

Product Data Sheet

Product Name: CCCP
 Cat. No.: GC14727

Chemical Properties

Cas. No. 555-60-2

Chemical Name (3-chlorophenyl)carbonohydrazonoyl dicyanide

SMILES C1C=CC(N/N=C(C#N)/C#N)=CC=C1

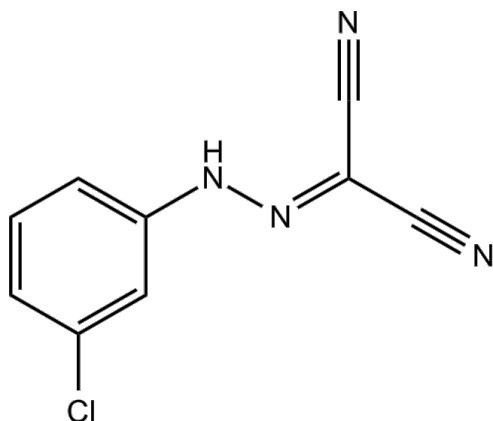
Formula $C_9H_5ClN_4$ M.Wt 204.62

Solubility $\geq 20.5\text{mg/mL}$ in DMSO Storage Store at 2-8°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	OMM1 cells

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

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Preparation Method	OMM1 cells were cultured in RPMI-1640 medium, supplemented with 10% fetal bovine serum (FBS), and 1% penicillin/streptomycin, at 37°C in an incubator with 5% CO ₂ . Cells were seeded into 96-well plates at a density of 3×10 ⁴ cells/ml and incubated for 24h at 37°C. Cells were exposed to gradient concentrations of CCCP (0, 0.1, 1, 10, and 100μM) for 72h, cell viability was measured.
Reaction Conditions	0, 0.1, 1, 10, and 100μM; 72h
Applications	CCCP treatment reduced cell viability in OMM1 cells in a dose-dependent manner.
Animal experiment [2]:	
Animal models	Male C57BL/6J mice

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Preparation Method	Male C57BL/6J mice (8 weeks old; 23-25g) were housed under standard conditions with ad libitum access to food and normal drinking water. After one week of routine feeding to acclimate to the environment, the mice underwent one week of preoperative adaptive exercise training and then underwent intracerebral hemorrhage surgery to induce an ICH model. Mice were fasted for 12 h and deprived of water for 4h prior to anesthesia. Initial anesthesia was induced using 1.5% isoflurane and maintained at 1% throughout the procedure. The mice were positioned in a prone orientation on a stereotaxic frame. A small hole (approximately 1mm in diameter) was drilled at the coordinates of the right striatum (0.8mm anterior to bregma, 2mm to the right of the midline, and 3.5mm deep). A solution containing 0.75U of type IV collagenase and 7U/μl heparin in PBS (0.4μl) was injected at a rate of 0.2μl/min over 5min. The needle was left in place for an additional 10min following the injection before withdrawal. The scalp was then sutured, and the mice were allowed to recover in a warm, ventilated environment. In the Sham group, an equal volume of saline was injected, with all other procedures remaining identical. CCCP and Mdivi-1 were diluted with DMSO and injected intraperitoneally at doses of 1mg/kg and 25mg/kg, respectively. 21 days later, the brain tissues of the mice were collected for analysis.
Dosage form	1mg/kg/day; 21 days; i.p.
Applications	CCCP treatment improved neurological functions after ICH in mice.

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- [1] Xie M, Gu X, Zhao Z, et al. Artemisinin synergizes with CCCP in autophagic cell death induction via ER stress in uveal melanoma[J]. Iscience, 2025, 28(8).
- [2] Xiaoyu L, Dandan L, Tianzhao O, et al. Resolvin D1 combined with exercise rehabilitation alleviates neurological injury in mice with intracranial hemorrhage via the BDNF/TrkB/PI3K/AKT pathway[J]. Scientific Reports, 2024, 14(1): 31447.

