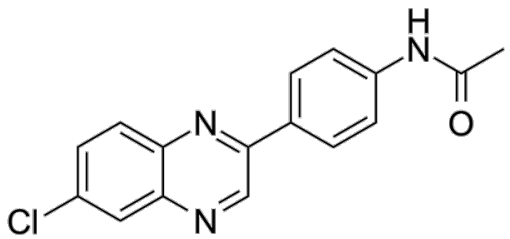
Product Data Sheet

Product Name: CA77.1
Cat. No.: GC62881

Chemical Properties

Cas. No.	2412270-22-3		
Formula	$C_{16}H_{12}ClN_3O$	M.Wt	297.74
Solubility	DMSO : 5 mg/mL (16.79 mM; ultrasonic and warming and heat to 60°C)|Ethanol : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)	Storage	4°C, protect from light
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 Murine microglial BV2 cells

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Preparation Method	BV2 cells were pretreated with 12μM CA77.1 for 12h and then exposed to LPS for 10min.
Reaction Conditions	12μM; 12h
Applications	CA77.1 decreased LPS-induced expression of iNOS and COX-2 proteins, lowered the proinflammatory factors NO and IL-6 and reduced the mRNA expression of iNOS, COX-2, IL-6, and IL-1β without affecting cell viability.

Animal experiment [2]:

Animal models	Male C57BL/6 mice
Preparation Method	Mice were randomly divided into the following groups: control group, 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47) group, BDE-47 + CA77.1 group. BDE-47 group mice were orally administered 20mg/kg/day BDE-47 and also intraperitoneally injected with normal saline containing 2 % DMSO for 8 weeks. BDE-47 + CA77.1 group mice were given an intraperitoneal injection of 10mg/kg CA77.1 every three days for the last 4 weeks prior to 20mg/kg/day BDE-47 administration.
Dosage form	10mg/kg; i.p.; every 3 days for the last 4 weeks
Applications	CA77.1 mitigated BDE-47-induced cognitive impairment in mice by suppressing ferroptosis, preventing synaptic loss in mice hippocampi and rescuing hippocampal neuronal structural atrophy.

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

References:
[1] Wu J, Han Y Y, Xu H, et al. Deficient chaperone-mediated autophagy facilitates LPS-induced microglial activation via regulation of the p300/NF-κB/NLRP3 pathway. Sci Adv. 2023 Oct 6;9(40):eadi8343.
[2] Yuan Q, Wang M W, Zhang Z X, et al. The ameliorative effects of melatonin against BDE-47-induced hippocampal neuronal ferroptosis and cognitive dysfunction through Nrf2-Chaperone-mediated autophagy of ACSL4 degradation. Ecotoxicol Environ Saf. 2025 Jan 15:290:117542.

Background

CA77.1 is an orally active small-molecule derivative of 7-chloro-3-(p-tolyl)-2H-benzo[b][1,4]oxazine (AR7) and can act as an activator of chaperone-mediated autophagy (CMA)[1]. CA77.1 exerts neuroprotective effects and is being developed for the treatment of various chronic CNS diseases including Alzheimer's disease, Parkinson’s disease and retinal degeneration[2].

In vitro, CA77.1 (12μM) pretreated murine microglial BV2 cells for 12h followed by LPS treatment. CA77.1 decreased LPS-induced iNOS and COX-2 protein expression, lowered the proinflammatory factors NO and IL-6 and reduced the mRNA expression of iNOS, COX-2, IL-6, and IL-1β without affecting cell viability[3]. CA77.1 (20μM) treatment on mouse neuroblast Neuro-2a (N2a) cells for 24h promoted the degradation of GAPDH (a canonical substrate of CMA) and augmented Na^+ /K^+ -ATPase β1 subunit elimination, accompanied by decreased Na^+ /K^+ -ATPase activity and increased concentration of intracellular Na^+, without mRNA levels changed[4]. CA77.1 (0-30μM) treatment on PK-15 porcine kidney cells expressing foot-and-mouth disease virus (FMDV) non-structural protein 3D for 24h effectively diminished FMDV 3D polymerase levels and suppressesd FMDV replication via activation of the CMA pathway[5].

In vivo, CA77.1 (10mg/kg/day) was intraperitoneally injected into intracerebral hemorrhage (ICH) mice models for six consecutive days. CA77.1 administration reversed ICH induced augmentation of A1 reactive astrocytes, myelin damage, neuronal death, and neurobehavioral disorders[6]. CA77.1 (10mg/kg) was administered intraperitoneally into mitochondrial toxin BDE-47-exposed mice with cognitive impairment every three days for 4 weeks. CA77.1 mitigated BDE-47-induced cognitive impairment in mice by suppressing ferroptosis, preventing synaptic loss in mice hippocampi and

rescuing hippocampal neuronal structural atrophy[7].

References:

[1] Bourdenx M, Martín-Segura A, Scrivo A, et al. Chaperone-mediated autophagy prevents collapse of the neuronal metastable proteome.Cell. 2021 May 13;184(10):2696-2714.e25.
[2] Wu J, Xu W, Su Y, et al. Targeting chaperone-mediated autophagy in neurodegenerative diseases: mechanisms and therapeutic potential.Acta Pharmacol Sin. 2025 Apr;46(4):816-828.
[3] Wu J, Han Y Y, Xu H, et al. Deficient chaperone-mediated autophagy facilitates LPS-induced microglial activation via regulation of the p300/NF-κB/NLRP3 pathway. Sci Adv. 2023 Oct 6;9(40):eadi8343.
[4] Zhang Y X, Wu H H, Zhang Q, et al. LAMP2A-mediated neuronal hyperexcitability by enhancing NKAβ1 degradation underlies depression-induced allodynia. Cell Rep. 2025 Apr 22;44(4):115489.
[5] Ren M, Zhou H Q, Wu J E, et al. Heat shock protein A1 inhibits the replication of foot-and-mouth disease virus by degrading viral RNA polymerase 3D through chaperone-mediated autophagy. J Virol. 2025 May 20;99(5):e0016825.
[6] Zheng Y, Peng L, Jiang G N, et al. Activation of chaperone-mediated autophagy exerting neuroprotection effect on intracerebral hemorrhage-induced neuronal injury by targeting Lamp2a. Exp Neurol. 2024 Dec:382:114986.
[7] Yuan Q, Wang M W, Zhang Z X, et al. The ameliorative effects of melatonin against BDE-47-induced hippocampal neuronal ferroptosis and cognitive dysfunction through Nrf2-Chaperone-mediated autophagy of ACSL4 degradation. Ecotoxicol Environ Saf. 2025 Jan 15:290:117542.

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