**S1RA hydrochloride**

**Cat. No.:** HY-18099A  
**CAS No.:** 1265917-14-3  
**Molecular Formula:** \( \text{C}_{20}\text{H}_{24}\text{ClN}_{3}\text{O}_{2} \)  
**Molecular Weight:** 373.88  
**Target:** Sigma Receptor  
**Pathway:** GPCR/G Protein; Neuronal Signaling  
**Storage:**  
- Powder: \(-20^\circ\text{C}\) 3 years  
- \(4^\circ\text{C}\) 2 years  
- In solvent: \(-80^\circ\text{C}\) 6 months  
- \(-20^\circ\text{C}\) 1 month

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**SOLVENT & SOLUBILITY**

<table>
<thead>
<tr>
<th>Mass (mg)</th>
<th>Solvent Concentration</th>
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<tbody>
<tr>
<td>1 mg</td>
<td>1 mM</td>
</tr>
<tr>
<td>2.6747 mL</td>
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</tr>
<tr>
<td>5 mg</td>
<td>5 mM</td>
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<tr>
<td>13.3733 mL</td>
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<tr>
<td>10 mg</td>
<td>10 mM</td>
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<tr>
<td>26.7465 mL</td>
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In Vitro: DMSO: \(\geq 57\text{ mg/mL}(152.46\text{ mM})\)

*“\(\geq\)" means soluble, but saturation unknown.*

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**BIOLOGICAL ACTIVITY**

**Description**

S1RA hydrochloride (E-52862 hydrochloride) is a potent and selective sigma-1 receptor (\(\sigma_1\)R, \(\text{Ki} = 17\text{ nM})\) antagonist, showed good selectivity against \(\sigma_2\)R (\(\text{Ki} > 1000\text{ nM})\) IC50 value: 17 nM (K) [1] Target: \(\sigma_1\)R antagonist

in vitro: S1RA behaved as a highly selective \(\sigma_1\) receptor antagonist. It showed high affinity for human (\(\text{Ki} = 17\text{ nM})\) and guinea pig (\(\text{Ki} = 23.5\text{ nM})\) \(\sigma_1\) receptors but no significant affinity for the \(\sigma_2\) receptors (\(\text{Ki} > 1000\text{ nM})\) for guinea pig and rat \(\sigma_2\) receptors). Moderate affinity (\(\text{Ki} = 328\text{ nM})\) and antagonistic activity, with very low potency (IC50 = 4700 nM) was found at the human 5-HT2B receptor. S1RA showed no significant affinity (\(\text{Ki} > 1\mu\text{M} \text{ or } \%\text{ inhibition at } 1\mu\text{M} < 50\%\)) for other additional 170 targets (receptors, transporters, ion channels and enzymes) [2].

in vivo: Control (non-operated) and nerve-injured mice received a single or repeated (twice daily for 12 days) i.p. administration of S1RA at 25 mg·kg·1, the same dose used for the assessment of behavioural hypersensitivity in the chronic treatment study. Acute treatment was given on day 12 post-surgery and repeated treatment with S1RA started the day of surgery, as in the behavioural studies [2]. Intrathecal pre-treatment with idazoxan prevented the systemic S1RA antinociceptive effect, suggesting that the S1RA antinociception depends on the activation of spinal \(\alpha_2\) -adrenoceptors which, in turn, could induce an inhibition of formalin-evoked glutamate release. When administered locally, intrathecal S1RA
inhibited only the flinching behavior, whereas intracerebroventricularly or intraplantarly injected also attenuated the lifting/licking behavior [3].

REFERENCES


Caution: Product has not been fully validated for medical applications. For research use only.
Tel: 609-228-6898                   Fax: 609-228-5909                   E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA