

Developed and designed by Thanal Conservation Action Information and Trading Network Private Limited





HIGHLY HAZARDOUS PESTICIDES

It is a systemic, broad-spectrum insecticide used to control sucking and

chewing insects such as aphids and beetles on vegetable crops. It is used for foliar and seed treatments, indoor and outdoor insect control, home

gardening and for control of fleas on pets. It is acutely toxic and can induce cytotoxic, carcinogenic, and reproductive toxic effects in

IMIDACLOPRID

(Insecticide)

IUPAC Name- (NE)-N-[1-[(6-chloropyridin-3-yl) methyl] imidazolidin-2-ylidene] nitramide

CAS NO: 138261-41-3

Substance Group-

Neonicotinoid Insecticide

Trade names - Gaucho 600 FS and Gaucho 70 WS(Bayer), Imida70WS (Apex Gro chemicals) Confidor (Bayer), Seamer (DuPont), Tatamida (Rallis), Jumbo (PI Ind.), Imidacel (Excel), Imida Gold (UPL), Atom (Indofil), Media (Dhanuka), Nagarjuna Mida (NACL), Victor (IIL), Kemida (Cheminova), Sumida (SuperCSL), Parrymida/Suzu (Coromandel),

Classification- (WHO) - Class II (Moderately hazardous)

Banned Countries- It is banned in 29 countries including UK Switzerland, Indonesia and Palestine

Mode of Action: It is a contact poison that works by interfering with the transmission of stimuli in the insect nervous system. Imidacloprid mimics the action of acetylcholine, but it is not degraded by the enzyme

animals. It has no specific antidote.

General properties

acetylcholinesterase

It is a neonicotinoid pesticide belonging to the chemical family, chloronicotinyl nitroguanidine

It is a synthetic derivative of nicotine, an alkaloid found in plants like tobacco

It is a colourless crystal with a distinguished smell

Imidacloprid is a nicotinic acetylcholinesterase inhibitor

It is highly soluble and non-volatile

Formulations: 9 (Imidacloprid 00.03 % w/w Gel, Imidacloprid 00.30 % GR, Imidacloprid 02.15 % w/w Gel, Imidacloprid 17.1 %

w/w SL, Imidacloprid 17.80 % SL, Imidacloprid 30.50 % m/m SC, Imidacloprid 48 % FS, Imidacloprid 70 % WG and Imidacloprid 70 % WS)

GHS Hazard Statements-

GHS Signal word: WARNING

H302: Harmful if swallowed (Acute toxicity, oral)

H400: Very toxic to aquatic life (Hazardous to the aquatic environment, acute hazard)

H410: Very toxic to aquatic life with long-lasting effects (Hazardous to the aquatic environment, long-term hazard)

Exposure Route: Exposure by inhalation and ingestion

Residues-In the US, Imidacloprid residues were found in 80% of bananas, 76% of cauliflower and 72% of spinach. Imidacloprid has been found in many foods including chestnuts, ginger, vegetables, potatoes, tea, wine, fruit, and fruit juices.

Rice and vegetable samples collected from 14 centres in India in 2019 have shown an exceeding level of Imidacloprid in the Annual project report of All India Network Project on Pesticide Residues, Indian Agricultural Research Institute 2018-19. Nonapproved uses were also noted.

Imidacloprid was detected in 38 samples of fruits, vegetables, and cereals in a total of 250 samples-including fruits, fruit juices, and baby foods (50 samples each), vegetables (70 samples), and cereals (30 samples) from Lucknow, India (Kapoor U et al, 2013)

The Maximum Residue Limit (MRL) prescribed for Imidacloprid according to Food safety and standards (Contaminants, toxins, and residues) Regulations, 2011 is given in mg/kg.

