

SYSTEMIC RESISTANCE IN GYPSY MOTH TO LdNPV

Kelli Hoover, Diana Cox-Foster, Mike Grove and Jim McNeil

Department of Entomology, The Pennsylvania State University, 501 ASI Building, University Park, PA, 16802

Abstract

Fourth-instar gypsy moth (*Lymantria dispar*) larvae become markedly more resistant to its host-specific baculovirus, *L. dispar* nucleopolyhedrovirus (LdNPV) as the insect ages within a stadium. This resistance is systemic because it cannot be overcome when bypassing the midgut and delivering the virus directly into the hemocoel. We report here that larvae were able to clear virus, and this was markedly more pronounced in insects inoculated at the most resistant stage (48 hours post-molt). Larvae were inoculated intrahemocoelically with budded virus of a recombinant of LdNPV that expresses lacZ under control of the hsp70 from *Drosophila* (LdNPV-hsp70/lacZ) either immediately after molting (4⁰s) or at 48 hours post-molt (4⁴⁸). Insects were bled at 24-hour intervals until larvae began to die (Day 11). Hemolymph from each insect was measured for (1) expression of lacZ in hemocytes; and (2) progeny BV titer in cell-free plasma by plaque assay. In both 4⁰s and 4⁴⁸s, evidence of viral infection (hemocytes signaling lacZ and infectious budded virus) was detected at 3 days post-infection (dpi), but in both stages the proportion of insects having infected hemocytes decreased late in

infection. Also, virus titers dropped to undetectable levels during the course of infection in 4⁴⁸-stage insects; whereas, titers increased in 4⁰-stage larvae. Our data suggest that both stages of insects can overcome viral infection by some form of immune response and/or apoptosis, but that resistant-stage insects clear virus far more effectively. We hypothesize that clearing of virus occurs by induction of immune responses against the virus itself or against virally-infected tissues. A potential immune response is indicated by 1) hemocytes encapsulating the tracheal system servicing the midgut; 2) chemical immuno-suppression by diethyldithio-carbamic acid decreasing developmental resistance in a dose-dependent manner; and 3) an increased activation of enzymes associated with immune responses. Intrastadial developmental resistance may also involve host tissues becoming refractory to infection and/or failure of hemocytes (and/or other tissues such as fat body) to amplify the virus. Preliminary studies in our lab, and published reports in other systems, suggest that these anti-viral responses are hormonally mediated.