

“A Review of Spinosad”

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Spinosad gets its name from the microbe that produces it, a soil-dwelling bacterium called *Saccharopolyspora spinosa*. This product may soon become a widely accepted alternative to the malathion sprays used today. Unlike malathion, which can kill insects that come in contact with it, spinosad kills mainly by ingestion. When applied at recommended rates, this product poses less risk than most insecticides to mammals, birds, fish, and beneficial insects. Spinosad is already approved for use on more than 100 crops including apples, almonds, citrus, eggplant, tomatoes, and cotton. (<http://www.ars.usda.gov>)

Discovery (<http://hortipm.tamu.edu/publications/Japbeetles.html>)

The product is produced by a microorganism, *Saccharopolyspora spinosa*, a rare actinomycete bacterium reportedly collected from soil in the Caribbean Island in 1982. It has not been found in nature since that time, and was subsequently described as a new species. The bacteria produces compounds (metabolites) while in a fermentation broth. Spinosad, was characterized in 1988 and since that time over 30 different compounds ("spinosyns") have been found. Further manipulation of these chemicals could yield even more compounds ("spinosoids").

Spinosad contains two chemicals, spinosyn A and spinosyn D. These are crystalline solids with low odor, no volatility, and with low water solubility. Soil sorption is moderately strong and they degrade primarily through photolysis. The half-life of these compounds on a plant leaf is about 2 days. When sprayed on leaves, the compounds have translaminar movement, moving from one leaf surface to the other through the leaf tissue. Phytotoxicity has not been observed at the 1x and 2x rates of the water-based formulations tested to date.

Target specificity

Conserve SC has a "moderate" spectrum of activity. It is most effective on chewing insects including beetles (particularly Chrysomelidae), caterpillars and sawfly larvae. In addition, leafmining flies, fungus gnat and shore fly larvae are also potential target pests. It appears to have good activity on thrips and sporadic activity against mite species. Although active against hymenoptera (honey bee $LC_{50} = 11.5$ ppm; *Encarsia formosa* $LC_{50} = 29.1$ ppm as topical treatments), toxicity of dry residues should be less toxic. No "flare-ups" of aphid populations have been observed after treatment. Furthermore, sucking insects are not affected unless very high concentrations are applied.

Mode of action

This insecticide causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotine acetylcholine receptors by a mechanism that is clearly novel and unique among known insect pest control products. Furthermore, it also has effects on GABA receptor function that may contribute further to its insect activity. The reason for extraordinary margins of selectivity between certain insects, mammals, and other non-target organisms is not fully understood.

In target organisms, the compound is 5 to 10 time more effective when ingested than when used as a contact insecticide. Thus, the chemical has little effect on sucking insects. Spinosad is considered to be a "fast-acting" insecticide. Death occurs in 1 to 2 days and there appears to be no recovery. Generally, treatment provides 7 to 14 days of control. Although spinosad is thought to have a novel mode of activity, resistance management is perceived to be an essential practice in perpetuating the long-term effectiveness of this insecticide.

Human Effects (Material Safety Data Sheet)

Acute toxicity:

A search of Medline and Toxline revealed no published incidences of human poisoning with spinosad. Spinosad has low acute toxicity in rats. The oral LD₅₀ in male rats is 3,738 mg/kg. The oral LD₅₀ in female rats is >5,000 mg/kg. According to an EPA factsheet, acute dermal doses in rabbits are >2,000 mg/kg. A Dow technical fact sheet gives >5,000 mg/kg. In any case acute toxicity through this route is low. The rat inhalation LC₅₀ is >5.18 mg/liter (EPA, 1997; Jachetta, 2001; Dow, 1997).

Metabolism:

Spinosad is rapidly absorbed and extensively metabolized in a rat. Within 48 hours of dosing, 60-80% of spinosad or its metabolites are excreted through urine or feces (EPA, 1997; Dow, 1997).

Chronic Toxicity:

13-week dietary studies showed no-effect levels of 4.98 mg/kg/day in dogs, 6 mg/kg/day in mice and 8.6 mg/kg/day in cats. No dermal or systemic toxicity occurred in a 21-day repeated dose dermal toxicity study in rabbits of 1,000 mg/kg/day. Based on these data, the EPA set the reference dose in humans at 0.0268 mg/kg/day. Presumably, daily doses of this amount would cause no harm (EPA, 1997),

Cancer and Developmental:

There was no evidence of carcinogenicity in two rodent species at all dosages tested. Mutagenic studies show no mutagenic activity. There were no development effects in rats and rabbits up to the highest dose tested. No effect levels were 10mg/kg/day. Neonatal effects at 100 mg/kg/day were attributed to maternal toxicity (EPA, 1997).

Eye: May cause slight eye irritation.

Skin: The LD₅₀ for skin absorption in rabbits is >5000 mg/kg. A single prolonged exposure is not likely to result in the material being absorbed through skin in harmful amounts. Prolonged exposure is not likely to cause significant skin irritation. Did not cause allergic skin irritation or reactions when tested in guinea pigs.

Ingestion: Single dose oral toxicity is extremely low. The oral LD₅₀ for rats and mice is >5000 mg/kg. No hazards anticipated from swallowing small amounts incidental to normal handling operations.

Inhalation: Single exposure to mist is not likely to cause adverse effects.

Systemic (other target organs) effects: In animals, has been shown to cause vacuolation of cells in various tissues and changes in blood and serum biochemistry. Dose levels producing these effects were many times higher than any dose levels expected from exposure due to use.

Teratology (Birth Defects): Spinosad did not cause birth defects in laboratory animals.

Reproductive Effects: For spinosad, in laboratory animal studies, effects on reproduction have been seen only at doses that produced significant toxicity to the parent animals.

Spinosad vs Malathion vs Phyloxine B

In coffee fields in Hawaii, the Agriculture Research Service (ARS) scientists compared spinosad to malathion insecticide and to phyloxine B, a dye that is also a promising alternative to malathion. Though malathion was the most effective, spinosad and phyloxine B gave impressive levels of control