



# ChemicalWatch Factsheet

A Beyond Pesticides/ NCAMP Factsheet

## Rotenone

The widely used botanical insecticide rotenone (Derris™, Prentox™, Chem Fish™) is often used by home and commercial “organic” gardeners as an alternative to commercial “chemical” insecticides. First used on crops in 1848 in British Malaysia, commercial rotenone is extracted from the Peruvian cubé root (genus *Lonchocarpus*) in the U.S. and marketed by several firms for use on fruits, vegetables, forage crops, to kill fish, for lice and tick control, and other uses. Farmers and gardeners should be aware of rotenone’s potential hazards and toxicity.

Despite widespread use and findings that residues are persistent, all agricultural uses of rotenone were exempted from tolerances (the establishment of maximum legal residue levels) in 1955 by the Food and Drug Administration (FDA), and, therefore, most data requirements were waived. EPA, now responsible for the establishment of tolerances, is reevaluating the 1955 exemption and has issued a special Data Call-in notice for residue data.

Rotenone and rotenone resins are found in the roots of 68 species of legume plants, but most commercial supplies come from the South American cubé, the Malaysian der-

ris, and Brazilian tembo plants. The resins are not very soluble in water, and are usually used either as a dust or in an oil or kerosene solution, sometimes mixed with the quicker-acting pyrethrins and the synergist piperonyl butoxide. Dust formulations have also long been used on animals to control lice and ticks, and on humans for treatment of chiggers and scabies. Noxfire™ has been used

extreme toxicity to fish has been exploited by Amazonian Indian hunters for centuries, and by the U.S. Fish and Wildlife Service to clear “trash” fish in the restocking of lakes for sport fishing. However, rotenone is difficult to use selectively, and can cause severe reductions in populations of aquatic invertebrates as well.

Toxicologically, rotenone is a slow-acting nerve poison which acts by inhibiting respiratory metabolism in cells, essentially paralyzing affected insects. Specifically, rotenone interferes with the mitochondrial electron-transport system. In animals, it is very poorly absorbed by the gastro-intestinal tract, and is so irritating that it promptly induces vomiting. However, in prolonged feeding tests in rodents, rotenone

caused growth depression. Test animals fed dust formulations of rotenone developed muscle tremors, severe pulmonary and skin irritation from exposure to dust, severe hypoglycemia (low blood sugar), clonic convulsions, and respiratory depression resulting in death. EPA has no record of human fatalities or clinical poisoning reports.

EPA conducted a Pre-Special Review investigation of rotenone in 1975. This was triggered by data

### *chemicalWATCH* Stats:

**Chemical Class:** Botanical

**Use:** Piscicide

**Toxicity rating:** Highly (as an emulsified concentrate) or slightly toxic (all other forms)

**Signal Word:** Danger (emulsified concentrate) or Caution (all other forms)

**Health Effects:** Neurological toxin- linked to Parkinson’s Disease. May cause kidney and liver damage.

**Environmental Effects:** Very highly toxic to fish. Very toxic to birds and swine. Slightly toxic to wildfowl. Residues are persistent.

as a drench to control fire ants on lawns, and one formulation is registered as a mosquito larvicide.

Development of insect resistance is reported to have been rare. Rotenone is non-phytotoxic and unstable to sunlight, air and water, so applications lose their efficacy within a week.

It is acutely toxic to most mammals; oral rat LD50 is 132 mg/kg, very toxic to birds, fish and swine, but spares bees. The property of

indicating that rotenone could arrest cell multiplication and cause developmental abnormalities in frog eggs and chick embryos. Also, a controversial Spanish carcinogenicity study purported to find high incidences of mammary tumors in rats. In 1981, the Agency concluded that the Spanish study had major deficiencies, and that attempts by an EPA contractor and the National Toxicology program to duplicate it in two species of rats and one mouse species had failed to detect any increase in tumors over the control animals.

In 1980, the U.S. Fish and Wild-

life Service took on the burden of paying for further toxicological studies (chemistry, environmental fate, teratology, mutagenicity, metabolism, and residue studies in water and fish) in order to maintain rotenone's registration when it became clear, according to EPA, that "no industrial sponsors [were] willing to conduct or fund the research studies needed to obtain registrations by fishery managers and fish culturists. At best, the gross sales of one of the major fishery chemicals is less than \$500,000 per year."

Researchers have found that rotenone dust residues persist on

lettuce and tomatoes nearly twice as long as do wettable powders, with half-lives between 3 and 5 days, and that both the parent and major metabolite are stable to boiling in tomato homogenate. Still, despite outstanding questions of ecological and health effects, the use of rotenone is widely accepted under organic certification programs.

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**Update, November 2007:**

Rotenone's Reregistration Eligibility Decision (RED) was signed in March 2007. EPA determined that all piscicidal uses of rotenone were eligible for reregistration, since the Agency concluded that none of these uses posed any unreasonable risks or adverse effects to humans or the environment. Previously, rotenone was used in agricultural and residential settings, including common household garden products and for tick and lice control in pets, but these uses were voluntarily cancelled in 2006. Rotenone that has been naturally derived is listed as a "restricted substance" for organic agriculture by the Organic Materials Review Institute (OMRI) and may be used only in special circumstances with designated limitations. The EPA designates it is a restricted use pesticide (RUP), which means it can only be sold to and applied by certified applicators.

Rotenone has high acute toxicity (toxicity category I) via oral and inhalation routes of exposure, and effects include conjunctivitis, dermatitis, sore throat, and congestion. In the RED, EPA states that there is uncertainty about the neurotoxicity of rotenone, saying it cannot accurately quantify the effect at doses to which people may be exposed. Recent studies have associated exposure to rotenone with Parkinson's disease (PD), a neurological disorder. A number of research articles have shown that high doses of rotenone can cause PD-like symptoms in animals. While the etiology of PD remains unknown, Alam and Schmidt (2002), found that rotenone is capable of destroying dopaminergic neurons and inducing parkinsonian symptoms in rats. Radad et al. (2006), supported the fact that rotenone kills dopaminergic neurons in a cell culture-based study. However, Richter et al. (2007), found that rotenone treatment only induced behavioral effects and no pathological signs of PD in mice, but noted that genetically disposed mice may be more sensitive to the neurotoxic effects of rotenone.

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## Rotenone *chemicalWATCH* Factsheet Bibliography

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