Imidacloprid

Cat. No.: HY-B0838

CAS No.: 138261-41-3

Molecular Formula: $C_9H_{10}ClN_5O_2$

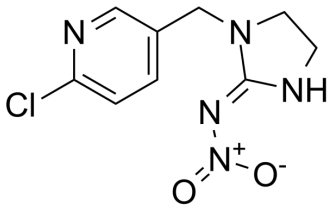
Molecular Weight: 255.66

Target: Others

Pathway: Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (391.14 mM)

H_2O : 1 mg/mL (3.91 mM; ultrasonic and warming and heat to 80°C)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.9114 mL	19.5572 mL	39.1144 mL
5 mM	0.7823 mL	3.9114 mL	7.8229 mL
10 mM	0.3911 mL	1.9557 mL	3.9114 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (9.78 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.78 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.78 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Imidacloprid is an effective and widely used neonicotinoid pesticide to control pests of cereals, vegetables, tea and cotton.

In Vitro

Insulin stimulated glucose uptake is reduced by imidacloprid in adipocytes (3T3-L1), hepatocytes (HepG2), and myotubes (C2C12) cell culture models. Treatment with imidacloprid reduced phosphorylation of protein kinase B (AKT), one of the major regulators of insulin signaling, without changing overall AKT expression. imidacloprid reduced phosphorylation of ribosomal S6 kinase (S6K), which is a downstream target of AKT and also a feed-back inhibitor of insulin signaling[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Imidacloprid in high doses causes deterioration in cognitive functions particularly in infant rats, and this deterioration may be associated with changes in the expressions of related genes. Learning activities are diminished significantly at 2 and 8 mg/kg doses in the infant model groups and at 8 mg/kg dose in adult rats. Also, expression levels of GRIN1, SYP and GAP-43 are found to be insignificantly altered[2]. Early developmental exposure to imidacloprid has both early-life and persisting effects on neurobehavioral function in zebrafish. In larvae, developmental imidacloprid exposure at both doses significantly decreased swimming activity. In adolescent and adult fish, developmental exposure to imidacloprid significantly decreased novel tank exploration and increased sensorimotor response to startle stimuli[3]. Decrease in the body weight gain is observed at 20 mg/kg/day and at necropsy the relative body weights of liver, kidney and adrenal is also significantly increased at this dose level. The spontaneous locomotor activity is also decreased at highest dose exposure where as there are no significant changes in hematological and urine parameters. The brain, liver and kidney of rats exposed with high dose of imidacloprid has showed mild pathological changes[4]. Imidacloprid at 20 mg/kg has produced significant changes in SOD, CAT, GPx, GSH, LPO in liver; SOD, CAT, and GPx in brain and LPO in kidney[5]. Imidacloprid at high dose, specifically suppresses cell-mediated immune response as is evident from decreased DTH response and decreased stimulation index of T-lymphocytes to PHA. Prominent histopathological alterations are also observed in spleen and liver. Histopathological analysis of footpad sections of mice reveal dose-related suppression of DTH response[6].

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PROTOCOL

Animal Administration [4][6]

Rats: Adult females are divided into four groups. One group is served as control and is given corn oil as vehicle through gavage. Three groups are given 5, 10, and 20 mg/kg/day imidacloprid to female rats for 90 days. Body weight, food consumption and clinical signs of toxicity are recorded throughout the period of experiment. Urine is collected at initial and 90 days for urine analysis. Individual animals from each group are weighed weekly and body weight is recorded[4].

Mice: Imidacloprid is administered orally daily at 10, 5, or 2.5mg/kg over 28 days. Specific parameters of humoral and cellular immune response including hemagglutinating antibody (HA) titer to sheep red blood cells (SRBC; T-dependent antigen), delayed type hypersensitivity (DTH) response to SRBC, and T-lymphocyte proliferation in response to phytohemagglutinin (PHA) are evaluated[6].

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CUSTOMER VALIDATION

- Environ Pollut. 25 August 2021, 118036.
- Aquat Toxicol. August 2022, 106204.
- J Sep Sci. 2019 Jul;42(14):2455-2465.

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REFERENCES

[1]. Kim J, et al. Imidacloprid, a neonicotinoid insecticide, induces insulin resistance. J Toxicol Sci. 2013;38(5):655-60.

[2]. Kara M, et al. Insecticide imidacloprid influences cognitive functions and alters learning performance and related gene expression in a rat model. Int J Exp Pathol. 2015 Oct;96(5):332-7.

[3]. Crosby EB, et al. Neurobehavioral impairments caused by developmental imidacloprid exposure in zebrafish. Neurotoxicol Teratol. 2015 May-Jun;49:81-90.

[4]. Bhardwaj S, et al. A 90 days oral toxicity of imidacloprid in female rats: morphological, biochemical and histopathological evaluations. Food Chem Toxicol. 2010 May;48(5):1185-90.

[5]. Kapoor U, et al. Effect of imidacloprid on antioxidant enzymes and lipid peroxidation in female rats to derive its No Observed Effect Level (NOEL). J Toxicol Sci. 2010 Aug;35(4):577-81.

[6]. Badgujar PC, et al. Immunotoxic effects of imidacloprid following 28 days of oral exposure in BALB/c mice. Environ Toxicol Pharmacol. 2013 May;35(3):408-18.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA