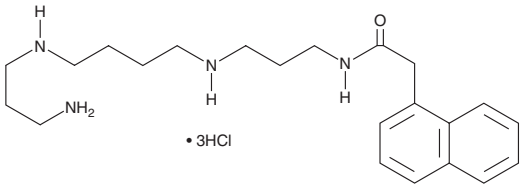
PRODUCT INFORMATION

NASPM (hydrochloride)

Item No. 18453

CAS Registry No.:	1049731-36-3
Formal Name:	N-(3-((4-((3-aminopropyl)amino) butyl)amino)propyl)-2-(naphthalen-1-yl)acetamide, trihydrochloride
Synonym:	1-Naphthylacetyl spermine
MF:	$C_{22}H_{34}N_4O \bullet 3HCl$
FW:	479.9
Purity:	≥95%
UV/Vis.:	λ_{max}: 224, 280 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

NASPM (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the NASPM (hydrochloride) in the solvent of choice, which should be purged with an inert gas. NASPM (hydrochloride) is soluble in the organic solvent DMSO at a concentration of approximately 1.7 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of NASPM (hydrochloride) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of NASPM (hydrochloride) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

NASPM is a synthetic analog of Joro spider toxin that blocks GluR2-lacking, Ca^{2+}-permeable AMPA receptors. The blocking effect of NASPM on these receptors is use and voltage-dependent. At -60 mV, NASPM can suppress current responses in kainate-induced type II neurons (which are typified by strong inward rectification and high permeability to Ca^{2+}) with an IC_{50} value of 0.33 µM.[1] However, at +40 mV, NASPM does not affect Ca^{2+}-permeable AMPA receptors.[1] NASPM has been shown to protect against ischemia-induced neuronal cell death by preventing influx of toxic levels of Ca^{2+} into injured neurons.[2] This compound can be used to study the role of GluR2 subunits in calcium permeability of AMPA receptors.[2,3]

References

1. Koike, M., Iino, M., and Ozawa, S. Blocking effect of 1-naphthyl acetyl spermine on Ca(2+)-permeable AMPA receptors in cultured rat hippocampal neurons. *Neurosci. Res.* **29(1)**, 27-36 (1997).
2. Oguro, K., Oguro, N., Kojima, T., *et al.* Knockdown of AMPA receptor GluR2 expression causes delayed neurodegeneration and increases damage by sublethal ischemia in hippocampal CA1 and CA3 neurons. *J. Neurosci.* **19(21)**, 9218-9227 (1999).
3. Dong, L.-D., Gao, F., Wang, X.-H., *et al.* GluA2 trafficking is involved in apoptosis of retinal ganglion cells induced by activation of EphB/EphrinB reverse signaling in a rat chronic ocular hypertension model. *J. Neurosci.* **35(13)**, 5409-5421 (2015).

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