Okra	-2.0
Citrus fruits	-1.0
Sugarcane	-0.1
Cotton seed	-0.05
Meat/Milk	-0.1
Soybean	-3.0
Chilli	-0.3
Grapes/Tomato	-1.0
Cucumber	-1.0

Regulatory status:

International regulation: It is not approved by U.K COPR regulation and EU regulation (1107/2009)

European Union legislative has lowered the MRL value of Imidacloprid to the specific limit of determination (LOD) for pecans, bananas, tomatoes, sweet peppers/bell peppers, aubergines/eggplants, cucumbers, gherkins, courgettes and products of animal origin. MRLs for escaroles/broad-leaved endives are identified as a risk for consumers stated in regulation 2021/1881 of 26 October 2021

The presence of imidacloprid in groundwater, in California has resulted in imidacloprid being subjected to groundwater monitoring requirements and DPR's program of continuous evaluation in 2021

Imidacloprid was voluntarily withdrawn in 2011, under pressure from the state government of California, from use on almonds, a major crop for bees.

National regulation:

Imidacloprid is Toxic-labelled yellow colour (Moderately Hazardous)

It is approved for 19 crops nationally which are cotton, rice, okra, cucumber, tomato, potato, sunflower, sorghum, pearl millet, maize, wheat, chilli, sugarcane, mustard, mango, citrus, ground nut, grapes, and soybean and are used to control pests Bollworm, Diamond back moth, Jassids, Aphids, Thrips, Whiteflies, Semi looper, Leaf miner, stem borer, Brown plant hopper, White backed plant hopper, Green leaf hopper etc.

It has a volume consumption of 317.17 metric ton in the year 2021 in India

Health Hazards

Acute toxicity: Imidacloprid is moderately toxic by ingestion, variable toxicity by inhalation, and very low toxicity by dermal contact: Toxicity Categories - oral II, dermal IV, inhalation I (aerosol), IV (dust)

Symptoms following exposure to agricultural formulations of **Imidacloprid** have included reduced activity, lack of coordination, tremors, diarrhoea, and weight loss. Nausea. vomiting, dizziness, disorientation, agitation, incoherence, breathlessness, and excessive sweating have resulted from inhalation and dermal exposure. Symptoms following ingestion include drowsiness, dizziness, disorientation, fever, sweating, increased vomiting, heart and respiratory rates. Mild cases of dermatitis from veterinary use of imidacloprid were also noted

Poisoning studies of 163 patients from Thailand concluded that patients with imidacloprid-only exposure developed mild toxicity. Patients who ingested large exhibited quantities signs including cardiovascular effects, central nervous system effects, dyspnea, and diaphoresis (Acute imidacloprid poisoning Thailand)

Oral LD50 values in rats were estimated to be 450 mg/kg for both sexes. The dermal LD50 in rats was estimated at greater than 5000 mg/kg (The Pesticide Manual, 2006)

The acceptable daily intake (ADI) was 0.06 mg/kg bw/day based on the chronic rat study, and the acute reference dose (ARfD) was 0.08

mg/kg bw/day based on the 90-day dog study (EFSA, 2008)

Chronic toxicity: Beagles when fed with imidacloprid showed reduced food intake in the highest dose group. Females in this group exhibited increased plasma cholesterol concentrations at 13 and 26 weeks. Both sexes exhibited increased cytochrome P450 activity in the liver and increase in liver weights at the end of the study (Allen T. R, 1989)

Carcinogenicity: The U.S. EPA has classified imidacloprid into Group E, with no evidence of carcinogenicity, based on studies with rats and mice

Reproductive Toxicity: Rats when fed imidacloprid during their pregnancies showed that, on day 21 of the pregnancy, rats at the highest doses showed reduced embryo development and signs of maternal toxicity. In addition, wavy ribs were observed in the foetuses (Vohra P, Khera KS, 2015)

On day 28 of pregnancy in imidacloprid-fed rabbits, researchers noted maternal toxicity including death in the highest dose group, and the animals that survived in this group carried embryos with reduced rates of growth and bone ossification. In some of these rabbits, the young were aborted or resorbed (Becker H et al, 1988)

Genotoxicity: Imidacloprid induced a dose-related increase in the micronucleus frequency in bone marrow chromosome aberration assay and micronucleus test in Wistar rats (Karabay NU and Oguz MG, 2005)

Endocrine disruption:

Imidacloprid was included in the draft list of initial chemicals for screening under the U.S. EPA Endocrine Disruptor Screening Program (EDSP).

