EPIZOOTIOLOGY OF GYPSY MOTH NUCLEAR POLYHEDROSIS VIRUS

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ABSTRACT

Recent experimental findings demonstrate that two distinct waves of mortality of gypsy moth larvae from nuclear polyhedrosis virus (NPV) occurs during larval development. The evidence suggests that early instars acquire lethal doses of NPV from the surface of the egg mass and the cadavers of these larvae produce inoculum that causes a second wave of mortality among late instars. Transmission of NPV between gypsy moth generations appears to occur primarily by way of contamination of egg masses from environmental sources during oviposition. Other factors influencing NPV epizootiology including Foliage chemistry, weather and genetic effects are discussed.

INTRODUCTION

Epizootics of nuclear polyhedrosis virus (NPV) frequently cause precipitous declines of high density populations of gypsy moth *Lymantria dispar* L. (Bess 1961, Campbell 1963, 1967, Campbell and Podgwaite 1971, Doane 1970). In low density gypsy moth populations, mortality caused by NPV is usually quite low (Campbell and Podgwaite 1971). Epizootics of NPV are typical of high density populations of many species of Lepidoptera (Stairs 1972) including other lymantriids (Murray and others 1989). With gypsy moth, mortality from NPV usually peaks among the late larval stadia (Glaser 1915, Doane 1970, Woods and Elkinton 1987). In this manuscript we review recent findings concerning the causes of gypsy moth NPV epizootics, the mechanisms of transmission between and within host generations and the impact of various factors that influence the level of mortality from NPV.

TRANSMISSION WITHIN A HOST GENERATION

The causes of NPV epizootics in insect populations and why they are associated with high host densities have not been fully elucidated. Possible causes fall into two major categories. The first, which was widely expounded in earlier literature, is that stress factors such as crowding or starvation induce expression of NPV infections that are latent in many, if not most, of the individuals in a population (Bergold 1958, Steinhaus 1958, Aruga 1963, Vago 1963). For gypsy moth, such stresses presumably attain peak values during late instars when defoliation reaches a maximum extent. Consequently, the idea that epizootics are induced by stress appears to explain why highest mortality from NPV occurs among late instars. To date, however, there has been no experimental demonstration that epizootics can be triggered by stress factors associated with high density for gypsy moth, or indeed, for any other Lepidoptera (Evans 1986).

The second major theory holds that NPV epizootics occur when the environment becomes heavily contaminated with polyhedral inclusion bodies (PIBs) that are released in great numbers from cadavers of NPV-killed larvae. For gypsy moth NPV, this idea was championed by Doane (1970, 1975, 1976), who suggested that a certain percentage of early instar larvae become infected with virus in the process of hatching, and that the PIBs released from these larvae when they die provide the inoculum for later instars. Consequently, in situations where both the insect density and the initial virus concentration are high enough, a virus epizootic results. In outbreak or pre-

outbreak populations, we would expect this mortality to be density dependent (i.e. reaches a maximum at high density). However, Doane (1976) predicted that following an epizootic mortality from NPV would be density independent. High levels of mortality from NPV would occur even in populations that have declined to very low levels because the concentration of PIBs in the environment remains very high. (Doane 1970, 1975, 1976; Podgwaite et al 1979).

Research by Woods and Elkinton (1987) has provided strong support for Doane's hypothesis concerning the development of epizootics. Mortality from NPV and other causes was quantified over several years from both high- and low- density populations on Cape Cod, Massachusetts. Larvae were collected from these populations once a week until the onset of pupation. These larvae were reared individually in cups with artificial diet (Bell and others 1981) in an outdoor insectary and inspected at frequent intervals to determine the proportion that died from NPV. Death from NPV was confirmed by examining the cadavers for the presence of PIBs in the body tissues.

