USE OF IGRs FOR PROTECTION OF STORED COMMODITIES FROM INDIAN MEAL MOTHS

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In stored product moths, juvenoid agonists mimic the action of the insect's juvenile hormone. The primary effect is to maintain the insect in the juvenile state by extending the larval growth period and preventing metamorphosis into a reproductively mature adult. In addition, juvenoid agonists can also have gonadotrophic effects such as stimulating egg laying and shortening adult longevity when applied to unmated females. The most effective compounds that came out of the early studies were methoprene and hydroprene, but, in the past few years they have been surpassed by two new compounds, fenoxycarb and pyriproxyfen. These juvenoid agonists are considerably more active than their predecessors in applications for stored product moths. Their greater effectiveness can be partially attributed to their increased stability to light and moisture, but in addition we have found that they have some unique effects on insect metabolism.

The efficacy of using juvenoid agonists for protection of stored commodities from moths has been demonstrated in several Laboratories. The application of these chemicals for protection of stored commodities has been limited, primarily because of cost considerations. The recent mandate eliminating the use of methyl bromide for protection of stored commodities prompted our reassessment of these chemicals for commodity protection. We have focused our recent studies on the actions of fenoxycarb and pyriproxyfen on embryonic and early larval development. This line of investigation was prompted by our earlier observation that almond moth larvae feeding on in-shell peanuts treated with low levels of fenoxycarb died shortly after hatching. This effect markedly reduced the damage to the commodity during storage when compared with other juvenoid agonists such as methoprene. The idea that some juvenoid agonists are effective during the early stages of development was reinforced by the observation (Grossniklaus-Burgin and Lanzrein, 1990) that the timing of juvenile hormone secretion during the first larval stadium of *Trichoplusia ni* was unique, differing from that in the other larval stadia. We decided to reexamine the effects of juvenoid agonists on embryogenesis and the early larval stages of the Indian meal moth so that we could more fully understand the action of these compounds on organ and tissue development.

The effects of fenoxycarb and pyriproxyfen on embryogenesis were tested by immersing eggs in an acetone solution of the juvenoid agonist and expelling them onto filter paper to remove excess liquid; total exposure time was 2 to 3

seconds. Egg hatch was prevented by applying this treatment during the first 18 hours of embryogenesis. However, egg hatch was not affected if the treatment was applied after 18 hours, up to the time of hatching at 64 hours. We concluded that the critical period for juvenoid agonist action on egg hatch was confined to the first 18 hours of embryogenesis. Pyriproxyfen was effective at doses as low as 0.5 ppm; fenoxycarb was effective at doses ranging down to 0.1 ppm. When doses were decreased to lower levels, egg hatch was not affected, but mortality was high when the larvae attempted to molt to the 2nd instar.

Examination of the developing embryo by light microscopy and scanning electron microscopy reveals that during the first two hours nuclear multiplication is the predominant activity. Cellularization is observed at 4 1/2 hours, with well-defined serosal and embryonic cells evident by 6 1/2 hours. By 18 hrs, the germ band has extended fully and is segmented, but dorsal closure has not begun. These observations indicate that the juvenoid agonist must be present before well-defined organ systems have developed in the embryo, but that the manifestation of the agonist's action only becomes apparent as organogenesis proceeds. Thus, it would appear that the juvenoid agonist interferes with one or more fundamental processes early in embryonic development that are required for the formation of larval organs and tissues during the latter half of embryogenesis.

These studies indicate that relatively low levels of fenoxycarb and pyriproxyfen can effectively interfere with embryogenesis when applied to the intact egg. Now the problem becomes one of delivery of the agonist to the egg at the time of laying. Placing freshly laid eggs on diet treated with either of the juvenoid agonists was not effective in preventing egg hatch. Our next approach was to topically treat the adults with a juvenoid agonist and observe if there was an effect on egg laying and egg hatch. We found that treating male moths and mating them with untreated females did not affect egg laying or egg hatch. However, when female moths were treated with either juvenoid agonist, egg laying was not impaired but egg hatch was prevented. This indicated that a maternal delivery system was possible. Since the adult moths do not feed, we had to find a way to dose the female with the juvenoid agonist. Exposing females to agonist vapors emanating from a filter paper cap on gallon jars was ineffective. However, when a screen barrier was removed and the female moths were allowed to walk on the treated paper cap, egg laying was normal but egg hatch was severely impaired. Under these conditions mating was not disrupted. The effective dosages on the filter paper disks ranged around 100 $\mu g/cm^2$ for the prevention of egg hatch.