Product Data Sheet

Product Name: Imidacloprid
Cat. No.: GC30895

Chemical Properties

Cas. No.	138261-41-3		
SMILES	O=[N+](/N=C1NCCN\1CC2=CC=C(Cl)N=C2)[O-]		
Formula	$C_9H_{10}ClN_5O_2$	M.Wt	255.66
Solubility	DMSO : ≥ 300 mg/mL (1173.43 mM)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Imidacloprid		

Protocol

Cell experiment [1]:

Cell lines	WPM-Y.1 cells (human prostate epithelial cell line)
Preparation Method	WPM-Y.1 cells were maintained in DEMEM supplemented with 10% fetal bovine serum and 1% penicillin/streptomycin at 37°C in a 5% CO_2 atmosphere. Cells were seeded at a concentration of 1×10^4 cells/ml and treated with Imidacloprid (0.46-23μM) for 24 hours.
Reaction Conditions	0.46-23μM; 24h

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Applications	Imidacloprid significantly decreased cell viability, increased lactate dehydrogenase (LDH) activity and malondialdehyde (MDA) level, decreased glutathione (GSH) content and glutathione-S-transferase (GST) activity, and increased activities of catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR). Transmission electron microscopy showed significant defects including organelle destruction, irregular nuclear membrane, chromatin migration, increased vacuoles, and reduced mitochondria.

Animal experiment [2]:

Animal models	Female C57BL/6J mice
Preparation Method	Mice were fed a low-fat (4% w/w) or high-fat (20% w/w) diet containing Imidacloprid (0.06, 0.6, or 6mg/kg/day) for 12 weeks. Water and diet were given ad libitum. At the end of the experiment, mice were fasted for 4h before being sacrificed by CO_2 asphyxiation.
Dosage form	0.06, 0.6, or 6mg/kg/day; i.p.; dietary administration; 12 weeks
Applications	Imidacloprid significantly enhanced high-fat diet-induced weight gain and adiposity, increased serum insulin levels, and inhibited AMPKα activation in white adipose tissue. Imidacloprid increased serum leptin levels and adipocyte size in high-fat diet-fed mice.

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References:
[1] Abdel-Halim KY, Osman SR. Cytotoxicity and Oxidative Stress Responses of Imidacloprid and Glyphosate in Human Prostate Epithelial WPM-Y.1 Cell Line. J Toxicol. 2020 Dec 8;2020:4364650.
[2] Sun Q, Qi W, Xiao X, et al. Imidacloprid Promotes High Fat Diet-Induced Adiposity in Female C57BL/6J Mice and Enhances Adipogenesis in 3T3-L1 Adipocytes via the AMPKα-Mediated Pathway. J Agric Food Chem. 2017 Aug 9;65(31):6572-6581.

Background

Imidacloprid is a neonicotinoid insecticide with systemic activity. Imidacloprid acts as a false neurotransmitter by binding to nicotinic acetylcholine receptors in the insect nervous system, disrupting normal nerve transmission, keeping the nerve channels open, causing abnormal excitation, paralysis, and death in insects. Imidacloprid can be used in research related to neurotoxicity, cytotoxicity, and metabolic interference[1-4].

In vitro, Imidacloprid (0.46-23μM) was used to treat human prostate epithelial WPM-Y.1 cells for 24 hours. Imidacloprid significantly induced cytotoxicity, oxidative stress, increased lactate dehydrogenase (LDH) activity, elevated malondialdehyde (MDA) levels, decreased glutathione (GSH) content, reduced glutathione-S-transferase (GST) activity, increased activities of catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR), and caused ultrastructural defects such as organelle destruction, irregular nuclear membrane, chromatin migration, increased vacuoles, and reduced

mitochondria[5]. Imidacloprid (1-500μg) was used to treat L-929 fibroblast cells for 24 hours. Imidacloprid (500μg) significantly increased L-929 cell lactate dehydrogenase (LDH) levels, decreased glutathione (GSH) levels, and had no significant effect on acetylcholinesterase (AChE) levels[6].

In vivo, Imidacloprid (0.06, 0.6, or 6mg/kg/day) was administered via diet for 12 weeks to female C57BL/6J mice fed a low-fat (4% w/w) or high-fat (20% w/w) diet. Imidacloprid significantly enhanced high-fat diet-induced weight gain and obesity and increased serum insulin levels[7]. Imidacloprid (5 and 20mg/kg/day) was administered via repeated oral gavage for 28 days to female KM mice. Imidacloprid (20mg/kg/day) significantly caused hippocampal formation cell band loss, liver vascular swelling, disordered hepatic cord arrangement, hepatocyte necrosis and nuclear pyknosis, and increased plasma ALT, AST, and AKP levels; Imidacloprid (5mg/kg/day) significantly altered the metabolic profiles of the hippocampus and liver, involving lipid metabolism, amino acid metabolism, nucleotide metabolism, carbohydrate metabolism, and energy metabolism pathways[8].

References:
[1] Gautam P, Kumar Dubey S. Biodegradation of imidacloprid: Molecular and kinetic analysis. Bioresour Technol. 2022 Apr;350:126915.
[2] Bhende RS, Dafale NA. Insights into the ubiquity, persistence and microbial intervention of imidacloprid. Arch Microbiol. 2023 May 2;205(5):215.
[3] Fouad MR, Abdel-Raheem SAA. An overview on the fate and behavior of imidacloprid in agricultural environments. Environ Sci Pollut Res Int. 2024 Nov;31(52):61345-61355.
[4] Mikolić A, Karačonji IB. Imidacloprid as reproductive toxicant and endocrine disruptor: investigations in laboratory animals. Arh Hig Rada Toksikol. 2018 Jun 1;69(2):103-108.
[5] Abdel-Halim KY, Osman SR. Cytotoxicity and Oxidative Stress Responses of Imidacloprid and Glyphosate in Human Prostate Epithelial WPM-Y.1 Cell Line. J Toxicol. 2020 Dec 8;2020:4364650.
[6] Sevİm Ç, Taghİzadehghalehjoughİ A, Kara M. In Vitro Investigation of the Effects of Imidacloprid on AChE, LDH, and GSH Levels in the L-929 Fibroblast Cell Line. Turk J Pharm Sci. 2020 Oct;17(5):506-510.
[7] Sun Q, Qi W, Xiao X, et al. Imidacloprid Promotes High Fat Diet-Induced Adiposity in Female C57BL/6J Mice and Enhances Adipogenesis in 3T3-L1 Adipocytes via the AMPKα-Mediated Pathway. J Agric Food Chem. 2017 Aug 9;65(31):6572-6581.

[8] Zheng M, Qin Q, Zhou W, et al. Metabolic disturbance in hippocampus and liver of mice: A primary response to imidacloprid exposure. Sci Rep. 2020 Mar 31;10(1):5713.