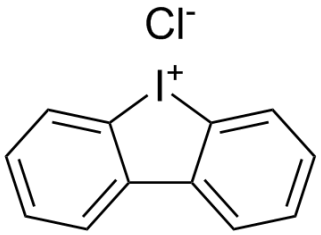
Diphenyleneiodonium chloride

Cat. No.:	HY-100965
CAS No.:	4673-26-1
Molecular Formula:	$C_{12}H_8ClI$
Molecular Weight:	314.55
Target:	TRP Channel; NADPH Oxidase
Pathway:	Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 6 mg/mL (19.07 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Mass / Solvent / 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.

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].

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].

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

- **Toxicol Appl Pharmacol**. 2019 Mar 1;366:83-95.
- **Am J Physiol Heart Circ Physiol**. 2018 Mar 1;314(3):H580-H592.

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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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