Background

CCCP is a protonophore altering the permeability of mitochondria inner membrane to protons [1]. CCCP increases membrane proton conductance by several orders of magnitude, thereby causing mitochondrial depolarization and uncoupling of respiration[2]. CCCP disrupts the cytosolic complex between the actin-binding protein Keap1 and the transcription factor Nrf2, releases Nrf2, and enables Nrf2 translocation to the nucleus, where Nrf2 activates the antioxidant response element (ARE)[3]. CCCP has been widely used in cell metabolism research to reduce ATP production and reverse bacterial resistance to antibiotics[4].

In vitro, CCCP treatment for 72 hours significantly inhibited the cell viability of 92-1, OMM1, and OMM2.3 cells, with the IC₅₀ values being 6.5μM, 10.7μM, and 11.1μM, respectively[5]. Incubation with 25μM CCCP for 12 hours markedly decreased the phosphorylation of mTORC1 substrates eIF4E-BP1 and S6K in HepG2 cells and promoted autophagy as well as mitochondrial dysfunction[6]. Treatment with 100μM CCCP for 12 hours induced Keap1 degradation and apoptosis in p62^{-/-} mouse embryonic fibroblast (MEF) cells, accompanied by an increase in ROS accumulation and the expression levels of cleaved PARP and caspase-3[7].

In vivo, CCCP treatment via intraperitoneal injection at a dose of 1mg/kg/day for 21 days improved the neurological deficits and motor dysfunction in the mouse model of intracerebral hemorrhage (ICH), and enhanced spatial learning and memory functions[8]. Intraperitoneal injection of CCCP at a dose of 1mg/kg/day for 2 weeks induced hepatic fibrosis without causing liver injury in mice[9].

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- [1] Kasianowicz J, Benz R, McLaughlin S. The kinetic mechanism by which CCCP (carbonyl cyanide m-chlorophenylhydrazone) transports protons across membranes[J]. The Journal of membrane biology, 1984, 82(2): 179-190.
- [2] Heytler P G. Uncoupling of oxidative phosphorylation by carbonyl cyanide phenylhydrazones. I. Some characteristics of m-Cl-CCP action on mitochondria and chloroplasts[J]. Biochemistry, 1963, 2(2): 357-361.
- [3] Kane M S, Paris A, Codron P, et al. Current mechanistic insights into the CCCP-induced cell survival response[J]. Biochemical pharmacology, 2018, 148: 100-110.
- [4] Osei Sekyere J, Amoako D G. Carbonyl cyanide m-chlorophenylhydrazine (CCCP) reverses resistance to colistin, but not to carbapenems and tigecycline in multidrug-resistant Enterobacteriaceae[J]. Frontiers in microbiology, 2017, 8: 228.
- [5] Xie M, Gu X, Zhao Z, et al. Artemisinin synergizes with CCCP in autophagic cell death induction via ER stress in uveal melanoma[J]. Iscience, 2025, 28(8).
- [6] Koncha R R, Ramachandran G, Sepuri N B V, et al. CCCP-induced mitochondrial dysfunction—characterization and analysis of integrated stress response to cellular signaling and homeostasis[J]. The FEBS journal, 2021, 288(19): 5737-5754.
- [7] Park J S, Kang D H, Bae S H. p62 prevents carbonyl cyanide m-chlorophenyl hydrazine (CCCP)-induced apoptotic cell death by activating Nrf2[J]. Biochemical and biophysical research communications, 2015, 464(4): 1139-1144.
- [8] Xiaoyu L, Dandan L, Tianzhao O, et al. Resolvin D1 combined with exercise rehabilitation alleviates neurological injury in mice with intracranial hemorrhage via the BDNF/TrkB/PI3K/AKT pathway[J]. Scientific Reports, 2024, 14(1): 31447.
- [9] Lee J H, Seo K H, Yang J H, et al. CCCP induces hepatic stellate cell activation and liver fibrogenesis via mitochondrial and lysosomal dysfunction[J]. Free Radical Biology and Medicine, 2024, 225: 181-192.

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