Diphenyleneiodonium chloride

Cat. No.: HY-100965

CAS No.: 4673-26-1

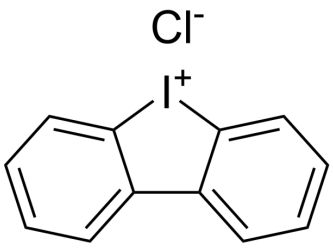
Molecular Formula: $C_{12}H_8ClI$

Molecular Weight: 314.55

Target: TRP Channel; NADPH Oxidase; Reactive Oxygen Species

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 1 years; -20°C, 6 months (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 6 mg/mL (19.07 mM; Need ultrasonic and warming)
H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1791 mL	15.8957 mL	31.7915 mL
	5 mM	0.6358 mL	3.1791 mL	6.3583 mL
	10 mM	0.3179 mL	1.5896 mL	3.1791 mL

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

BIOLOGICAL ACTIVITY

Description

Diphenyleneiodonium chloride is a NADPH oxidase (NOX) inhibitor and also functions as a TRPA1 activator with an EC_{50} of 1 to 3 μM. Diphenyleneiodonium chloride selectively inhibits intracellular reactive oxygen species.

IC_{50} & Target

NOX[1]
EC50: 1 to 3 μM (TRPA1)[1]

In Vitro

Diphenyleneiodonium chloride is a NADPH oxidase (NOX) inhibitor and also functions as a TRPA1 activator with an EC_{50} of 1 to 3 μM. Application of Diphenyleneiodonium chloride to HEK-TRPA1 cells at a concentration ranges of 0.03 to 10 μM effectively induces a Ca^{2+} response. However, Diphenyleneiodonium chloride fails to evoke a Ca^{2+} response in control HEK cells, even at a relatively high dose of 10 μM[1]. When Diphenyleneiodonium chloride is included in the co-cultures, lipopolysaccharide (LPS)-induced preOL apoptosis is significantly inhibited. Treatment with Diphenyleneiodonium chloride is found to significantly attenuate the LPS-induced O_2^- production by 2.0-fold, reducing it to within 27% of the controls[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Intraplantar injection of 2 mM Diphenyleneiodonium chloride to the hindpaw causes licking or biting behavior[1].

	Diphenyleneiodonium chloride treatment immediately or 24 h after lipopolysaccharide (LPS) injection significantly attenuates the LPS-induced loss of O4 positive cells. Treatment with Diphenyleneiodonium chloride either immediately or 24 h after LPS injection significantly ameliorates the LPS-induced disorganization of the white matter nerve fibers. However, treatment with DPI 48 h after LPS injection does not appear to correct the LPS-induced white matter damage. DPI treatment either immediately or 24 h after LPS injection significantly reduces the accumulation of both gp91phox and p67phox in the membrane fraction[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Purified microglia and preOLs are co-cultured using a Transwell culture system. Co-cultured cells are divided into three groups: control, lipopolysaccharide (LPS)-activated, and LPS plus Diphenyleneiodonium chloride. Microglia are cultured in Transwells over established preOL layers and exposed to either LPS (100 ng/mL), LPS+Diphenyleneiodonium chloride (10 μM) or untreated. The medium supernatants and cellular protein fractions from the co-cultures are then collected for further analysis after 48 h incubation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	The ddy mice (6 to 7 wk of age) are individually placed in transparent cages for 30 min before experiments. An intraplantar injection of 10 μL Diphenyleneiodonium chloride (2 mM, solvent: Kolliphor EL with 20% DMSO) is then injected into the right hindpaw with or without intraperitoneal administration with HC030031 (300 mg/kg at 0.5 h prior to injection of Diphenyleneiodonium chloride; solvent: saline with 0.5% methyl cellulose). The time spent licking or biting the injected paw is recorded for 45 min after injection[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancer Cell. 2021 Mar 8;39(3):423-437.e7.
- Adv Healthc Mater. 2021 Dec 3;e2102439.
- Sci Total Environ. 2023 Jul 26;165821.
- Biomed Pharmacother. 2020 Jan;121:109615.
- J Transl Med. 2023 Mar 25;21(1):218.

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REFERENCES

[1]. Suzuki H, et al. The NADPH oxidase inhibitor diphenyleneiodonium activates the human TRPA1 nociceptor. Am J Physiol Cell Physiol. 2014 Aug 15;307(4):C384-94.

[2]. He YF, et al. Diphenyleneiodonium protects preoligodendrocytes against endotoxin-activated microglial NADPH oxidase-generated peroxynitrite in a neonatal rat model of periventricular leukomalacia. Brain Res. 2013 Jan 25;1492:108-21.

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