The results indicated that in both high and low density gypsy moth populations, mortality from NPV usually follows a bimodal temporal pattern. There is a first wave of mortality among first to third instars followed by a period of reduced mortality and then a second wave when the majority of the larvae are fifth and sixth instars. The highest rates of mortality from NPV always occurred during the second wave (Woods and Elkinton 1987). In addition, the amount of NPV on foliage was measured in samples of oak leaves collected each week from one of the plots. NPV was extracted from the foliage (Podgwaite and others 1979) and the extract was bioassayed by soaking gypsy moth eggs from a laboratory colony in the extract and recording the mortality from NPV among neonates that hatched from the eggs. The bioassay results showed that NPV contamination of foliage also exhibited a bimodal wave that coincided with peak mortality from NPV among the larvae in the natural population. Finally, the bimodal pattern was also produced in laboratory experiments. Gypsy moth egg masses were collected in the field and permitted to hatch. The neonates were divided into two groups. Members of the first group were reared individually in 30 ml cups with artificial diet (Bell and others 1981). The remaining larvae were reared in groups of 10 in 180 ml cups. In both groups there was a first wave of mortality that occurred among instars 1-3. Among the larvae reared individually there was no mortality from NPV among older instars. In contrast, the larvae reared in groups of 10 exhibited a pronounced second wave of mortality among late instars. Both field and laboratory results support Doane's hypothesis that early instar mortality contaminates the foliage with NPV and provides the primary inoculum for late instar infections.

There have been several other studies that support Doane's hypothesis or show the bimodal pattern of mortality in gypsy moth populations. Higashiura and Kamijo (1978) describe a bimodal or trimodal wave of mortality from NPV in Japanese populations of gypsy moth. Woods and others (1988) showed that *Bacillus thuringiensis* (Bt) applications to gypsy moth populations during the first or second stadium resulted in substantially less mortality from NPV among later instars than was observed among late instars from untreated plots. A similar reduction in mortality from NPV was noted among larvae emerging from egg masses the following year in the treated plots. There are several possible explanations for these results, but an obvious one is that the Bt killed early instars before they died from NPV, thereby reducing the inoculum available to later instars.

Finally, a simulation model of gypsy moth/NPV interaction (Valentine and Podgwaite 1982) has been constructed and reincarnated as the pathogen submodel of the Gypsy Moth Life System Model (Sheehan 1987). The model simulates the deposition and movement of NPV PIBs in the forest canopy and the consumption of foliage by gypsy moth larvae. The model exhibits a clear bimodal pattern of mortality very similar to that observed by Woods and Elkinton (1987) in real populations.

These results should not be interpreted as implying that there is only enough time for two cycles of viral replication and host death in the gypsy moth larval stage. In the laboratory at 28° C, 7-14 days elapse between ingestion of a lethal dose and death of the larvae (Woods and Elkinton 1987), whereas larval development at this temperature takes ca. 28 days for males and 31 days for females (Casagrande and others 1987). Late instar larvae can readily ingest inoculum and die from NPV before pupation. The second wave of mortality in the population consists in part of a third wave (documented by Higashiura and Kamijo 1978) that is rarely distinct from the second. After pupation, however, levels of mortality from NPV are much lower (Murray and others 1990).

All of these studies support, but do not prove, the idea that contamination of foliage with NPV from cadavers of early instars is the primary source of inoculum for the second wave of mortality among late instars. We have initiated studies to examine the alternative hypothesis that late instar gypsy moths acquire NPV from protected daytime resting locations. Beginning with the fourth instar, gypsy moths in low density populations leave the canopy of the forest at dawn and seek resting locations in the litter or under bark flaps (Forbush and Fernald 1896), where they spend the daylight hours. Previous research has shown that such protected locations become highly contaminated with NPV and that infectious NPV persists in such locations for at least a year (Podgwaite and others 1979, Weseloh and Andreadis 1986). In contrast, NPV deposited on foliage is short-lived (Podgwaite and others 1979). It is rapidly denatured by ultra violet light (Jaques 1985) or washed off by rain (Doane 1970). Furthermore, previous studies have shown that gypsy moth neonates can acquire lethal infections of NPV merely by walking over such contaminated substrates (Weseloh and Andreadis 1986, Woods and others 1989). We therefore thought that it was possible that a proportion, perhaps a majority, of larvae that die as late instars acquire NPV from such contaminated resting locations rather than foliage. On the other hand, large larvae require substantially higher doses of NPV to obtain a lethal infection than smaller larvae (Briese and Podgwaite 1985), and it is not clear that any of the NPV contaminant of resting sites would actually be ingested.

In 1989 we ran two experiments designed to investigate this possibility. Both experiments involved application of NPV to burlap bands. In the first experiment Gypchek, a formulated NPV insecticide (Lewis 1981), was applied to burlap bands wrapped around trees in a moderate density gypsy moth population. Burlap bands around other nearby trees remained untreated. The burlaps were visited daily, to prove that the trees were far enough apart that there was negligible movement of larvae between trees. To prove that the trees were far enough apart that there was negligible movement of larvae between trees, the larvae underneath the burlap were marked with a dot of paint that was distinct for each day and treatment. After several days, larvae were collected and reared individually in 30 ml cups on artificial diet. Mortality from NPV was monitored. Results from these experiments will be described elsewhere in detail, but, in summary, there was virtually no mortality from NPV among larvae collected from control trees and a small, but significantly greater, level of mortality among larvae from the treated trees.