Neurotoxicity: An in vitro study suggested that excitation or desensitisation of nicotinic acetylcholine receptors (nAChRs) occurs by Imidacloprid, which might affect developing mammalian nervous systems as occurs with nicotine (Kimura-Kuroda et al., 2012).

The panel for Scientific Opinion on the developmental neurotoxicity potential of acetamiprid and imidacloprid, 2013 concluded that Imidacloprid may affect neuronal development and function

It was studied that, gestational exposure to a single large, nonlethal, dose of imidacloprid produces significant neurobehavioral deficits and an increased expression of GFAP in several brain regions of the offspring of rats (Abou-Donia, M. B et al, 2008)

Mutagenicity: Ziram was found to be mutagenic in bacteria. Plate incorporation assay with S. typhimurium demonstrated direct mutagenicity of ziram (Franekic J et al, 1994)

Poisoning Data

The Maharashtra government banned Imidacloprid along with other pesticides in 2017 following Yavatmal poisoning.

Imidacloprid was responsible for 8 admissions in Warangal poisoning, in 2002.

A case report of a 25-year-old following suicidal ingestion of 30.5% imidacloprid exhibited severe hypoxia, bilateral infiltrates in the lower lung, metabolic acidosis and hypotension.

Antidote- No specific antidote is available

Environmental fate and effects: It is metabolised by photodegradation from soil and water surfaces.

It is moderately to highly persistent in soil under aerobic conditions (half-life of 40-997 days) and shows moderate to high persistence in natural sediment, and water systems (half-life of 30-162 days). It has high mobility in soil and has the potential to leach into groundwater

It has a low risk of bioaccumulation

EFSA in 2013 stated that the spray applications of imidacloprid pose a high risk to bees. The spray application of imidacloprid will cause severe impacts on nontarget arthropods in the infield and off-field areas.

Ecotoxicity Mammals- Moderate acute toxicity

Birds- High acute toxicity

Earthworms- Moderate acute toxicity

Honeybees- High acute toxicity

Fish - Moderate acute toxicity

Aquatic crustaceans- Moderate acute toxicity

Aquatic invertebrates- High acute toxicity

Alternate Pest management

Sustainable ecological solutions to replace chemical Pesticides include the use of biopesticides

and numerous cultural, mechanical and biological solutions to pest control, as well as natural sprays that can be used depending on the pest and the situation that relies on the utilization of agroecological practices.

Notes on HHPs

Highly Hazardous pesticides or HHPs are a group of pesticides, that can pose serious risks to humans and cause irreversible damage to the environment. They are listed in international conventions and are banned in many countries. The handling and use of these HHPs are beyond the safety level of PPE as stated by SAICM.

HHPs upon exposure enter the body through food, inhalation, or dermal contact. These pesticides cause lethal effects, especially when exposed for the long term. It includes acute toxicity (Headache, Nausea, Vomiting etc) to Chronic hazards (Gene mutations, Cancer, Reproductive dysfunction etc). Farmers, applicators, and their families are mostly exposed to pesticides. The increased closeness of residents to farming areas worsens the situation and their exposure can occur under deplorable conditions, such as handling, storing, mixing, loading, spraying, disposing, and washing pesticide containers or pesticide-soaked clothes.

Women are the most affected by the ill effects of HHP use, as they have a higher proportion of hormone-sensitive tissues, fats, and primary reproductive tasks. HHPs can cause birth defects, miscarriage, early onset of puberty, sexual maturation, infertility, and abortions in female children. Children are exposed to the HHP-contaminated environment as they consume more air, water and food per unit of body weight. They have a higher metabolism and their immunity and developing functions are compromised at a young age.

