PROTECTION OF STORED COMMODITIES WITH JUVENOID AGONISTS

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The primary effect of juvenile hormone (JH) in stored product moths is to maintain the insect in the juvenile state by extending the larval growth period and preventing their metamorphosis into a reproductively mature adult. In the adult, JH can have gonadotrophic effects such as stimulating egg laying and shortening adult longevity. Juvenoid agonists (JH_{Ag}) mimic the action of the insect's JH. The most effective compounds that came out of our early studies were methoprene and hydroprene, but, in the past few years they have been surpassed by two new compounds, fenoxycarb and pyriproxyfen. We have found the latter two JH_{Ag} are considerably more active than their predecessors in applications for stored product moths.

In our recent studies we have focused on the effects of fenoxycarb and pyriproxyfen during embryonic and early larval development. Intervention at these early stages of development would minimize damage to the commodity. This rationale was demonstrated in a pilot test where we observed that the progeny of almond moths infesting freshly harvested in-shell peanuts treated with low levels of fenoxycarb died either prior to or shortly after hatching. This effect markedly reduced the damage to the commodity during storage when compared to the control and even with other juvenoid agonists such as methoprene. Laboratory tests indicated that the Indian meal moth was similarly affected.
In order to develop an effective treatment using this technology, we needed to more fully understand the basis for the lethal action of the JH_{Ag} on embryonic and early larval development. Using the Indian meal moth, we found that topical application of either JH_{Ag}, fenoxycarb or pyriproxyfen, to eggs had deleterious effects on embryonic development and egg hatch. In these laboratory studies, an effective application consisted of immersing freshly layed eggs in an acetone solution of the agonist for less than 3 seconds. This application was most effective when applied during the initial 18 hours of embryogenesis. Mortality occurred later in embryogenesis or during subsequent larval development; longevity after treatment was dependent upon agonist concentration. Our observations indicated that these juvenoid agonists had the potential to be used effectively to minimize moth damage to stored commodities. The problem was how to deliver the agonist to the egg during the initial 18 hours of embryogenesis.

Topical application of female moths with either juvenoid agonist did not alter the number of eggs laid, but the egg

hatch was greatly reduced. Similar treatments of male moths were ineffective. Subsequently, we found that untreated females allowed to contact an agonist-treated surface over a 24-hour period laid nonviable eggs. We found also that eggs did not hatch when they were layed on an agonist-treated surface by untreated moths. Likewise, the transfer of freshly-layed eggs onto a treated surface also prevented hatch. The effectiveness of these treatments was increased appreciably when temperature was increased to 35° C.

Next, we examined the mechanism(s) underlying the lethal effects of fenoxycarb and pyriproxyfen during embryogenesis and the early larval stages so that we could more fully understand the action of these compounds on organ and tissue development. Histological studies revealed that during embryogenesis, JH_{Ag} prevented the closure of the midgut which normally occurs about 35 hours after the eggs are layed. Less predictably, abnormal development of other tissues was also observed, especially the tracheal system. However, we feel that the failure in midgut closure is the lesion primarily responsible for embryonic mortality.

At lower JH_{Ag} concentrations, mortality from failed midgut closure may be deferred until after larval hatching, occurring during the first larval stadium. Another failure frequently encountered when low JH_{Ag} concentrations are administered to either the egg or larva is the inability to shed the old larval cuticle. We found that the failed molts result from the inability to shed the old cuticle because incompletely digested chitin adheres it to the new cuticle.

We conclude that fenoxycarb and pyriproxyfen have the potential for protecting stored commodities from moth damage, especially when processed commodities can be stored in treated packages or
cartons. We are currently conducting larger scale tests to determine the efficacy of applying this technology for the protection of packaged commodities.