The second experiment was similar to the first except that NPV was applied to burlap in the form of larvae that were recently killed by NPV and that experiments were performed on branches of oak foliage inside rearing cages. There were three treatments. In the first treatment dead larvae were smeared on the foliage, in the second treatment the dead larvae were smeared on the burlap bands and the third treatment (controls) received no dead larvae. Most of the larvae recovered from cages that had NPV smeared on the foliage subsequently died from NPV. None of the larvae from the control cages died and a small, but significant, proportion of the larvae from the burlap-treated cages died from NPV.

These experiments prove that it is possible for late instar gypsy moth larvae to acquire lethal infections of NPV from contaminated resting locations. However, it tells us very little about how important this is as a source of inoculum compared to foliage contamination in natural populations.

The only data we have that sheds light on this question are results from experiments in which we created populations of gypsy moths on 1 ha plots by releasing large number of field collected egg masses (Liebhold and Elkinton 1989, Gould and others 1990, Elkinton and others unpublished). The eggs were collected from high density populations and were therefore expected to produce a substantial number of infected neonates (Doane 1969, Woods and others 1988, 1990). However, we surface-disinfected the eggs in a 10% formalin solution before we released them on our plots. Doane (1969) showed that such surface treatments will virtually eliminate the mortality NPV among the emerging neonates. We observed virtually no mortality from NPV among larvae collected from these populations either as early or late instars. In a follow-up study in 1988, we created seven additional populations of gypsy moths of which three received eggs that were not surface-disinfected. One of the surface-disinfected populations was created in a stand that had experienced a high density of gypsy moth and had collapsed the previous year. Presumably this site was heavily contaminated with NPV. Once again the populations created with surface-disinfected eggs experienced little or no mortality from NPV. In contrast, all three populations receiving eggs that were not surface-disinfected experienced a pronounced bimodal wave of mortality from NPV that mirrored that seen in natural populations. We believe that these results strongly imply that late instar mortality larvae originates from larvae that die as early instars as Doane (1970, 1975, 1976) proposed.

Other possible mechanisms of horizontal transmission of NPV in gypsy moth populations include vectoring by predators and parasitoids. Lautenschlager and Podgwaite (1977, 1979) showed that NPV PIBs remain viable after passage through the alimentary tracts of avian and small predators of gypsy moth. Raimo and others (1977) showed that the parasitoid *Cotesia melanoscela* is capable of transmitting NPV infections from diseased to healthy larvae. The importance of these findings to NPV epizootiology has not been investigated.

TRANSGENERATIONAL TRANSMISSION

In the foregoing discussion we presented evidence that acquisition of lethal infections by early instars is the key to epizootic development. How, then do early instars become infected? In other words, how is NPV transmitted between generations of gypsy moth? There are two possible basic mechanisms: either the infection is transmitted from the female to her offspring (vertical transmission) or else larvae or eggs become infected from NPV inoculum in the environment. The latter mechanisms are usually considered horizontal transmission (Andreadis 1987), although some researchers use the term vertical transmission to refer to both forms of transmission across host generations.

Shapiro and Robertson (1987) presented evidence for vertical (maternal) transmission of NPV in gypsy moth. They fed LD_{50} and LD_{80} doses of NPV to gypsy moth larvae and observed PIBs in the host tissues of the adult survivors. They found that 4.7% and 11.5% of the respective progeny of these adults died from NPV. However, other studies have failed to demonstrate vertical transmission. Shields (1984) found no mortality from NPV among the offspring of the survivors of LD_{50} doses of NPV nor any developmental or physiological differences between these and undosed (control) larvae. Murray and Elkinton (1989) found no evidence for maternal transmission among the offspring of larvae that survived low doses (LD_{17}) of NPV. Finally, Murray and others (1990) found no viral DNA in the adult tissues of gypsy moths that survived various doses of NPV. Viral DNA was detected in hemolymph extracted from some of the pupae arising from dosed larvae but all of these individuals died from NPV before adult eclosion. At present we cannot explain the differences between these results and those of Shapiro and Robertson (1987).

