

RESPONSE OF GYPSY MOTH LARVAE TO HOMOLOGOUS AND HETEROLOGOUS NUCLEAR POLYHEDROSIS VIRUS

Kathleen S. Shields¹ and Edward M. Dougherty²

¹USDA Forest Service, Northeastern Forest Experiment Station,
51 Mill Pond Rd., Hamden, CT. 06514

²USDA, Agricultural Research Service, Insect Pathology Laboratory, Beltsville, MD 20705

ABSTRACT

The gypsy moth, *Lymantria dispar*, is not particularly susceptible to baculoviruses other than the nuclear polyhedrosis virus originally isolated from the species (LdMNPV). The multiple enveloped nuclear polyhedrosis virus of *Autographa californica* (AcMNPV), a very virulent baculovirus that replicates in a large number of Lepidopteran species, only rarely produces mortality in gypsy moth larvae regardless of the dose ingested.

We were unable to obtain a LC₅₀ for AcMNPV in first instar gypsy moth larvae, and observed only occasional mortality among larvae fed concentrations of polyhedral inclusion bodies (PIB) exceeding 10⁹ PIB per cup. This dose was more than six million times the dose required for a LC₅₀ with the homologous virus.

The pathogenicity of AcMNPV for gypsy moth was not enhanced when PIB were produced *in vivo* in alternate hosts or *in vitro* in susceptible gypsy moth cell lines, or when PIB were fed to larvae in combination with inactivated homologous virus. Alkali-disrupted AcMNPV PIB were not infectious *per os* or when injected into the hemocoel of gypsy moth larvae, although when injected into *Trichoplusia ni* larvae, 100% died of nucleopolyhedrosis.

Infections were readily established in gypsy moth larvae when extracellular non-occluded AcMNPV, produced in TN-368 cells *in vitro*, was injected into the hemocoel, but only low yields of PIB were obtained from cadavers and lethal times varied from 9 to 20 days.

The pathway of infection and histopathological effects of AcMNPV on *L. dispar* tissues were aberrant compared to what is observed during LdMNPV infection. Larvae infected with AcMNPV often had ruptures in the cuticle, presumably the result of dense accumulations of fibrous material that were observed in infected epidermal cells. Midgut cells, while not obviously infected, gradually degenerated and the midgut became grossly distended. Hemocytes became infected but little budding of non-occluded virus was seen. Other tissues in the body cavity were infected in a haphazard fashion. Pericardial and circumesophageal nephrocytes did not replicate AcMNPV, but accumulated electron-dense granules and underwent a significant color change. It is unclear whether the changes in nephrocytes are merely a form of necrosis due to an aberrant infection or whether these cells play a role in insect defense.