</td>
<td>1 mg</td>
</tr>
<tr>
<td>1 mM</td>
<td>3.3068 mL</td>
<td>16.5338 mL</td>
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<tr>
<td>5 mM</td>
<td>0.6614 mL</td>
<td>3.3068 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.3307 mL</td>
<td>1.6534 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
Alniditan is a potent 5-HT₁B/₁D receptors agonist, with IC₅₀ of 1.7 and 1.3 nM in HEK 293 cells, and pKᵢ value of 8.96 and 9.40 for 5-HT₁B/₁D receptors, respectively.

IC₅₀ & Target
IC₅₀: 1.7 nM (HT₁B, in HEK 293 cell), 1.3 nM (HT₁D, in HEK 293 cells)[3]

In Vitro
In vitro, alniditan exhibits little vasoconstrictive effects on the rat basilar artery, although at a very high concentration 1 mM, alniditan causes intensive constriction, most likely through a mechanism independent from 5-HT receptor activation[1]. Alniditan is 10 times more potent than sumatriptan at the h5-HT1B receptor, and twice as potent at the h5-HT1D receptor[3].

In Vivo
The intraperitoneal administration of alniditan ED₅₀=9 μg/kg and sumatriptan ED₅₀=70 μg/kg dose dependently reduces [₁²⁵I]-BSA extravasation in the rat meninges when done 30 min before stimulation. The estimated ED values for alniditan are 9 μg/kg in the absence and 190 μg/kg in the presence of GR 127935[1]. Alniditan (3, 10, 30 and 100 μg/kg) produces a dose-dependent increase in the arteriovenous oxygen saturation difference, which seems to be attenuated in animals treated with GR127935. Alniditan dose-dependently decreases total carotid and arteriovenous anastomotic blood flow and concomitant conductance values; nutrient blood flow and conductance increase.
Alniditan also produces significant increases in vascular conductance to the skin, ear, bone, salivary gland, fat, tongue, brain and dura mater; no changes are observed in the muscles and eyes[2].

**PROTOCOL**

**Animal Administration**[2]

After a stabilisation period of about 1 h, the animals are divided into three groups. In the first group (n=4), values of heart rate, mean arterial blood pressure, carotid blood flow and its distribution, as well as arterial and jugular venous blood gases are measured at baseline, and after four consecutive injections of physiological saline (0.5 mL, every 20 min). The second and third groups of animals (n=6 each) are pre-treated with saline (i.v.) or GR127935 (0.5 mg/kg, i.v.), respectively, given over a period of 5 min at a rate of 1 mL/min. After a waiting period of 15 min, baseline values of heart rate, mean arterial blood pressure, carotid blood flow and its distribution, as well as arterial and jugular venous blood gases are measured. Subsequently, these groups of animals receive sequential i.v. doses of alniditan (3, 10, 30 and 100 μg/kg) every 20 min. Fifteen minutes after each dose of alniditan, all haemodynamic variables are assessed again.

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

**REFERENCES**