Doane's finding (1969) that nearly all of the mortality of larvae reared from field collected egg masses could be eliminated by surface disinfection suggests that maternal transmission, if it exists,

occurs largely by way of surface contamination (transovum transmission) as opposed to within the egg (transovarial transmission). To date there has been no conclusive demonstration of transovarial transmission of NPV in any Lepidoptera (Evans 1986), although there is circumstantial evidence for it (reviewed in Burand and others 1986). For gypsy moths this evidence includes experiments which showed that NPV mortality can be induced with chemical stressors (Yadava 1971) or by foreign viruses (Longworth and Cunningham 1968). However, various treatments or stressors that seem to induce infections may merely increase the susceptibility of larvae to normally sublethal laboratory contaminants of NPV. We believe that polymerase chain reaction (PCR) technology will soon enable us to determine if minute quantities of viral DNA exist in gypsy moth embryos and help determine if transovarial transmission occurs.

Murray and Elkinton (1989) reported experiments indicating that gypsy moth egg masses acquire NPV contaminant primarily from the surface on which they are deposited. In the first experiment the mated adult females that had survived a dose of NPV as larvae as well as mated undosed females were allowed to oviposit on tree stems in a site with a low density natural population of gypsy moths. Other dosed and undosed females were allowed to oviposit into plastic cups that were held over the winter in an outdoor insectary. The following spring the egg masses from the four groups were collected and the neonates were reared on diet to determine the proportion that acquired a lethal dose. There was no difference in NPV-caused mortality among neonates from dosed and undosed parents but more of the neonates from egg masses that were laid on trees in the field died from NPV than neonates from egg masses deposited into cups. In another study, that we called the site switching experiment, females were collected as pupae from a high density site that had experienced high levels of mortality from NPV and from a low density site that experienced little mortality from NPV. The adult females from the high density site were caged on trees, mated and allowed to oviposit in the low density site and the low density females oviposited in the high density site. Other females were caged and oviposited in the same site from which they were collected and another group of females from the high density site were mated and allowed to oviposit into cups in the laboratory. The following spring the eggs were collected and the neonates reared on diet to determine NPV-caused mortality as before. Higher levels of mortality occurred among larvae reared from egg masses oviposited in the high density site regardless of the source of the mother. Mortality among neonates that hatched from eggs deposited in the low density site was not different from the mortality among those hatched from eggs deposited into cups.

A third experiment examined the role of rainfall in the contamination of egg masses with NPV. Mated laboratory-reared females were allowed to oviposit on tree stems in the high and low density sites underneath waxed cardboard shelters that kept off the rain but left the egg mass otherwise exposed to the open air. Other females oviposited on tree stems without shelters. Again these egg masses were collected just before hatch the following spring and mortality of neonates from NPV was recorded. There was no difference in mortality among neonates from sheltered versus unsheltered egg masses, but again there was much higher levels of mortality among larvae hatched from egg masses that were oviposited in the high density site compared with larvae from egg masses deposited in the low density site. The level of mortality among neonates from the egg masses laid in the high density site ranged from 20 - 46% and was comparable to that observed among neonates from naturally occurring egg masses at this site. These values are far higher than the 11% mortality reported by Shapiro and Robertson (1987) arising from maternal transmission. Our conclusion is that if maternal transmission does occur, it probably is less important than environmental contamination of eggs as a mechanism of transgenerational transmission, at least in high density sites.

Circumstantial evidence in support of this view has been obtained by Woods and others (1990) who found that egg masses collected from tree stems are more highly contaminated with NPV than egg masses collected from the ground or from understory vegetation. During an epizootic, late

instar larvae die in great numbers in resting locations on tree stems and contaminate the bark with PIBs.

The exact mechanism by which egg masses become contaminated by NPV remains to be determined. Murray and Elkinton (1989) found that egg masses recovered in August a few days after oviposition and held over the winter in an insectary did not have a significantly different NPV load than egg masses that overwintered on the stems of trees. This result suggested that the contamination occurs during the process of oviposition. The adult females may become externally contaminated as they drag their abdomen over the bark surface or the egg masses may be deposited directly onto contaminated substrates. In follow-up studies, Murray and Elkinton (1990) sprayed NPV (Gypchek) onto the bark surface of bolts cut from living oak trees. Uninfected laboratory-reared females were mated and allowed to oviposit on the contaminated bark surface and on bolts that had received no NPV. The egg masses were held over the winter, removed from the bark prior to hatch and the neonates were assayed as above. Neonates emerging from egg masses on the contaminated bark had much higher levels of mortality from NPV than neonates from uncontaminated bark. Furthermore, neonates from the innermost part of the egg masses near the bark surface, had higher levels of mortality than from the outer layer of the egg masses. These results support the conclusion that NPV is transferred from the contaminated substrate to the egg mass during oviposition. The egg mass may provide a protective cover for the PIBs that otherwise might not survive exposure to UV radiation or other factors over the 9 month interval between oviposition and hatch.

