VII.8 Grasshopper Viruses

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Introduction

Insect poxviruses or "entomopoxviruses" (EPV's) infect insects from the following five insect orders: Coleoptera (beetles), Lepidoptera (moths and butterflies), Orthoptera (grasshoppers and crickets), Diptera (flies), and Hymenoptera (bees and wasps). The grasshopper EPV's are found in the genus Entomopoxvirus B, which also includes viruses from Lepidoptera and Orthoptera (Esposito 1991). All grasshopper viruses are physically similar and have roughly the same deoxyribonucleic acid (DNA) size. They differ from EPV's in other insect orders and other animal poxviruses. Indeed, there is no evidence to suggest any close relationship or similarity between grasshopper entomopoxviruses and other viruses of vertebrate or invertebrates (Langridge 1984).

Virus particles are embedded in a crystalline proteinaceous matrix referred to as an occlusion body (OB). OB's vary in size from 3 to 12 microns (μm) in diameter and may each contain up to several hundred virus particles. Twelve μm equal about 1/20,000th of an inch. OB's offer the virus particles some protection from environmental conditions and are thought to be responsible for transmission of a virus from one grasshopper to another. When OB's are ingested by a grasshopper, the virus particles are released and penetrate through the digestive tract into the body of the grasshopper. Infection by grasshopper EPV's appears to be restricted to the fat body, a tissue which is used to store food reserves and metabolize food. After the virus particles enter a fat body cell, they replicate and pack the cytoplasm with new OB's that contain virus particles. Virus particles will also spread to other fat body cells until nearly all the cells in the fat body are infected with virus (Henry et al. 1969, Granados 1981).

EPV's are the only viruses containing DNA that have been found in field grasshoppers. Typically, an EPV will be named after the host species of the original isolation. Following this convention, there are at least 15 grasshopper EPV's reported in the literature (Henry and Jutila 1966, Langridge et al. 1983, Oma and Henry 1986, Henry et al. 1985, Wang 1994).

EPV Laboratory Studies

Cross-infection studies have been reported for only seven grasshopper and locust EPV's (Henry et al. 1985, Oma and Henry 1986, Streett et al. 1990, Lange and Streett 1993). Relative susceptibility of grasshoppers to a given EPV is usually limited to grasshoppers within the same subfamily (Lange and Streett 1993). However, it is interesting to note that some grasshopper EPV's have been found to infect grasshoppers from several different subfamilies.

Henry and Jutila (1966) isolated the first grasshopper EPV from the lesser migratory grasshopper, *Melanoplus sanguinipes*, a frequent pest on crops and rangeland. The virus, referred to as the *Melanoplus sanguinipes* entomopoxvirus (MsEPV), infects mostly species in the genus *Melanoplus* (Oma and Henry 1986). Grasshoppers infected with a sufficient amount of the virus develop slowly, are sluggish, and die from the effects of the virus (Henry and Jutila 1966).

MsEPV is the only grasshopper EPV that has been grown in vitro (outside the body) (Kurtti et al. 1990 unpubl). The *M. sanguinipes* cell culture lines designated UMMSE–1A, UMMSE–4, and UMMSE–8 have proven susceptible to infection by MsEPV. The UMMSE–4 cell cultures show cytopathic effects (undergo cell changes) when inoculated with MsEPV. The virus produced in vitro is both infectious and virulent (poisonous) against *M. sanguinipes*. Occlusion bodies produced in vitro, though, were somewhat smaller—each about 6 μm in diameter (1/40,000 of an inch)—than occlusion bodies produced in vivo (inside the body). The latter were each about 12 μm in diameter.

In the laboratory, mortality from MsEPV occurs in two distinct timeframes over 5 or more weeks. Infectious OB's are not present in grasshoppers that die during the first interval of mortality, so these cadavers are of little importance for pathogen transmission. As dosage increases, the proportion of inoculated grasshoppers that die prior to OB formation increases dramatically. Consequently, the proportion of infected grasshoppers that survive long enough to produce OB's actually decreases

with dosage (Woods et al. 1992). These observations suggest that the strategy for using this virus in an integrated pest management program may well depend on the specific objectives at the time of application. Maximum transmission rates are likely to be attained by applying the virus at low rates, and so an EPV treatment may be an appropriate strategy for grasshopper populations that are increasing in density. A high-density population that is already causing significant damage should be treated with high rates to cause substantial early mortality.

Sublethal effects that have been observed for virus-infected grasshoppers include a delay in development, reduction in food consumption, and potential reduction in egg production by the female. All of these sublethal factors can have a profound effect on grasshopper populations.

The delay in development was reported first by Henry et al. (1969) and later by Olfert and Erlandson (1991). In some cases, grasshopper nymphs infected with MsEPV will remain 9 to 18 days longer in an instar. Total food consumption by grasshoppers infected with MsEPV was reduced by 25 percent at 5 days after infection and up to 50 percent at 25 days after infection. This reduction in food consumption in MsEPV-infected nymphs was directly related to dose.

The effects of MsEPV infection on *M. sanguinipes* egg production are unclear. While it has been difficult to thoroughly describe the effects of MsEPV on *M. sanguinipes* egg production, we have observed that development to the adult stage is delayed by infection, and none of the infected adults in our laboratory studies have produced any eggs.

Routes of Transmission

One of the more likely routes of EPV transmission is through the consumption of infected cadavers. Grasshoppers will commonly consume other grasshoppers that are sick or dying. When grasshopper cadavers were placed in the field, nearly 92 percent of the cadavers were almost entirely consumed after 30 minutes (O'Neill et al. 1994). Under high density conditions, there may be considerable competition for these cadavers with the larger individuals successfully defending the resource against smaller intruding grasshoppers (O'Neill et al. 1993). When both infected and uninfected cadavers were placed in the field, there were no significant differences in the number of cadavers that were partially consumed (K. M. O'Neill, unpublished data).

EPV Field Studies

The Environmental Protection Agency granted an Experimental Use Permit (EUP) for field evaluations of MsEPV in 1988. Field evaluations were conducted from 1988 to 1990. Human and domestic-animal safety studies were completed, and no evidence of infectivity was detected in any of the studies. Toxicology data to identify hazards that MsEPV might present to nontarget organisms were also conducted with no evidence of toxicity or pathogenicity (poisonous or disease-related effects) observed in any of the animals examined in these studies. In addition, Vandenberg et al. (1990) did not observe reductions in longevity or pathological effects when MsEPV was tested against newly emerged adult workers of the honeybee, *Apis mellifera.*

Field evaluations of the potential for using MsEPV for grasshopper control were conducted during 1989. Plots were treated with virus that was formulated in starch granules (McGuire et al. 1991). At 13 days after application, prevalence (the number of diseased insects at any given time) was estimated at 14 percent and 23 percent in the plots receiving the low or high application rates, respectively. Prevalence was estimated at 9.2 percent in the control plots at 13 days after application, indicating that considerable dispersal between plots had already occurred (Streett and Woods 1990 unpubl.). Our field studies from 1989 emphasize the problems associated with evaluation of microbial insecticides against insects with considerable dispersal capabilities. That we can infect at least 23 percent of the population with a rate of 10 billion OB's/acre (24.7 billion OB's/ha) is clear. The actual infection levels, in view of the dispersal problem and early mortality from the pathogen, are probably much higher.

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