

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])CCCN)([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)=O)([H])CCCCN)([H])CC1=CC=C C=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.

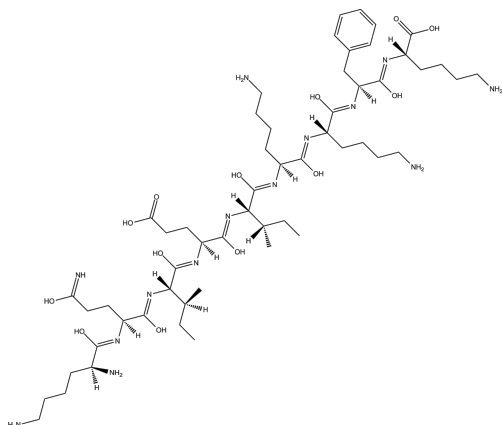
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:	
Cell lines	Human umbilical vein endothelial cells (HUVECs)
Preparation Method	HUVECs were cultured in DMEM medium, supplemented with 10% fetal bovine serum (FBS), and 1% penicillin/streptomycin at 37°C in an incubator with 5% CO ₂ . Cells in the logarithmic growth phase were seeded in a 6-well plate at a density of 3×10^5 cells/well overnight. Cells were treated with 100μM Gap19 and 400ng/ml LPS for 24h. Subsequently, the cell viability was detected.
Reaction Conditions	100μM; 24h

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Applications	Gap19 treatment reduced the LPS-induced cell death in HUVECs.
Animal experiment [2]:	
Animal models	Male ICR mice
Preparation Method	Male ICR mice (25-30g) were maintained on a 12:12h light-dark schedule in a temperature-controlled, specific pathogen-free facility and fed standard laboratory mouse chow. Mice were randomly divided into different groups as follows: (I) vehicle group (sham); (II) Gap19-treated vehicle group (Gap19 group); (III) I/R group; (IV) Gap19-treated I/R group (I/R + Gap19 group). Gap19 in 10µl ddH₂O was injected into the lateral ventricle (300µg/kg; i.c.v.). Mice were anesthetized with chloral hydrate (300mg/kg) intraperitoneally. The right common carotid artery was exposed and isolated. The middle cerebral artery was occluded by inserting a monofilament nylon suture into the internal carotid artery, and the heat-rounded tip of the nylon suture was advanced further until it closed the origin of the middle cerebral artery. Forty-five minutes after induction of ischemia, reperfusion was initiated by withdrawal of the monofilament for 24h. Rectal temperature was maintained at 37°C from the beginning of the surgery until the animals recovered from anesthesia. Sham-operated mice underwent the same surgical procedure without inserting a filament. After 24h of reperfusion, the mice's brains were collected for analysis.
Dosage form	300µg/kg; once; i.c.v.

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Applications	Gap19 treatment reduced the I/R induced cerebral infarction volume in mice.
References: [1] Ma J, Ji D, Li Q, et al. Inhibition of connexin 43 attenuates oxidative stress and apoptosis in human umbilical vein endothelial cells[J]. BMC pulmonary medicine, 2020, 20(1): 19. [2] Chen B, Yang L, Chen J, et al. Inhibition of Connexin43 hemichannels with Gap19 protects cerebral ischemia/reperfusion injury via the JAK2/STAT3 pathway in mice[J]. Brain research bulletin, 2019, 146: 124-135.	

Background

Gap19 is a Cx43 hemichannel blocker derived from the second cytoplasmic loop of Cx43 [1]. Gap19 inhibits the Cx43 hemichannel, thereby preventing the inflammatory cascade reaction, reducing the production of inflammatory cytokines, and promoting cell survival and tissue repair in models of heart, spinal cord injury, and eye damage[2]. Gap19 has been widely used in oxidative stress experiments to prevent radiation-induced cell death and the generation of reactive oxygen species (ROS)[3].

In vitro, Gap19 treatment (100μM) for 12 hours reduced the overexpression of Cx43 induced by heme stimulation in astrocytes, increased the expression of YAP in the cell nucleus, and decreased the expression of YAP in the cytoplasm[4]. Treatment with 100μM Gap19 for 24 hours reduced lipopolysaccharide (LPS)-induced apoptosis in human umbilical vein endothelial cells (HUVECs) and downregulated the mRNA expression of GSTZ1 and CYBB[5]. 200μM of Gap19 pre-incubation for 30 minutes improved mouse cardiomyocyte viability after 120 minutes of oxygen-glucose deprivation (OGD)/acidosis (pH 6.5) followed by 3 minutes of normoxia treatment[6].

In vivo, Gap19 treatment via intraperitoneal injection at a dose of 25mg/kg/day for 5 days inhibited the alleviating effect of glial cell line-derived neurotrophic factor (GDNF) on dextran sulphate sodium (DSS)-induced ulcerative colitis in mice, resulting in reduced body weight and impaired intestinal function[7]. A single injection (i.c.v) of Gap19 (300μg/kg) 30 minutes before ischemia reduced the cerebral infarction volume induced by cerebral ischemia/reperfusion (I/R) injury in mice and alleviated

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neuronal damage^[8].

References:

- [1] Crespo Yanguas S, Da Silva T C, Pereira I V A, et al. TAT-Gap19 and carbenoxolone alleviate liver fibrosis in mice[J]. International journal of molecular sciences, 2018, 19(3): 817.
- [2] Coutinho F P, Green C R, Acosta M L, et al. Xentry-Gap19 inhibits Connexin43 hemichannel opening especially during hypoxic injury[J]. Drug Delivery and Translational Research, 2020, 10(3): 751-765.
- [3] Ramadan R, Vromans E, Anang D C, et al. Connexin43 hemichannel targeting with TAT-Gap19 alleviates radiation-induced endothelial cell damage[J]. Frontiers in pharmacology, 2020, 11: 212.
- [4] Yu H, Cao X, Li W, et al. Targeting connexin 43 provides anti-inflammatory effects after intracerebral hemorrhage injury by regulating YAP signaling[J]. Journal of Neuroinflammation, 2020, 17(1): 322.
- [5] Ma J, Ji D, Li Q, et al. Inhibition of connexin 43 attenuates oxidative stress and apoptosis in human umbilical vein endothelial cells[J]. BMC pulmonary medicine, 2020, 20(1): 19.
- [6] Wang N, De Vuyst E, Ponsaerts R, et al. Selective inhibition of Cx43 hemichannels by Gap19 and its impact on myocardial ischemia/reperfusion injury[J]. Basic research in cardiology, 2013, 108(1): 309.
- [7] Yang W, Liu R, Xu F. Glial cell line-derived neurotrophic factor improves impaired colonic motility in experimental colitis mice through connexin 43[J]. World Journal of Gastroenterology, 2025, 31(8): 100069.
- [8] Chen B, Yang L, Chen J, et al. Inhibition of Connexin43 hemichannels with Gap19 protects cerebral ischemia/reperfusion injury via the JAK2/STAT3 pathway in mice[J]. Brain research bulletin, 2019, 146: 124-135.

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