# Danegaptide Hydrochloride

**Cat. No.:** HY-10913A

**CAS No.:** 943133-81-1

**Molecular Formula:** $C_{14}H_{18}ClN_3O_4$

**Molecular Weight:** 327.76

**Target:** Gap Junction Protein

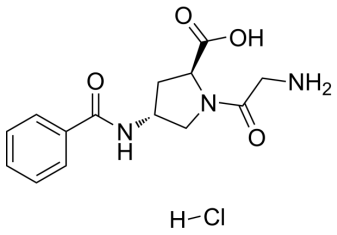
**Pathway:** Cytoskeleton

**Storage:** Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)




## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : ≥ 55 mg/mL (167.81 mM)

$H_2O$ : ≥ 50 mg/mL (152.55 mM)

* "≥" means soluble, but saturation unknown.

**Preparing Stock Solutions**

| Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 3.0510 mL | 15.2551 mL | 30.5101 mL |
| 5 mM | 0.6102 mL | 3.0510 mL | 6.1020 mL |
| 10 mM | 0.3051 mL | 1.5255 mL | 3.0510 mL |

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

**In Vivo**

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (305.10 mM); Clear solution; Need ultrasonic

## BIOLOGICAL ACTIVITY

**Description**

Danegaptide Hydrochloride (GAP-134 Hydrochloride) is a potent, selective and orally active gap-junction modifier with an antiarrhythmic effect[1][2].

**$IC_{50}$ & Target**

Gap junction.

**In Vitro**

Danegaptide (GAP-134, compound 9f; 0.01 nM-100 μM) dose dependently reduces dye uptake in cultured C6 glioma cells in a manner[1].

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

**In Vivo**

Danegaptide (GAP-134, compound 9f) is evaluated for in vivo efficacy in the mouse $CaCl_2$ model after oral administration.

For doses of 5-20 mg/kg po, Danegaptide significantly prolongs the time to conduction block in mice after the infusion of $CaCl_2$[1].

?Danegaptide (GAP-134) is biologically active upon oral administration at an average plasma concentration of 250 nM, and reduces atrial fibrillation in a dog model. Danegaptide has no effect on heart rate, arterial blood pressure or other electrocardiogram (ECG) parameters. Danegaptide is an effective antiarrhythmic compound in the setting of ischaemia/reperfusion-induced arrhythmogenesis in barbiturate-anesthetized, open-chest beagles[2].

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

## CUSTOMER VALIDATION

- Lab Invest. 2021 Sep 14.

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## REFERENCES

[1]. John A Butera, et al. Discovery of (2S,4R)-1-(2-aminoacetyl)-4-benzamidopyrrolidine-2-carboxylic acid hydrochloride (GAP-134)13, an orally active small molecule gap-junction modifier for the treatment of atrial fibrillation. J Med Chem. 2009 Feb 26;52(4):908-11.

[2]. Elke De Vuyst, et al. Pharmacological modulation of connexin-formed channels in cardiac pathophysiology. Br J Pharmacol. 2011 Jun;163(3):469-83.



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