Mole Cricket Sensory Perception of Insecticides

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Objectives:

1. Determine the type, location, and abundance of different sensilla on the antennae and mouthparts of S. vicinus and S. borellii.
2. Demonstrate the physiological effect of insecticides on the mole cricket nervous system and/or ability of mole crickets to detect chemical stimuli.
3. Demonstrate the behavioral response of mole crickets to sub-lethal insecticide doses.

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The more that mole crickets (Scapteriscus spp.) move in the soil, the more damage they cause to turfgrass. Ideally, the insecticides used against them should stop their tunneling movements and kill them quickly. However, insecticides like acephate (Orthene), bifenthrin (Talstar), fipronil (TopChoice), imidacloprid (Merit), bifenthrin plus imidacloprid (Allecctus), and indoxacarb (Advion) are neurotoxins, and their effect on mole cricket mobility is not well defined. They could either excite or inhibit the insect's nervous system, which could lead to more or less tunneling before an affected mole cricket dies. In addition, the amount of time it takes to kill a mole cricket is also unknown because they tend to die in the soil. So, if spot treatments of neuroexcitatory insecticides are made, rather than "wall to wall" applications, more tunneling could occur at the edge of treated areas as the insects try to escape.

This first test was done to determine if the previously mentioned insecticides would excite or inhibit mole cricket nervous systems. We recorded the spontaneous nerve cord activity of tawny mole crickets using a suction recording electrode. Adults were dissected and their intact nerve cords were exposed within the abdominal cavity. For each specimen, the recording was conducted in saline solution for the first 5 minutes to establish a baseline. Then 10 µL of an insecticide solution was added to the abdominal cavity and recording continued for another 15 minutes. Technical grade insecticides were used (in 0.04% solution of DMSO), and saline alone and saline mixed with solvent (0.04% DMSO) were the controls.

Acephate, bifenthrin, fipronil, imidacloprid, and bifenthrin plus imidacloprid all had a significant neuroexcitatory effect, but indoxacarb and its metabolite were neuroinhibitory. Bifenthrin, fipronil, and bifenthrin plus imidacloprid caused the strongest neuroexcitatory effects on spontaneous neural activity.

We determined and compared the toxic effect of the insecticides by estimating the time needed for a product to kill 50% of the tested insects (LT50) for acephate, bifenthrin, fipronil, imidacloprid, bifenthrin plus imidacloprid, indoxacarb, and the indoxacarb metabolite (DCJW). Technical grade (95% and higher of active ingredient) insecticides were injected into the thorax of intact tawny mole cricket adults and nymphs, and the insects were held in petri dishes with moist sand and a food source. Mole crickets injected with saline solution of solvent served as controls. Their behavior was observed and mortality was recorded every hour for the first 12 hours and every 4 hours for the following 7 days.

Bifenthrin, fipronil, and bifenthrin plus imidacloprid provided the fastest mortality (38.3, 35.5 and 10.3 hours for adults, and 9.5, 10.4 and 6.5 hours for nymphs, respectively). Bifenthrin, fipronil, indoxacarb and its metabolite kill nymphs significantly faster than they kill adults.

Behavioral changes were noticed after treatment with most of the insecticides in the injection assay. Tawny mole crickets became immobile within 30 seconds after being injected with imidacloprid or bifenthrin plus imidacloprid, within 2-3 minutes after treatment with bifenthrin alone, and 1-2 hours after fipronil injection. However, mole crickets partially recovered after imidacloprid treatment and could walk but not tunnel.

Acephate increased the spatial movement and tunneling activity of mole crickets compared to the mole crickets injected with solvent solution only. Indoxacarb caused trembling, erratic leg and wing movements, and the insects kicked or jumped if disturbed. These data and observations correspond with the data in earlier behavioral assays. Our future research will focus on the ability of mole cricket antennae to detect insecticides using an electroantennogram.

Summary Points

- All tested insecticides (except indoxacarb and its metabolite) caused neural excitation, so their use could lead to increased tunneling, especially at sublethal doses.
- Fipronil, bifenthrin, and bifenthrin plus imidacloprid killed mole cricket adults and nymphs faster than imidacloprid, indoxacarb, the indoxacarb metabolite (DCWJ), and acephate.
- Fipronil, bifenthrin, and bifenthrin plus imidacloprid immobilized mole crickets within an hour, completely disrupting their tunneling activity at the tested dose.
- Insects injected with acephate and then held in petri dishes had increased tunneling and crawling.
- Imidacloprid caused immediate knockdown (within 30 seconds), but although the insects partially recovered, they could still walk but not tunnel.