
Product Data Sheet

Product Name: Gap19
 Cat. No.: GC17681

Chemical Properties

Cas. No. 1507930-57-5

SMILES CC[C@]([C@@]([N=C(O)]/[C@]([N=C(O)]/[C@]([N=C(O)]/[C@]([N=C(O)]/[C@](N)([H])CCCCN)([H])CCC(O)=N)([H])[C@@](CC)([H])C)([H])CCC(O)=O)([H])/C(O)=N/[C@@]([C(O)=N/[C@@]([C(O)=N/[C@@]([C(O)=N/[C@@]([C(O)=N/[C@@](C(O)=O)([H])CCCCN)([H])CC1=CC=CC=C1)([H])CCCCN)([H])CCCCN)([H])C

Formula C₅₅H₉₆N₁₄O₁₃ M.Wt 1161.45

Solubility ≥ 58.0725mg/mL in Water Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure

Protocol**Cell experiment [1]:**

Cell lines Astrocyte cell

Preparation Method Concentration-dependent inhibition by Gap19 (30 min pre-incubation) of ATP release in cultured cortical astrocytes triggered by glutamate.

Reaction Conditions 0-104uM Gap19 for 30 min

Caution: Product has not been fully validated for medical applications. For research use only.

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Applications

Gap19 (30 min treatment) inhibited glutamate-triggered ATP release (Figure 2A). Etd+ uptake was inhibited in the presence of Gap19 in a dose-dependent manner, with the peptide applied prior to (30 min) and during Etd+ uptake. Gap19 was without any effect on gap junctional communication in astrocytes which in culture express only Cx43.

Animal experiment [2]:

Animal models

Male ICR mice (25-30 g)

Preparation Method

Animals were randomly divided into different groups as follows: (I) vehicle group (sham); (II) Gap19 treated vehicle group (Gap19 group); (III) I/R group; (IV) Gap26 treated I/R group (I/R + Gap26 group); (V) Gap19 treated I/R group (I/R + Gap19 group). 10 µg Gap19/Gap26 in 10 µl ddH₂O was injected in lateral ventricle (300 µg/kg body weight, i.c.v.). TAT-Gap19 was administered at a dose of 25 mg/kg body weight (i.p.) in the post-treatment groups.

Dosage form

10 µg Gap19

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Applications

When investigated the role of Gap19 on cerebral ischemia/reperfusion (I/R) injury in a mouse model of middle cerebral artery occlusion (MCAO). Ventricle-injected Gap19 significantly alleviated infarct volume, neuronal cell damage and neurological deficits after ischemia, the neuroprotective effect of Gap19 was significant stronger than Gap26. Post-treatment with TAT-Gap19 still provided neuroprotection when it was administered intraperitoneally at 4 h after reperfusion.

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References:

[1]. Abudara V, Bechberger J, et,al. The connexin43 mimetic peptide Gap19 inhibits hemichannels without altering gap junctional communication in astrocytes. Front Cell Neurosci. 2014 Oct 21;8:306. doi: 10.3389/fncel.2014.00306. PMID: 25374505; PMCID: PMC4204617.

[2].Chen B, Yang L, et,al. Inhibition of Connexin43 hemichannels with Gap19 protects cerebral ischemia/reperfusion injury via the JAK2/STAT3 pathway in mice. Brain Res Bull. 2019 Mar;146:124-135. doi: 10.1016/j.brainresbull.2018.12.009. Epub 2018 Dec 26. PMID: 30593877.

Background

Gap19 (KQIEIKKFK) blocks HCs but not GJCs and is specific for Cx43. Gap19 may resist myocardial ischemic injury^[2]. Gap19 has a bimodal effect on Cx43 HC gating, decreasing gating to the fully open state while increasing substate gating, suggesting that Gap19 acts like a gating modifier on Cx43 HC. Gap19 disrupts the CT-CL interaction, thus making hc difficult to open^[7].

Gap19, as a nonapeptide derived from the cytoplasmic loop of Cx43, inhibits astroglial Cx43 hemichannels in a dose-dependent manner, without affecting gap junction channels. This peptide, which not only selectively inhibits hemichannels but is also specific for Cx43^[5].

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References:

- [1]. Chen B, Yang L, et,al. Inhibition of Connexin43 hemichannels with Gap19 protects cerebral ischemia/reperfusion injury via the JAK2/STAT3 pathway in mice. *Brain Res Bull.* 2019 Mar;146:124-135. doi: 10.1016/j.brainresbull.2018.12.009. Epub 2018 Dec 26. PMID: 30593877.
- [2]. Wang JP, Yang ZT, et,al. L-carnosine inhibits neuronal cell apoptosis through signal transducer and activator of transcription 3 signaling pathway after acute focal cerebral ischemia. *Brain Res.* 2013 Apr 24;1507:125-33. doi: 10.1016/j.brainres.2013.02.032. Epub 2013 Feb 27. PMID: 23454231.
- [3]. Wang N, De Vuyst E, et,al. Selective inhibition of Cx43 hemichannels by Gap19 and its impact on myocardial ischemia/reperfusion injury. *Basic Res Cardiol.* 2013 Jan;108(1):309. doi: 10.1007/s00395-012-0309-x. Epub 2012 Nov 8. PMID: 23184389; PMCID: PMC3666173.
- [4]. Chen Y, Wang L, et,al. Inhibition of Connexin 43 Hemichannels Alleviates Cerebral Ischemia/Reperfusion Injury via the TLR4 Signaling Pathway. *Front Cell Neurosci.* 2018 Oct 17;12:372. doi: 10.3389/fncel.2018.00372. PMID: 30386214; PMCID: PMC6199357.
- [5]. Abudara V, Bechberger J, et,al. The connexin43 mimetic peptide Gap19 inhibits hemichannels without altering gap junctional communication in astrocytes. *Front Cell Neurosci.* 2014 Oct 21;8:306. doi: 10.3389/fncel.2014.00306. PMID: 25374505; PMCID: PMC4204617.

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[6]. Ahmed F, Rahman M, et,al. Role of Connexin 43 in an Inflammatory Model for TMJ Hyperalgesia. Front Pain Res (Lausanne). 2021 Aug 3;2:715871. doi:

10.3389/fpain.2021.715871. PMID: 35295418; PMCID: PMC8915650.

[7]. Lissoni A, Wang N, et,al. Gap19, a Cx43 Hemichannel Inhibitor, Acts as a Gating Modifier That Decreases Main State Opening While Increasing Substate Gating. Int J Mol Sci. 2020 Oct 5;21(19):7340. doi: 10.3390/ijms21197340. PMID: 33027889; PMCID: PMC7583728.

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