SN50

Cat. No.: HY-P0151  
CAS No.: 213546-53-3  
Molecular Formula: C₁₂₉H₂₃₀N₃₆O₂₉S  
Molecular Weight: 2781.5  
Sequence: Ala-Ala-Val-Ala-Leu-Pro-Ala-Val-Leu-Ala-Leu-Ala-Pro-Val-Gln-Arg-Lys-Arg-Gin-Lys-Leu-Met-Pro  
Sequence Shortening: AAVALLPAVLALLAPVQRKRQKLMP  
Target: NF-κB  
Pathway: NF-κB  
Storage: Powder  
-80°C  2 years  
-20°C  1 year  
In solvent  
-80°C  6 months  
-20°C  1 month

SOLVENT & SOLUBILITY

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>H₂O</td>
<td>≥ 50 mg/mL (17.98 mM)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In Vitro  

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

BIOLOGICAL ACTIVITY

Description  
SN50 is a cell permeable inhibitor of NF-κB translocation.

IC₅₀ & Target  
NF-κB

In Vitro  

Pretreatment with SN50 results in a significant reduction in amount of PI-positive cells at 12, 24, and 48 h time-point post TBI compared with vehicle-treated groups[1]. Topical SN50 suppresses nuclear factor-κB activation in local cells and reduces the incidence of epithelial defects/ulceration in healing corneas. Myofibroblast generation, macrophage invasion, activity of matrix metalloproteinases, basement membrane destruction, and expression of cytokines are all decreased in treated corneas compared with controls[2]. Treating the human gastric cancer cells SGC7901 with SN50
Could significantly enhance the effects of LY294002 on inducing cell death after 24 h\textsuperscript{[3]}. SN50 can inhibit translocation of NF-κB and production of inflammatory cytokines that are implicated in lipopolysaccharide (LPS)-induced lung injury\textsuperscript{[4]}.

| In Vivo | Treatment with SN50 accelerates the recovery of motor functional outcome from 1st to 4th day. Animals subjected to SN50 pretreatment demonstrate a significant decrease in the visuospatial learning latencies relative to the control group at 7 and 8 days post-TBI. Pretreatment with SN50 results in a significant reduction of NF-κB p65 protein levels from 6 to 48 h post-TBI and TNF-α protein levels from 12 to 48 h post-TBI\textsuperscript{[1]}. |

**PROTOCOL**

**Cell Assay**\textsuperscript{[3]}

SN50 is diluted in distilled sterilization water to create a stock solution. The final concentration of the SN50 solution used is 18 µM. Cell viability is assessed with MTT assay. To determine the effects of SN50 on enhancing the role of LY294002 on SGC7901 cells, cells are plated into 96-well microplates (7×1000 cells/well) and cultured for 24 h. Then LY294002 (50 µM), SN50 (18 µM) and LY294002+SN50 are added to the culture medium and cell viability is assessed with MTT 24 h after drug treatment\textsuperscript{[3]}.

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

**Animal Administration**\textsuperscript{[1]}

Mice: SN50 is prepared in saline (total volume: 1 µL, concentration: 0.1 µg/µL). SN50 is administered into the ipsilateral cerebral ventricle 10 min before TBI. After TBI, the bone flap is replaced, the scalp incision is sutured, and then mice are allowed to awaken and returned to their cages. Mice are killed at 1, 6, 12, 24, 48, and 72 h after operation. Loss of plasmalemma integrity is evaluated by intraperitoneal injection of PI 1 h before killing the animal\textsuperscript{[1]}.

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

**CUSTOMER VALIDATION**

- Life Sci. 2019 May 1;224:212-221.

See more customer validations on [www.MedChemExpress.com](http://www.MedChemExpress.com)

**REFERENCES**


