

# Diphenyleneiodonium chloride

Catalog No: tcsc0020645



## Available Sizes

**Size:** 10mg

**Size:** 50mg

**Size:** 100mg



## Specifications

**CAS No:**

4673-26-1

**Formula:**

$C_{12}H_8Cl$

**Pathway:**

Membrane Transporter/Ion Channel

**Target:**

TRP Channel

**Purity / Grade:**

>98%

**Solubility:**

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

**Alternative Names:**

DPI

**Observed Molecular Weight:**

314.55

## Product Description

Diphenyleneiodonium chloride is a **NADPH oxidase (NOX)** inhibitor and also functions as a **TRPA1** activator with an **EC<sub>50</sub>** of 1 to 3  $\mu\text{M}$ .

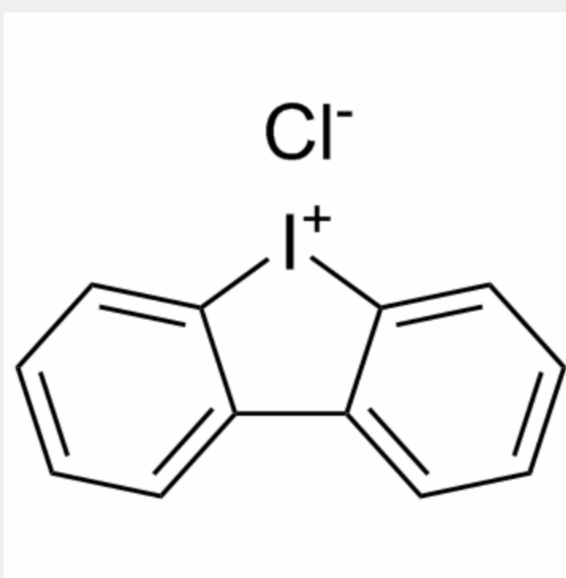
IC<sub>50</sub> & Target: NOX<sup>[1]</sup>

EC<sub>50</sub>: 1 to 3  $\mu\text{M}$  (TRPA1)<sup>[1]</sup>

**In Vitro:** Diphenyleneiodonium chloride is a NADPH oxidase (NOX) inhibitor and also functions as a TRPA1 activator with an EC<sub>50</sub> of 1 to 3  $\mu\text{M}$ . Application of Diphenyleneiodonium chloride to HEK-TRPA1 cells at a concentration ranges of 0.03 to 10  $\mu\text{M}$  effectively induces a  $\text{Ca}^{2+}$  response. However, Diphenyleneiodonium chloride fails to evoke a  $\text{Ca}^{2+}$  response in control HEK cells, even at a relatively high dose of 10  $\mu\text{M}$ <sup>[1]</sup>. 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  $\text{O}_2^-$  production by 2.0-fold, reducing it to within 27% of the controls<sup>[2]</sup>.

**In Vivo:** Intraplantar injection of 2 mM Diphenyleneiodonium chloride to the hindpaw causes licking or biting behavior<sup>[1]</sup>.

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<sup>[2]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!