Other possible sources of egg mass contamination may be from the adult male during the process of mating or from egg parasitoids such as *Ooencyrtus kuvanae*. None of these mechanisms have been investigated experimentally. However, in the experiments of Murray and Elkinton (1989), cited above, females in the site switching experiment were mated with naturally occurring males at the site of oviposition whereas in the other two experiments (dosed versus undosed parental females and sheltered versus unsheltered egg masses) the females were mated with laboratory reared, uninfected males. High levels of NPV mortality were observed among neonates from egg masses deposited in the high density site regardless of parental source, suggesting that the male parent contributed little to egg mass contamination.

Other studies suggest that infection of neonates after they leave the egg mass may be another important route of transgenerational transmission. In laboratory experiments Weseloh and Andreadis (1986) showed that neonates can acquire lethal infections of NPV by walking over naturally occurring contaminated surfaces including soil and pupal-debris mats from high density populations. These findings were corroborated in field experiments by Woods and others (1989) who showed that neonates acquired lethal infections by crawling over contaminated bark. A limitation of both of these studies was that larvae were transferred onto artificial diet. As far as we know, larvae can acquire NPV infections only by ingestion. This either occurred while the larvae were crawling over the contaminated surfaces or they may have transferred the PIBS onto the diet from which they were later consumed. Such transfer may occur more readily on diet than on foliage in the field. However, we investigated this in the experiments described above with older larvae. We found that older larvae exposed to contaminated burlap bands and reared subsequently on foliage acquired NPV infections to the same extent as larvae reared on diet. The importance of such mechanisms of between generation transmission remains to be determined. Based on the limited results with artificial populations of gypsy moth described above, we suspect that egg mass contamination is more important. We anticipate future experiments to settle this issue.

FACTORS INFLUENCING MORTALITY FROM NPV

Rainfall and Relative Humidity

Many researchers believe that rainy conditions induce NPV epizootics. Analyses of the Melrose Highlands data by Campbell (1967) indicate that population declines were associated with heavy rainfall occurring in June, most notably during the region-wide collapse of gypsy moth populations that occurred in 1922. Of course, in most of these studies, the actual cause of population decline were unknown. In 1989 a dramatic epizootic of *Entomophaga maimaiga* decimated populations of gypsy moth in New England, a year with near record rainfall in May and June. The evidence suggests that this agent was introduced in 1910 or 1911 (Hajek and others unpublished) from Japan. The known association of *E. maimaiga* epizootics with rainy conditions in Japan (Shimazu and Soper 1987) and its ubiquitous occurrence throughout the northeast in 1989 suggests that it may have caused many of the previous population declines in rainy years.

Experiments by Wallis (1957) suggested that high relative humidity was associated with NPV infection. However, the laboratory data cited were never actually presented and the observations of NPV mortality in field populations show no more than onset of high mortality among late instars which happened to coincide with a period of heavy rainfall in that particular place and year. No such correlations were observed in the populations studied by Woods and Elkinton (1987). Furthermore, the mechanisms by which rainfall would promote NPV infection have not been established. Rainfall might serve to distribute PIBs more evenly across the foliage surface thereby increasing the chance of larval encounter. This has been demonstrated with NPV of Douglas fir tussock moth (Thompson 1978). However, rainfall also washes larval cadavers off the foliage entirely (Doane 1970), thereby removing the inoculum from potential consumption. We conclude, therefore, that there is little evidence for rainfall or relative humidity enhancing gypsy moth infection from NPV.

