

Product Data Sheet

Product Name: Diamide
Cat. No.: GD10507

Chemical Properties

Cas. No. 10465-78-8

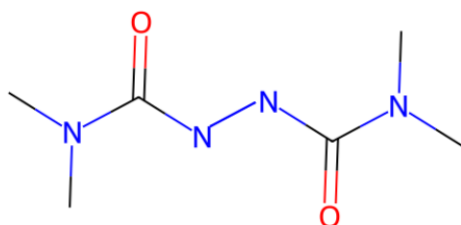
Formula $C_6H_{12}N_4O_2$ M.Wt 172.19

Solubility DMSO : >22mg/ml, water: 19.60-21.00 mg/mL, clear to slightly hazy, orange
Storage Storage at -20 ° C, filled with argon

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 LoVo cells (human colorectal carcinoma cell line)

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

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Preparation Method LoVo cells were maintained in ATCC-formulated Ham's F12K medium supplemented with 10% fetal bovine serum (FBS), 100U/mL penicillin, and 100µg/mL streptomycin at 37°C, 5% CO₂. LoVo cells were treated with diamide at concentrations of 0, 50, 100, 200, and 400µM for 15 minutes.

Reaction Conditions 0-400µM; 15h

Applications Diamide significantly reversed DDR1a knockdown-induced intracellular lactate accumulation in LoVo cells by inhibiting pyruvate kinase (PK) activity. Diamide treatment also reduced phosphorylated AKT (p-AKT) levels and suppressed PI3K/AKT/PKM2 signaling pathway activation, thereby regulating glycolytic flux and metabolic reprogramming in CRC cells.

Animal experiment [2]:

Animal models C57BL/6 mice

Preparation Method Mice were stereotactically administered a single unilateral dose of Diamide (1.45µmol) into the substantia nigra (SN). Mice were sacrificed 14 days post-injection for behavioral, histopathological, and biochemical analyses.

Dosage form 1.45µmol; stereotaxic injection; Single administration.

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Applications

Diamide injection led to hemiparkinsonian motor deficits, including reduced rotarod latency, impaired grip strength, and asymmetric contralateral rotations. Diamide selectively induced dopaminergic neurodegeneration in the ipsilateral SNpc and striatal fiber loss, accompanied by α -synuclein aggregation, glutathione depletion, and activation of the ASK1-p38 MAPK cell death signaling pathway.

References:

- [1] Xiong B, Xie Z, Song F, et al. DDR1 promotes LoVo cell proliferation by regulating energy metabolism. *Acta Biochim Biophys Sin (Shanghai)*. 2022 May 25;54(5):615-624.
- [2] Ray A, Kambali M, Ravindranath V. Thiol Oxidation by Diamide Leads to Dopaminergic Degeneration and Parkinsonism Phenotype in Mice: A Model for Parkinson's Disease. *Antioxid Redox Signal*. 2016 Aug 10;25(5):252-67.

Background

Diamide is a commonly used thiol oxidant, also recognized as a highly cell-permeable reagent that can oxidize reduced glutathione (GSH) to oxidized glutathione (GSSG) without generating reactive oxygen species (ROS)^[1-2]. Diamide has been widely applied in cell-free systems, in vitro cellular experiments, and in vivo studies to rapidly oxidize GSH and/or protein thiols^[3-4].

In vitro, Diamide (0–400 μ M) treatment of LoVo cells significantly reversed the increase in intracellular lactate levels induced by DDR1a knockdown and counteracted the regulatory effect of DDR1a on pyruvate kinase activity^[5]. Diamide (0.1–0.5mM) treatment of rat aortic smooth muscle cells concentration-dependently increased protein glutathionylation, induced mitochondrial proton leak, and reduced ATP-linked oxygen consumption; higher concentrations ($\geq$ 0.25mM) led to bioenergetic failure and cell death^[6].

In vivo, stereotaxic injection of Diamide (1.45 μ mol) into the substantia nigra of C57BL/6 mice (single injection), Diamide significantly reduced glutathione levels in brain tissue, increased oxidative stress markers, and induced degeneration of dopaminergic neurons in the substantia nigra, which can be utilized to establish a mouse model of Parkinson's disease^[7].

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- [1] Kosower NS, Kosower EM, Koppel RL. Sensitivity of hemoglobin thiol groups within red blood cells of rat during oxidation of glutathione. *Eur J Biochem.* 1977 Aug 1;77(3):529-34.
- [2] Kosower NS, Kosower EM, Wertheim B, et al. Diamide, a new reagent for the intracellular oxidation of glutathione to the disulfide. *Biochem Biophys Res Commun.* 1969 Nov 6;37(4):593-6.
- [3] Liu Y, Min W. Thioredoxin promotes ASK1 ubiquitination and degradation to inhibit ASK1-mediated apoptosis in a redox activity-independent manner. *Circ Res.* 2002 Jun 28;90(12):1259-66.
- [4] Marey-Semper I, Gelman M, Lévi-Strauss M. A selective toxicity toward cultured mesencephalic dopaminergic neurons is induced by the synergistic effects of energetic metabolism impairment and NMDA receptor activation. *J Neurosci.* 1995 Sep;15(9):5912-8.
- [5] Xiong B, Xie Z, Song F, et al. DDR1 promotes LoVo cell proliferation by regulating energy metabolism. *Acta Biochim Biophys Sin (Shanghai).* 2022 May 25;54(5):615-624.
- [6] Hill BG, Higdon AN, Dranka BP, et al. Regulation of vascular smooth muscle cell bioenergetic function by protein glutathiolation. *Biochim Biophys Acta.* 2010 Feb;1797(2):285-95.
- [7] Ray A, Kambali M, Ravindranath V. Thiol Oxidation by Diamide Leads to Dopaminergic Degeneration and Parkinsonism Phenotype in Mice: A Model for Parkinson's Disease. *Antioxid Redox Signal.* 2016 Aug 10;25(5):252-67.

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