Lycorine hydrochloride

Cat. No.: HY-N0289
CAS No.: 2188-68-3
Molecular Formula: C₁₆H₁₈ClNO₄
Molecular Weight: 323.77
Target: Autophagy
Pathway: Autophagy
Storage: Powder
-20°C 3 years
4°C 2 years
In solvent
-80°C 6 months
-20°C 1 month

Solvent & Solubility

In Vitro

DMSO: ≥ 31 mg/mL (95.75 mM)

* “≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solvent Concentration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 mM</td>
<td>3.0886 mL</td>
<td>15.4431 mL</td>
<td>30.8861 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM</td>
<td>0.6177 mL</td>
<td>3.0886 mL</td>
<td>6.1772 mL</td>
<td></td>
</tr>
<tr>
<td>10 mM</td>
<td>0.3089 mL</td>
<td>1.5443 mL</td>
<td>3.0886 mL</td>
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</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description

Lycorine (hydrochloride) is VE-cadherin inhibitor, and has IC50 of 1.2 μM in Hey1B cell. IC50: 1.2 μM (Hey1B cell) [2]

In vitro: Lycorine (hydrochloride) executed an anti-melanoma vasculogenic effect by inhibiting VE-cadherin gene expression in C8161 cells and caused a decrease in cell surface exposure of VE-cadherin protein. Consistently, LH significantly suppressed VE-cadherin gene promoter activity. [1] Lycorine (hydrochloride) effectively inhibited mitotic proliferation of Hey1B cells (half maximal inhibitory concentration = 1.2 μM) with very low toxicity, resulting in cell cycle arrest at the G2/M transition through enhanced expression of the cell cycle inhibitor p21 and marked down-regulation of cyclin D3 expression. Moreover, LH suppressed both the formation of capillary-like tubes by Hey1B cells cultured in vitro. [2] In vivo: Lycorine effectively suppressed C8161 cell-dominant tumor formation and generation of tumor blood vessels in vivo with low toxicity. [1] Lycorine (hydrochloride) suppressed the formation of the ovarian cancer cell-dominant neovascularization in vivo when administered to Hey1B-xenotransplanted mice, suggest that LH selectively inhibits ovarian cancer cell proliferation and neovascularization and is a potential drug candidate for anti-ovarian cancer therapy. [2]
REFERENCES