Host tree species and foliage chemistry

Keating and Yendol (1987) demonstrated that larvae fed NPV on aspen leaves were much more likely to become infected larvae fed NPV than on oak leaves. These findings are supported by observations of Bess (1961) that epizootics of NPV occur more frequently and at lower population density in aspen than in oak stands. Follow-up studies (Keating and others 1988) indicated that foliage containing large quantities of tannins in foliage have reduced susceptibility to NPV. Tannins apparently bind to virus particles in the midgut and perhaps inhibit passage across the peritrophic membrane. Tannins also affect midgut pH (Schultz and Lechowicz 1986) which in turn affects the dissolution rates of PIBs and the consequent release of virus particles. Other research demonstrates that tannin concentrations increase in the foliage of trees experiencing defoliation. (Schultz and Baldwin 1982, Rossiter and others 1988). These findings have lead to speculation that larvae feeding on trees undergoing defoliation may become more resistant to NPV epizootics.

Genetics and evolutionary considerations

Theoretical studies of host/pathogen interactions (Levin and others 1982, May and Anderson 1983) indicates that the virulence of the pathogen is related to the mechanisms of transmission. In systems where vertical transmission predominates, pathogens frequently evolve to produce benign avirulent infections. There are many examples of such trends towards benign mutual coexistence of host and pathogen including myxomatosis in rabbits in England and Australia (Fenner and Ratcliffe 1965), trypanosomiasis in native versus introduced ruminants in Africa, (Allison, 1982) and plague bacillus in rats (Levin and others 1982). However, if transmission depends primarily upon infective particles which are released from host cadavers, as is the case for insect NPVs, then, in general, selection should favor more virulent strains. On the one hand, selection should favor viruses which replicate quickly and are released into the environment in the shortest period of time.

On the other hand, if the virus kills its host too quickly, then the larva may not have grown to a size that permits maximum production of virus particles. Intermediate levels of virulence and replication rates seem to be the rule among insect NPVs (Anderson and May 1980, 1981, May and Anderson 1983). Even when selection favors the most virulent strain of the virus, selection acting on the host favors resistance so the resulting populations may reflect a balance between these opposing forces.

The experiments cited above suggest that there are a variety of mechanisms of transmission of NPV and we do not yet know their relative importance. We believe that the importance of these individual mechanisms may vary between high and low density populations. When population densities are high there is a sufficient density of cadavers that died of virus during early instars so that older instars have a high probability of consuming a lethal dose. Similarly, high concentrations of NPV on tree stems may heavily contaminate egg masses (Murray and Elkinton 1989). In low population densities, which are often less than 10 larvae per tree, the probability of encountering a lethal dose of virus is obviously quite small. Indeed, theoretical studies by Anderson and May (1981) suggest that without some mechanism of survival or reservoir, NPVs are likely to go extinct when their hosts are at low density. If maternal transmission occurs, as Shapiro and Robertson's (1987) results indicate, it seems likely that it would be a far more reliable mechanism of transmission relative to environmental transmission at low population density, even if the probability that an offspring would become infected by maternal transmission is quite low. Consequently, we believe that viral reproductive strategies may vary with host population density and that more virulent genotypes may be selected for at higher population densities. In many systems stable polymorphisms of mixed genotypes or intermediate levels of virulence may be maintained.

There already exists a considerable body of evidence that different strains of NPV vary in virulence. A ten fold difference in LD_{50} was reported by Vasiljevic and Injac (1973) for Yugoslavian isolates of the virus, while Shapiro and others (1984), found as much as a sixteen fold difference in the activity of North American virus isolates. A large number of NPV wild isolates have been shown to be genotypically heterogeneous (Falkner and Carstens 1986). We do not know whether any of these differences are related to the density of the population from which they were collected.

Selection for resistance of gypsy moth to NPV infections may also occur, particularly during an epizootic. Gypsy moths from different locations vary in susceptibility to a given viral isolate (Rollinson and Lewis 1973, Skatulla 1987). Myers (1988) has speculated that short term genetic changes in susceptibility to pathogens may account for the cyclic pattern of population densities of many forest insects, although there is no direct evidence for this. We anticipate future studies to determine if there are systematic changes in NPV virulence and host susceptibility before during and after an NPV epizootic.

SUMMARY

Two distinct waves of mortality from NPV occur during larval development of gypsy moth. The evidence suggests that early instars acquire lethal doses of NPV from the surface of the egg mass and the cadavers of these larvae provide inoculum that causes a second wave of mortality among late instars. Transmission of NPV between gypsy moth generations appears to occur primarily by way of contamination of egg masses from environmental sources during oviposition. Other factors including foliage chemistry, and genetic effects influence the level of mortality caused by NPV. Future studies involving experimental manipulation of field populations coupled with simulations with the gypsy moth life system model will serve to validate and extend these findings.

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