REFERENCES

- 1. Abou-Donia, M. B., Goldstein, L. B., Bullman, S., Tu, T., Khan, W. A., Dechkovskaia, A. M., & Abdel-Rahman, A. A. (2008). Imidacloprid induces neurobehavioral deficits and increases expression of glial fibrillary acidic protein in the motor cortex and hippocampus in offspring rats following in utero exposure. Journal of toxicology and environmental health. Part A, 71(2), 119–130.
- 2. Allen, T. R.; Frei, T.; Leutkemeier, H.; Vogel, O.; Biedermann, K.; Wilson, J. A 52-week oral toxicity (feeding) study with NTN 33893 technical in the dog. Unpublished Report no. R 4856, 1989, amendment no. R 4856A, 1992, submitted to WHO by Bayer AG, Mannheim, Germany. INCHEM Toxicological Evaluations: Imidacloprid; International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, 1989.
- 3. Becker, H.; Vogel, W.; Terrier, C. Embryotoxicity study (including teratogenicity) with NTN 33893 technical in the rabbit. Unpublished Report no. R 4583, 1988, submitted to WHO by Bayer AG, Mannheim, Germany. INCHEM Toxicological Evaluations: Imidacloprid; International Programme on Chemical Safety, World Health Organization: Geneva, Switzerland, 1988b.

- 4. Cox C. 2001. Imidacloprid. J Pestic Reform 21(1):15-21.
- 5. Draft list of initial pesticide active ingredients and pesticide inerts to be considered for screening under the Federal Food, Drug, and Cosmetic Act. Fed. Regist. June 18, 2007, 72 (116), 33486-33503.
- 6. EFSA. 2008. Conclusion regarding the peer review of the risk assessment of the active substance imidacloprid. EFSA Scientific Report (2008) 148,1-120. European Food Safety Authority. http://www.efsa.
- 7. Gervais JA, Luukinen B, Buhl K, Stone D. 2010. Imidacloprid Technical Fact Sheet; National Pesticide Information Centre, Oregon State University Extension Services. http://npic.orst.edu/factsheets/imidacloprid.pdf.
- 8. Imidacloprid: Pesticide Tolerances for Emergency Exemptions. Fed. Regist. October 12, 2005, 70 (196), 59268-59276.
- 9. Kapoor U, Srivastava MK, Srivastava AK, Patel DK, Garg V, Srivastava LP. Analysis of imidacloprid residues in fruits, vegetables, cereals, fruit juices, and baby foods, and

daily intake estimation in and around Lucknow, India. Environ Toxicol Chem. 2013 Mar;32(3):723-7.

- 10. Karabay NU, Oguz MG. 2005. Cytogenetic and genotoxic effects of the insecticides, imidacloprid and methamidophos. Genet Mol Res 4(4):653-62.
- 11. Kimura-Kuroda J, Komuta Y, Kuroda Y, Hayashi M, Kawano H. Nicotine-like effects of the neonicotinoid insecticides acetamiprid and imidacloprid on cerebellar neurons from neonatal rats. PLoS One. 2012;7(2):e32432
- 12. Thyssen, J.; Machemer, L. Imidacloprid: Toxicology and Metabolism. Nicotinoid Insecticides and the Nicotinic Acetylcholine Receptor; Yamamoto, I.; Casida, J. E., Eds.; Springer-Verlag: Tokyo, 1999; Chapter 9, pp 213-222.
- 13. Tomlin, C. D. S. The Pesticide Manual, A World Compendium, 14th ed.; British Crop Protection Council: Surry, England, 2006; pp 598-599.
- 14. Vohra P, Khera KS. A Three Generation Study with Effect of Imidacloprid in Rats: Biochemical and Histopathological Investigation. Toxicol Int. 2015 Jan-Apr;22(1):119-24. doi: 10.4103/0971-6580.172270. PMID: 26862272; PMCID: PMC4721159.

15. Tomlin, C. D. S. The Pesticide Manual, A World Compendium, 14th ed.; British Crop Protection Council: Surry, England, 2006; pp 598-599.

Web references

https://www.efsa.europa.eu/en/efsajournal/pub/3471

 $\frac{https://www.panna.org/sites/default/files/PAN\%20AP\%20pest}{icides-factsheet-hhps-neonicotinoids.pdf}$

https://agricoop.nic.in/sites/default/files/PPfinal2732015.pdf

 $\frac{https://www.dovepress.com/acute-imidacloprid-poisoning-in-thailand-peer-reviewed-fulltext-article-TCRM$

imfname_11087008.pdf (parlament.gv.at)

https://www.cdpr.ca.gov/docs/emon/grndwtr/imidacloprid/imidacloprid director's decision.pdf

https://www.fao.org/3/i4854e/i4854e.pdf