

Product Data Sheet

Product Name: Calmidazolium chloride

Cat. No.: GC14326

Chemical Properties

Cas. No. 57265-65-3

Chemical Name (S)-1-(bis(4-chlorophenyl)methyl)-3-(2-((2,4-dichlorobenzyl)oxy)-2-(2,4-dichlorophenyl)ethyl)-1H-imidazol-3-ium chloride

SMILES ClC1=CC(Cl)=CC=C1[C@@H](C[N+]2=CN(C=C2)C(C(C=C3)=CC=C3Cl)C(C=C4)=CC=C4Cl)OCC(C(Cl)=C5)=CC=C5Cl.[Cl-]

Formula $C_{31}H_{23}Cl_7N_2O$ M.Wt 687.7

Solubility 100mM in ethanol, or in DMSO 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.

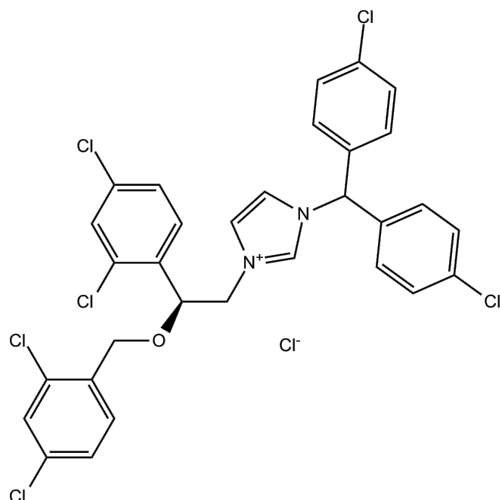
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines Head kidney macrophage (HKM)

Preparation Method HKM were pre-treated with or without Calmidazolium chloride for 1h, and then HKM were infected with *Aeromonas hydrophila* for 60min.

Reaction 4nM; 1h
Conditions

Applications Calmidazolium chloride inhibited activity of calmodulin in cells.

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Animal experiment [2]:

Animal models	Male and female C57BL/6 mice
Preparation Method	The mice in the injury group received cardiotoxin (CTX) injection into the tibialis anterior (TA) muscle and mice in the control group were injected intraperitoneally with an equal volume of saline. Twenty-four hours after the CTX injection, the mice were injected with Calmidazolium chloride.
Dosage form	3.75µg (150µl, 25µg/ml) per animal; i.p.
Applications	Calmidazolium chloride significantly decreased the level of the phosphodiesterase 1 (PDE1) in damaged muscles of mice.

References:

- [1] Banerjee C, Khatri P, Raman R, et al. Role of calmodulin-calmodulin kinase II, cAMP/protein kinase A and ERK 1/2 on *Aeromonas hydrophila*-induced apoptosis of head kidney macrophages [J]. PLoS Pathog, 2014, 10(4): e1004018.
- [2] Hu J, Shi D, Ding M, et al. Calmodulin-dependent signalling pathways are activated and mediate the acute inflammatory response of injured skeletal muscle [J]. The Journal of physiology, 2019, 597(21): 5161-5177

Background

Calmidazolium chloride is a very potent inhibitor of several Ca^{2+} -calmodulin-dependent enzymes, the IC_{50} values are 0.01µM for brain phosphodiesterase, 0.2µM for erythrocyte Ca^{2+} -ATPase and 5.0µM for phosphorylase b kinase [1]. Calmidazolium chloride is a kind of derivative of the antimycotic miconazole, and proposed as a more specific and useful tool for studying the involvement of calmodulin in biological processes [1].

Calmidazolium chloride (4nM; 1h) inhibited activity of calmodulin (CaM) in *Aeromonas hydrophila*-induced head kidney macrophage (HKM) [2]. Calmidazolium chloride (5µM) decreased the Ca^{2+}

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current in mouse pancreatic β -cells; Calmidazolium chloride (5 μ M; 10min) stimulated insulin release from mouse pancreatic β -cells [3].

Calmidazolium chloride (3.75 μ g; i.p.) significantly decreased the level of the phosphodiesterase 1 (PDE1) in damaged muscles of C57BL/6 mice induced by cardiotoxin; Calmidazolium chloride (3.75 μ g; i.p.) increased the expression of TGF- β 2 and IL-10 and down-regulated the mRNA levels of pro-inflammatory TNF- α and IL-6 in CD45⁺ cells in damaged muscles of C57BL/6 mice induced by cardiotoxin [4].

References:

- [1] Van Belle H. R 24 571: A potent inhibitor of calmodulin-activated enzymes [J]. Cell Calcium, 1981, 2(5): 483-494.
- [2] Banerjee C, Khatri P, Raman R, et al. Role of calmodulin-calmodulin kinase II, cAMP/protein kinase A and ERK 1/2 on *Aeromonas hydrophila*-induced apoptosis of head kidney macrophages [J]. PLoS Pathog, 2014, 10(4): e1004018.
- [3] Kindmark H, K  hler M, Larsson O, et al. Dissociation between exocytosis and Ca²⁺-channel activity in mouse pancreatic β -cells stimulated with calmidazolium (compound R 24571) [J]. FEBS letters, 1995, 369(2-3): 315-320.
- [4] Hu J, Shi D, Ding M, et al. Calmodulin-dependent signalling pathways are activated and mediate the acute inflammatory response of injured skeletal muscle [J]. The Journal of physiology, 2019, 597(21): 5161-5177.

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